Neural Markers of Antisocial Behaviour in Offenders and their Relationship with Risk-Factors of Offending

by

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The above committee determined that the thesis is acceptable in form and content and that a satisfactory knowledge of the field covered by the thesis was demonstrated by the candidate during an oral examination. A signed copy of the Certificate of Approval is available from the School of Graduate and Postdoctoral Studies.

ABSTRACT

The relationship between neural integrity and offending patterns has been increasingly evaluated to better understand the mechanisms that predispose towards offending behaviours. While this work has made inroads into establishing the neural underpinnings of offending behaviour, some important knowledge gaps remain. First, the extent to which neural abnormalities extend across both rest and task-based contexts in offenders remains unclear. Thus, Studies 1 and 2 compared whole-brain *resting-state power spectra* (rs-PS) between offenders and non-offenders, and Study 3 performed a meta-analysis of task-based neural activity differences between offenders and non-offenders. Results from Studies 1 and 2 indicated that offenders were characterized by decreased rs-PS in five (Study1) and six (Study2) of the eight evaluated resting-state networks compared to non-offenders (which occurred as a result of both decreased low-frequency activity and increased high-frequency activity). In Study 3, offenders presented aberrant task-based activity in the left IFG and increased activity in the left MOG compared to non-offenders. Thus, some dysfunctions spanned across both resting-state and task-based metrics (i.e., within left IFG and left MOG), suggesting stable abnormalities between offenders and non-offenders, while rest-related dysfunctions were more extended than those observed in offenders' task-based activity. In addition, this work aimed to assess the degree to which offenders' aberrant neural processes were influenced by several antisocial and criminogenic variables (e.g., psychopathic traits, drug use and features of criminal history). In Studies 1 and 2, cocaine use and number of criminal convictions predicted rs-PS disruptions, but psychopathy and cocaine-dependence status did not. In Study 3, activity within left MOG and left PCC appeared

more specific to offenders with violent offence histories, while left IFG activity appeared to be more specific to contexts within which cognitive processes (rather than emotional processes) were interrogated. Overall, this work uncovered several regions of abnormal neural activity, across two different neuroimaging modalities, that differed between offenders and non-offenders. Potentially, the application of neurophysiological treatments (e.g., neurofeedback) to these sites could have treatment or rehabilitative benefits. Nonetheless, these results suggest that neural disruptions are not uniform across offenders, but rather vary as a function of antisocial/criminogenic features and processing type.

Keywords: Offenders; fMRI; resting-state; meta-analysis; risk-factors

AUTHOR'S DECLARATION

I hereby declare that this thesis consists of original work of which I have authored. This is a true copy of the thesis, including any required final revisions, as accepted by my examiners.

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STATEMENT OF CONTRIBUTIONS

The work presented in Chapter 1 has been published as:

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In accordance with the license under which the article was published, and as the main author of the article, I have the right to include this work in my dissertation. In collaboration with my co-authors, I formulated the research questions, performed data analysis, and wrote the article.

Throughout this dissertation, I have used standard referencing practices to acknowledge ideas, research techniques, or other materials that belong to others. I performed the data analysis and writing of Studies 2 and 3. Undergraduate research assistants volunteering at the Clinical Affective Neuroscience Laboratory for Discovery and Innovation (CANdiLab), directed by Dr. Matthew Shane, helped with gathering and vetting of background check data in Study 2 and screening the articles included in Study 3's meta-analysis.

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LIST OF ABBREVIATIONS AND SYMBOLS

β	Beta
χ	Chi
ACC	Anterior Cingulate Cortex
ALE	Activation Likelihood Estimation
ALFF	Amplitude of Low-Frequency Fluctuation
ANOVA	Analysis of Variance
ASI-X	Addiction Severity Index – Expanded
ASPD	Antisocial Personality Disorder
AU	Auditory
AUC	Area Under the Curve
BAS	Behavioural Approach
BG	Basal Ganglia
BOLD	Blood-Oxygen-Level Dependent
BPD	Borderline Personality Disorder
CAD	Canadian Dollar
CBT	Cognitive-Behavioural Therapy
CD	Conduct Disorder
CU	Callous-Unemotional
Ctls	Controls
DAN	Dorsal Attention Network
DF	Degrees of Freedom
DMN	Default-Mode Network

DLPFC	Dorsolateral Prefrontal Cortex
DO	Dependent Offenders
DSM-IV	Diagnostic and Statistical Manual of Mental Disorders, fourth edition
DWI	Driving While Intoxicated
ECN	Executive Control Network
EEG	Electroencephalogram
e.g.	*exempli gratia*
EPI	Echo Planar Imaging
ERN/Ne	Error-Related Negativity
ERPs	Event-Related Potentials
F	F-Test score
FA	Flip Angle
fALFF	Fractional Amplitude of Low-Frequency Fluctuation
fMRI	Functional Magnetic Resonance Imaging
FNC	Functional Connectivity
FOV	Field of View
FWE	Familywise Error
FWHM	Full-Width-as-Half-Maximum
GAD	General Anxiety Disorder
GICA	Group Independent Component Analysis
HZ	Hertz
I_q	Cluster Stability Index
ICA	Independent Component Analysis

i.e.	*id est*
IFG	Inferior Frontal Gyrus
IQ	Intellectual Quotient
LCU	Lifetime Cocaine Use
LFPR	Low Frequency Power Ratio
LG	Language
LOOCV	Leave One Out Cross-Validation
M	Mean
MA	Model Activation
MDD	Major Depression Disorder
MKDA	Multilevel Kernel Density Analysis
MNI	Montreal Neurological Institute
MOG	Middle Occipital Gyrus
mm	Millimeter
MRN	Mind Research Network
ms	Millisecond
N	Number
NDO	Non-Dependent Offenders
N-O	Non-Offenders
N-V O	Non-Violent Offenders
O	Offenders
OR	Odds Ratio
p	Probability

PCC	Posterior Cingulate Cortex
PCL-R	Psychopathy Checklist-Revised
PCL-SV	Psychopathy Checklist Screening Version
Pe	Error Positivity
PET	Positron Emission Tomography
PS	Power Spectra
r	Correlation value
rCBF	Regional Cerebral Blood Flow
ROC	Receiver Operating Characteristic
ROI	Region of Interest
RSN	Resting-State Network
rs-FNC	Resting-State Functional Connectivity
rs-PS	Resting-State Power Spectra
SCID-I/P	Structured Clinical Interview for the DSM-IV
SE β	Unstandardized Beta
sec	Second
SEM	Standard Error of the Mean
SD	Standard Deviation
SDM	Signed Differential Mapping
SDM-PSI	Seed-based d-Mapping with Permutation of Subject Images
SES	Socio-Economic Status
SMN	Sensorimotor Network
SMOTE	Synthetic Minority Oversampling Technique

SMs	Spatial Maps
SN	Salience Network
SPECT	Single-Photon Emission Computerized Tomography
SPM12	Statistical Parametric Mapping version SPM12
SPSS	Statistical Package for the Social Sciences
SUD	Substance Use Disorder
SVC	Support Vector Classifier
Sz	Schizophrenia
t	T-Test score
T	Tesla
TCs	Time Courses
tDCS	Transcranial Direct Current Stimulation
TE	Echo Time
TOM	Theory of Mind
TR	Repetition Time
UK	United Kingdom
VO	Violent Offenders
WAIS-III	Wechsler Adult Intelligence Scale 3rd Edition
z	z-score

Chapter 1. General Introduction

In 2018-2019, 5,874 criminal incidents (Moreau et al., 2020) and 127 incarcerations (Malakieh, 2020) were reported by Canadian police forces per 100,000 adults. Figures from the United States are even more striking, with 539 per 100,000 adults incarcerated in 2019 (Carson et al., 2020); indeed, a third of the U.S. population can expect to be arrested at least once by the time they turn 23 (Brame et al., 2012). This level of criminal activity has important societal consequences. Economically, the cost of apprehension, punishment, and victimization averages $85 billion CAD per year (Easton et al., 2014). Moreover, crime negatively affects victims psychologically, socially, physically, and spiritually (Wasserman & Ellis, 2007). For instance, victims of crimes – particularly violent crimes – often suffer lasting physical injuries. And, beyond these injuries, medical professionals report that victims of violent assaults show increased stress and risk of mental health problems (Resnick et al., 1997) such as higher levels of vulnerability, fear, post-traumatic stress symptoms and are more likely to engage in protective behaviours (Lurigio, 1987). These negative consequences can, in turn, lead to long-term health complications, such as impaired immune system functioning, increased health risk behaviours and inappropriate health care utilization (Resnick et al., 1997). Thus, developing a thorough understanding of the factors underlying offending behaviour may lead to wide-ranging benefits at both societal and individual levels.

In addition to these harms to the victim, there are potential consequences for the accused themselves. This latter category of harm is much less frequently focused on, but may be equally important to consider. For instance, offenders' executive functions

(Meijers et al., 2015, 2018), cognitive control and emotion regulation systems (Umbach et al., 2018) have each been reported to be negatively affected by incarceration. Moreover, offenders often face stigma after conviction and/or imprisonment (Feingold, 2021), negatively affecting their adjustment back into their community (Moore et al., 2016).

To better understand the mechanisms that predispose towards offending behaviours, the scientific community has been increasingly evaluating the relationship between neural integrity and offending patterns (see Aharoni et al., 2013, 2014; Kiehl et al., 2018; Steele et al., 2015). This work has made inroads into establishing potential neural characteristics that could underpin offending behaviours and into identifying those who are most at risk to offend or re-offend (Aharoni et al., 2013, 2014; Coppola, 2018; Gaudet et al., 2016; Kiehl et al., 2018; Poldrack et al., 2018; Steele et al., 2015). Much still needs to be done, however. For instance, the majority of work to date has narrowly selected specific sub-populations of individuals with antisocial features, but little work has considered the broad group of offenders as a potentially homogenous population. Considering the potential homogeneity of the offender population may be important, as this is the most direct way to evaluate whether their common commission of an antisocial behaviour that infringes the law could be related to specific neural features.

Several recent meta-analyses have aimed to summarize the existing knowledge regarding the neural integrity of offending (Deming & Koenigs, 2020; Dugré et al., 2020; Nickerson, 2014; Poeppl et al., 2019; Yang & Raine, 2009). However, these meta-analyses have tended to use a highly heterogenous inclusion strategy that included studies that recruited based on the commission of an antisocial act *and/or* the existence of

antisocial personality traits. Indeed, only a fraction of the studies included in these meta-analyses recruited exclusively from active offender populations (25% in Yang and Raine; 32% in Nickerson; 41% in Peoppl et al.; 37% in Dugré et al.; 36% in Deming & Koenigs). Rather, they recruited heterogeneous groups of community/offender populations based on personality diagnoses/characteristics (i.e., *antisocial personality disorder* (ASPD), *conduct disorder* (CD), psychopathic traits) that are often, but not necessarily, linked to offending behaviour (the link between antisocial *traits* and antisocial *behaviour* is only moderate in magnitude; Black et al., 2010; Forsman et al., 2010). Indeed, some studies included non-offenders, and some studies included only specific subsets of offenders (e.g., psychopathic individuals). One additional meta-analysis has examined neural connectivity patterns that may be associated with antisocial behaviour (Dugré & Potvin, 2021). However, again, only 38 % of the included studies recruited exclusively from active offender populations (indeed, the majority of studies recruited children/juveniles who presented non-criminal antisocial behaviour and/or conduct disorders; Dugré & Potvin, 2021). Thus, while the results from these existing meta-analyses provide important insights into the neural features associated with antisocial *personalities,* the extent to which they similarly identify neural features related to offending *behaviours* remains an open question. With this in mind, a primary goal of my dissertation aims to identify the neural underpinnings of offending behaviour, as rigorously defined by offender status.

Of particular focus is the integrity of the brain's resting-state activity. The past several decades have seen significant interest in this resting-state activity (Raichle, 2015), which occurs in a variety of well-established networks that are believed to underlie the

‘baseline’ activity in the brain (i.e., what the brain is doing when it isn’t specifically involved in effortful tasks; Cieri & Esposito, 2018; Raichle, 2015). Activity within these networks has shown great potential as markers of neural health (Lee et al., 2013; Liu et al., 2008), and activity disruptions in these networks have been associated with various clinical disorders (Barkhof et al., 2014; Cieri & Esposito, 2018; Northoff & Duncan, 2016). Hence, investigating resting-state activity may provide an informative measure of an individual’s overall neural health/integrity level.

Considerable research shows that resting-state activity can be usefully segregated into specialized, modular networks (Wig, 2017), formed by linking temporally synchronized brain regions, each purported to support a particular set of neurocognitive processes. For instance, the DMN is believed to support emotional processing, self-referential mental activity and memory recollection (Raichle, 2015); the Salience Network (SN) and Dorsal Attention Network (DAN) appear to show a substantive role in attributing salience and/or allocating attention to specific environmental features (Seeley et al., 2007; Szczepanski et al., 2013); and the Executive Control Network (ECN) is believed to be involved in highlighting task-relevant information, inhibiting information that is not relevant to the task and using relevant information to select between appropriate behavioural responses (Gratton et al., 2017). Interestingly, several resting-state networks (RSNs) show connectivity increases (e.g., DMN), while other RSNs show connectivity decreases (e.g., SN, DAN, ECN) during rest. The DMN, for instance (which includes the ventral and dorsal medial prefrontal cortex, medial temporal lobe, posterior cingulate cortex, precuneus and lateral parietal cortex) appears particularly active, and its components highly synchronized, when one is at rest (Fox et al., 2015; Raichle, 2015),

but less active, and less synchronized when one is actively engaged in mental tasks. In contrast, regions within the ECN (which includes the dorsal anterior cingulate, anterior insula, dorsolateral frontal cortex, and intraparietal sulcus; Gratton et al., 2017) appear characterized by higher levels of coordinated activity (i.e., synchronization) during active task performance and less active/synchronized during rest. While the precise functions and implications of these rest-related dynamics remain an ongoing concern, several influential theories posit that the underlying non-task-related activity may be related to the brain's intrinsic neural processes (Gohel & Biswal, 2015; Raichle et al., 2001).

Commonly, resting-state activity is studied through spatial maps, functional connectivity (FNC) and Power Spectra (PS). Spatial maps represent the intensity of neural activity within a specific time frame and thus afford insights into which neural regions are most activated or de-activated while one is at rest or performing a task. FNC measures the temporal synchrony of different brain regions (i.e., as activity in one brain region changes, does activity in other brain regions adjust similarly). Power spectra, like spatial maps, is a measure of the intensity of neural activity. However, power spectra allows the decomposition of this signal to investigate the distribution of neural activity across various electrical frequencies. Thus, power spectra represents the signal's mean amplitude across all recordable frequencies (i.e., the strength/amount of signal oscillations at each frequency). Typically, the resting-state spectral activity consists of peak activity within low-frequencies (< 0.10 Hz), a sharply decreased activity within mid-frequencies (0.10-0.149 Hz), and consistently low activity within high-frequencies (0.15- 0.25 Hz; Allen et al., 2011; Kalcher et al., 2014). While the function and dynamics of mid-frequency activity are significantly less characterized in the broader clinical

literature, a higher proportion of low-frequency activity at rest is believed to indicate a more stable, functional network (Biswal et al., 1996), whereas a higher proportion of high-frequency activity at rest has been linked to instability of network activity (Buzsáki & Draguhn, 2004; DeRamus et al., 2020; Salvador et al., 2008). More generally, frequency oscillations underlie the synchrony of brain activity (Fransson, 2005; Greicius et al., 2003), and coherent neural oscillations are essential for the development and maintenance of healthy brain functions (Pizoli et al., 2011). Thus, if our brain activity were music, power spectra would be a measure of the rhythm/coherence between the different instruments (e.g., RSNs). Studies 1 and 2 of this dissertation focus careful attention on the relationship between high/low-frequency activity and offending behaviour to comprehensively evaluate the relationship between offending and overall RSN-network health.

1.1. Neural functioning in Offenders

A substantial proportion of the work investigating rest-related neural activity in offenders has focused on the DMN and ECN. Disruptions in the DMN's power spectra activity in offenders have previously been related to antisocial traits (Thijssen & Kiehl, 2017), suggesting that offenders with heightened antisocial traits have less efficient DMN activity. Consistent with this interpretation, offenders show reduced white matter tracts (Waller et al., 2017; which carry information to/from disparate brain regions) – reduced grey matter integrity (Raine & Yang, 2006; where the signals are created and received) and reduced functional connectivity (FNC) (Shannon et al., 2011; the coactivation of various brain regions) within brain regions that comprise the DMN. ECN disturbances, in turn, have also been noted in offenders, primarily with regard to FNC (Jiang et al., 2017).

Given DMN/ECN involvement in self-referential and cognitive control processes, respectively, these DMN and ECN network abnormalities suggest that offenders may present with disruptions in these important cognitive processes.

Much of the work in this area has used a 'targeted analysis approach', which in neuroscience means that they zoned analyses right in on specific brain regions or RSNs (e.g., DMN/ECN). Indeed, fewer studies to date have taken a 'whole-brain analysis approach' that seeks a more bird's eye view of all activity in the brain. Using both approaches is important as an over-focus on specific RSNs may preclude the ability to differentiate network-specific abnormalities from broader abnormalities that are common across all resting-state networks. Two studies have used such a whole-brain approach to compare neural integrity between offenders and non-offenders, albeit within largely adolescent samples. Liu et al. (2014) reported that offenders with antisocial personality disorder showed reduced low-frequency fluctuations compared to 35 healthy controls within the right orbitofrontal cortex, left temporal pole, right inferior temporal gyrus and left cerebellum (Liu et al., 2014). Similarly, Lu et al. (2015) reported that adolescents with conduct disorder displayed decreased connectivity within the DMN, somatosensory network (SMN) and visual networks (Lu et al., 2015). Thus, these studies have generally confirmed DMN-/ ECN-based abnormalities, but have also identified abnormalities within regions not previously hypothesized. This work suggests that extending our search space to the whole brain when investigating the integrity of resting-state networks may elicit useful biomarkers of offending. However, evaluation within an adult sample would be necessary to help provide a more comprehensive portrait of neural functioning and point towards possible neural-markers of offending. It is also important to note that this

work was performed only within subsets of offenders with heightened antisocial traits (Liu et al., 2014; Lu et al., 2015). Thus, these results might not generalize across offenders. Hence, there is a need for whole-brain investigations in adult offenders that can deepen our understanding of the involvement of RSN activity in offending behaviour.

1.1.1. Emotion and cognition related neural functioning in offenders

A wide array of work has investigated cognitive deficits in offenders. Some studies have found deficits in executive functioning, mostly in set-shifting, planning, working memory, inhibition, attention, and problem solving (Meijers et al., 2015). These executive functioning deficits are also associated with neural disruptions. Indeed, Schiffer et al. (2014) indicated that offenders present vast disruptions in brain regions underlying cognitive control. While performing a non-verbal Stroop task, offenders displayed altered neural activity in the anterior cingulate, dorsolateral prefrontal, superior temporal and postcentral cortices, and the putamen, thalamus, and amygdala (Schiffer et al., 2014). Similarly, youth offenders have demonstrated altered performance monitoring, illustrated by behavioural and EEG-related deficits in error processing and response inhibitions (Vila-Ballo et al., 2014). Some studies have found deficits in cognitive domains other than executive functioning. For instance, in a sample of 488 Canadian male offenders, 55% presented cognitive deficits in language, memory, calculations, reasoning and construction domains (Stewart et al., 2016). Additionally, a study investigating 88 145 inmates found that those with lower verbal and math cognitive abilities showed a higher frequency of inmate misconduct while they were imprisoned (Silver & Nedelec, 2018). Thus, various cognitive domains are disrupted in offenders; however, exactly which ones are central to offending remains to be determined.

Work focusing on emotion and empathic abnormalities has also revealed deficits in offenders' emotion processing and underlying neural activity. For instance, Raine and Yang (2006), in their review of the neural deficits associated with moral reasoning in antisocial individuals, theorized that rule-breaking might be related to disruptions in neural regions involved in the development of moral cognition and/or the generation of emotions (i.e., dorsal and ventral prefrontal cortex, amygdala, angular gyrus). Similarly, Baumeister & Lobbestael (2011) suggested that emotions may influence the production of both prosocial and antisocial behaviour, but suggested that this effect may occur indirectly, such that disruptions in emotional processing lead to changes in cognitive processing that in turn direct behavioural outcomes (Baumeister & Lobbestael, 2011). Both theories recognize the importance of cognitive and emotional deficits in antisocial behaviour. However, Yang and Raine (2006) theorized that emotional deficits are more central, while Baumeister & Lobbestael (2011) suggested that only cognitive deficits directly relate to antisocial behaviour. Other work has identified that offenders present deficits in emotion regulation (Umbach et al., 2018), emotion recognition (Umbach et al., 2018), empathy (Domes et al., 2013; Meffert et al., 2013) and the emotional component of morality (Pujol et al., 2012). Neurally, offenders present increased activity in the left amygdala in response to highly emotionally salient pictures (Prehn, Schulze, et al., 2013), decreased response in empathy-related brain regions when spontaneously reacting to emotional stimuli (Meffert et al., 2013), and disruptions in DMN connectivity during moral decision making (Pujol et al., 2012).

Thus, although offenders present neural disruptions and related processing disruptions in both cognitive and emotional processing domains, it is unclear if

disruptions are more prevalent in one domain than the other in offenders. Alternatively, neural disruptions may present across both cognitive and emotion processing deficits in offenders. This topic is further explored in the introduction to Study 3.

1.2. Risk factors of offending

One way to evaluate the risk factors that predict offending behaviour is to consider whether those factors tend to be more 'individual' or 'environmental' in nature. Individual risk factors are factors that pertain to an individuals' personality, genetics or behaviour; they include risk factors such as antisocial disorders (Hemphill et al., 1998; Yu et al., 2012), substances use disorders (DeLisi et al., 2015) and lower intelligence (Schwartz et al., 2015). Environmental risk factors, in contrast, are factors that pertain to an individual's social, familial or societal influences; they include risk factors such as low parental education, poor child-rearing skills and parental discord (Derzon, 2010; Leschied et al., 2008), low socioeconomic status, poor housing and living in a high-crime neighbourhood (Farrington, 2010).

Both individual and environmental risk factors of offending are important to consider; however, this dissertation focuses primarily on individual risk factors - specifically, on personality traits/behaviours that tend to relate to offending behaviours. This focus was based on both practical and theoretical considerations. Theoretically, individual risk factors are well-established to explain considerable variance in offending behaviours. For instance, offending behaviour is associated with higher levels of diagnosed substance abuse disorders and antisocial disorders (i.e., antisocial personality disorder and/or psychopathy) in comparison to non-offenders (DeLisi et al., 2015; Hare, 1998; Hemphill et al., 1998; Innes, 1988; Lester et al., 2003; Makkai & Payne, 2003;

Nurco et al., 1991; Western et al., 2017). Moreover, Psychopathy Checklist-Revised scores (PCL-R; Hare, 2003) (i.e., a measure of psychopathic traits) and lifetime substance use metrics have shown themselves to be associated with criminal behaviour (Dowden & Brown, 2002; Hare et al., 2000; Hemphill et al., 1998). Practically, the dataset that served as the basis for this dissertation, previously acquired at the Mind Research Network (MRN) by Dr. Shane, includes rich data on individual risk factors of offending, but only limited data on environmental risk factors. Thus, while the study of environmental factors may elicit important insights, this dissertation focuses primarily on the relationship between neural integrity and individual risk factors in an offender population.

1.2.1. Psychopathy

Psychopathy is a forensically relevant personality disorder characterized by pervasive antisocial behaviour, deficits in empathy and remorse, as well as boldness, disinhibition and egocentricity (Cleckley, 1951). Offenders with heightened psychopathic traits are three times more likely to re-offend (Hemphill et al., 1998). Given the high correlation between psychopathic traits and offending, it is not surprising that the prison population presents a higher prevalence of psychopathy diagnosis (i.e., 15 to 25%) in comparison to the general population (i.e., less than 1%; Hare, 1998).

The disorder has been commonly separated via factor analysis into two factors, featuring interpersonal and affective traits (such as shallow affect, lack of empathy, grandiose sense of self-worth and glibness), and lifestyle and antisocial traits (such as impulsivity, poor behavioural control, parasitic lifestyle and proneness to boredom; Hare et al., 1990; Harpur et al., 1989). Complicating the diagnosis is its somewhat heterogeneous nature, with individuals reaching forensic threshold for the disorder via

different levels and combinations of these characteristics (Fanti et al., 2018; Hecht et al., 2016). Thus, the definition of the disorder remains somewhat malleable and in some need of further distinction. Moreover, psychopathy is highly comorbid with substance use disorders (Derefinko & Lynam, 2007; Taylor & Lang, 2006) and Antisocial Personality Disorder (ASPD; Dahl, 1998). Indeed, individuals with heightened psychopathic traits tend to present more periods of acute drug use in their lives (Derefinko & Lynam, 2007), tend to start using drugs at a younger age (Derefinko & Lynam, 2007) and show a significantly higher prevalence of comorbid ASPD diagnosis (Werner et al., 2015). However, the relationship between ASPD and psychopathy is somewhat asymmetric, with only few diagnosed with ASPD also meeting the criteria for psychopathy (Hare, 1996). The asymmetry of this comorbidity is attributable to the fact that psychopathy is mainly evaluated in forensic populations, while ASPD is not, that psychopathy and ASPD both share antisocial behaviour in their diagnostic criteria (Werner et al., 2015), and that psychopathic traits differ from ASPD in their evaluation of lack of empathy, inflated self-image and superficial charm (Hare, 1996).

1.2.1.1. Measurement of Psychopathy. The 'gold standard' for diagnosis of psychopathy is the Psychopathy Checklist-Revised (Hare, 2003). The PCL-R presents strong risk assessment capabilities (Hare, 2003; Hemphill et al., 1998; Olver & Wong, 2015; Salekin et al., 1996; Serin, 1996; Walters, 2003), which have been consistently demonstrated in many North American and European samples (Grann et al., 1999; Hare et al., 2000). In these samples, PCL-R scores have been successfully used to assess risk probabilities in offenders at the start of their prison sentence and in the context of probation and parole proceedings. It is similarly used for risk prediction in psychiatric

patients (Salekin et al., 1996). It is an interview-based diagnostic assessment that evaluates 20 traits (Hare, 2003; Hare & Neumann, 2005). The diagnosis is usually made categorially, with individuals scoring above 30 (in North America) or 25 (in the UK) meeting the criteria for the disorder. However, PCL-R scores, a composite of Factor 1 (interpersonal and affective traits) and Factor 2 (lifestyle and antisocial traits) scores, are often used continuously to reflect the full spectrum of symptom variance in research contexts. The PCL-R has strong psychometric properties, tends to be quite stable across the lifespan (Hare et al., 1990; Harpur et al., 1988, 1989) and has good interrater and test-retest reliability (Forth et al., 1996; Hare, 2003; Hare et al., 1990). Its capacity to predict re-offending is similar to or better than that of other risk assessment tools (Gendreau et al., 1996; Salekin et al., 1996).

1.2.1.2. Neural disruptions associated with psychopathy. Numerous studies have investigated rest-related neural disruptions associated with psychopathy. This work indicates that individuals with heightened psychopathy traits present decreased DMN connectivity during rest (Philippi et al., 2015; Pujol et al., 2012), as well as weaker functional connectivity in the amygdala and insula (Espinoza et al., 2019) and disruptions in FNC between the DMN and ECN networks (Dotterer et al., 2020).

There is also a rich literature on the task-related neural deficits associated with heightened psychopathic traits in offenders. Most notably, two meta-analyses have aimed to identify neural activity associated with heightened psychopathic traits (Deming & Koenigs, 2020; Poeppl et al., 2019). Both studies reported an association between psychopathic traits and activity in various cortical and subcortical regions, including the bilateral lateral prefrontal cortex, fronto-insular cortex (Poeppl et al., 2019), posterior

cingulate, precuneus, and dorsal anterior cingulate cortex (Deming & Koenigs, 2020). Of particular note, both meta-analyses also uncovered a relationship between heightened psychopathic traits and activity in the amygdala and the dorsomedial prefrontal cortex. However, while Poeppl et al. (2019) identified a negative relationship between heightened psychopathic traits and activity in these regions, Deming & Koenigs (2020) identified a positive relationship. Despite the inconsistency regarding the directionality of these effects, these meta-analytic studies suggest that heightened psychopathic traits are most consistently related to activity disruptions in the prefrontal cortex and the amygdala (Blair, 2010; Deming & Koenigs, 2020; Poeppl et al., 2019).

1.2.2. Substance use disorders

A high proportion of offenders present with a wide variety of substance use disorders (DeLisi et al., 2015; Innes, 1988; Kouri et al., 1997; Makkai & Payne, 2003; Nurco et al., 1991; Peters et al., 1998). Lifetime prevalence rates of substance dependence in offending populations may be as high as 74% (Peters et al., 1998) or 95% (Kouri et al., 1997). Moreover, between 39% (Makkai & Payne, 2003) and 58% (Kouri et al., 1997) of inmates report that drug use at the time of their offence contributed to the criminal behaviour for which they were incarcerated. Similarly, a survey from 1986 showed that around 43% of offenders had used illegal substances, while 19% stated that they had used major drugs (e.g., cocaine, heroin) daily or almost daily prior to the offence they were incarcerated for (Innes, 1988).

Research consistently demonstrates a clear relationship between the severity of substance use and the propensity to commit crimes (DeLisi et al., 2015; Innes, 1988; Makkai & Payne, 2003). DeLisi et al. (2015) categorized offenders in three clusters based

on their level of substance use diagnoses: offenders with limited substance or no substance use disorders (i.e., used alcohol or marijuana but not more serious types of drugs; 62.70%), offenders with comorbid alcohol and marijuana use disorders (28.80%), and offenders with polydrug abuse and dependence (8.52%). The severity and frequency of crimes committed by the offenders were directly related to their drug use, with offenders with limited/no substance use disorders having committed mostly a few petty crimes and offenders with polydrug diagnoses having committed more numerous and severe crimes (DeLisi et al., 2015). This coincides with additional work demonstrating that the chances of incarceration and re-offence are highest for polydrug users (e.g., Sweeney & Payne, 2011).

1.2.2.1. Neural disruptions associated with substance use disorders. Substance use disorders are well-established to impart long-lasting changes to neural integrity (e.g., Barrós-Loscertales et al., 2011; Cisler et al., 2013; Ma et al., 2015; Beard et al., 2019). Indeed, cocaine use has been associated with decreased grey matter volumes in the striatum and the supramarginal gyrus (Barrós-Loscertales et al., 2011) and increased FNC between the right insula and elements of the pre-frontal and frontal cortices (Cisler et al., 2013) in comparison to non-dependent individuals. Moreover, increased years of cocaine use are directly associated with decreased grey matter volume in the cortico-striatal system in cocaine-dependent individuals (Barrós-Loscertales et al., 2011).

Additionally, a growing body of work has begun reporting power spectra disturbances within substance-dependent populations (Ide et al., 2016; Jiang et al., 2011; Wang et al., 2013), including evidence of increased thalamic *fractional amplitude of low-frequency fluctuation* (fALFF; a measure of low-frequency activity; Ide et al., 2014) and

decreased power spectrum scale invariance (i.e., weaker autocorrelation and signal persistence) in various frontoparietal regions (Ide et al., 2016) in cocaine-dependent individuals. Similar results have been reported in heroin-dependent individuals within various frontal, temporal, occipital and parietal regions (Jiang et al., 2011; Wang et al., 2013). However, it should be noted that the vast majority of this work has not been conducted within an offender population.

Finally, one study has investigated the relationship between substance use and power spectra integrity in offenders. This study investigated the contribution of alcohol and cannabis use to resting-state activity within various RSNs within a youth offender sample (Thijssen et al., 2017). Results demonstrated that increased cannabis use was related to decreased low-frequency power spectra in the DMN, ECN and several sensory networks and decreased FNC between several networks. In contrast, increased alcohol use was related to decreased low-frequency power spectra in the right frontoparietal, salience and dorsal attention networks as well as several sensory networks (Thijssen et al., 2017). However, this work was performed in a juvenile offender sample and might not generalize to the adult offender population. Thus, important gaps remain regarding the extent to which dependence status underpins spectral abnormalities in the adult offender population.

1.2.3. Handling the comorbidity between psychopathy and substance use disorders

One complication in evaluating the influence of psychopathy and substance use disorders on offending behaviour is that the two constructs are highly comorbid (Derefinko & Lynam, 2007; Taylor & Lang, 2006). This comorbidity makes it difficult to determine if abnormalities in neural systems are due to increases in substance use or

psychopathic traits (or a combination of both). It could even be that psychopathic traits and substance use affect specific subsets of offenders differently. Research evaluating this comorbidity remains nascent but may hold important implications for our understanding of the nature of both disorders. Some work suggests that psychopathic traits modulate neural response in substance-dependent offenders. Indeed, a study by Cope et al. (2014) investigated how psychopathic traits modulate neural responses related to cue-elicited drug craving within substance-dependent offenders. Results indicated that psychopathic traits negatively influence activity in brain regions involved in drug craving responses (Cope et al., 2014). However, other work suggests that this comorbidity might only be present in a specific subset of offenders. To provide a finer-grained assessment of the behavioural relationship between substance use and psychopathic traits, a study used K-means clustering to categorize inmates into 4 clusters based on their PCL-R factor scores, drug abuse and dependence diagnosis (Vassileva et al., 2005). Offenders presenting with the most severe drug and alcohol issues also presented the highest factor 2 scores on the PCL-R. Another cluster presented low PCL-R scores, low anxiety, and high drug and alcohol abuse levels, but not dependence. The third cluster presented higher Factor 1 scores, lower anxiety and less severe alcohol and drug use. Finally, the last cluster did not display any alcohol use disorder and displayed less severe drug use, average Factor 1 and Factor 2 scores and very low anxiety. Thus, not all clusters of offenders presented equal comorbidity between substance use disorders and psychopathic traits, highlighting the complex relationship between these two constructs. Additionally, recent work by our laboratory investigated the neural response of drug-dependent and non-drug-dependent offenders while viewing videos eliciting either drug or food cravings

(Denomme et al., 2018). Drug-dependent offenders presented more reactivity to drug stimuli than food stimuli in various neural regions. In contrast, non-dependent offenders presented increased reactivity to food stimuli compared to drug stimuli (Denomme et al., 2018). Moreover, offenders with heightened psychopathic traits showed a negative relationship between neural reactivity to drug versus food stimuli and lifetime drug use, whereas individuals with lower psychopathic traits showed a positive relationship (Denomme et al., 2018). Taken together, this evidence shows just how complicated the comorbidity patterns between psychopathy and substance use are. The relationship between these two risk-factors of offending should thus be further investigated.

1.2.4. Criminal history

Another characteristic of the offender to consider is the heterogeneous nature of their criminal history, which can vary with regard to both the type and number of crimes committed. Regarding type of crime, 55.5% of American offenders were imprisoned in 2019 for having committed violent offences (e.g., murder, robbery), while 16% had committed property crimes (e.g., burglary, fraud), 14.1% drug-related crimes and 12.3% public order crimes (Carson et al., 2020). Research suggests that the individuals who commit each of these crime types may differ in important ways. Violent and non-violent offenders present differing cognitive (Hoaken et al., 2007; Meijers et al., 2017), intellectual (Ttofi et al., 2016) and personality (Walker & Gudjonsson, 2006) profiles, with violent offenders tending to be more impulsive, less empathic (Nussbaum et al., 2002) and lower in IQ (Ttofi et al., 2016). Regarding the number of crimes committed, this has been shown to be one of the strongest predictors of future crimes (Phillips et al., 2005). Indeed, most crimes are committed by a small proportion of offenders, making

them so-called ‘prolific offenders’ (Falk et al., 2014). Public policies often target these prolific offenders to decrease offending rates (Machin & Marie, 2007; Mawby & Worrall, 2004). Gaining a better understanding of the neural integrity of those who commit more crimes could help prevent crime and decrease offending rates.

1.2.4.1 Neural disruptions associated with type and number of crimes committed. A small but developing literature has begun to report on neural differences associated with specific types of crimes in offenders. However, very little remains known about brain structure/function differences associated with different criminal acts. The most commonly reported comparison may be between violent and non-violent offenders, where a number of studies have suggested differences in structure/function. For instance, one study reported increased activity in the amygdala and the striatum in violent offenders in reaction to provocation (da Cunha-Bang et al., 2017), while another study reported increased grey matter volume in the amygdala, nucleus accumbens and caudate nucleus, and decreased grey matter in the insula (Schiffer et al., 2011). However, a variety of studies have also investigated more specific crime types. For instance, some work has reported that pedophilic offenders present aberrant functional connectivity in neural areas associated with sexual arousal (Poeppl et al., 2015; but see Polisois-Keating & Joyal, 2013). Moreover, sexual abusers of children (but not pedophiles) have been shown to present with decreased connectivity between the amygdala, the dorsolateral prefrontal cortex (Kärgel et al., 2015; Kneer et al., 2019), and the orbitofrontal cortex (Kärgel et al., 2015). Other work has focused specifically on homicidal offenders. This literature is sparse; however, one study suggested that offenders who committed homicide in adulthood display decreased grey matter volume in prefrontal, temporal,

insular, parietal and cerebellar regions compared to offenders who did not commit homicide (Sajous-Turner et al., 2019), while another study found that offenders who have committed homicide as juveniles may present with decreased grey matter volume in the medial and temporal lobes (Cope, Ermer, et al., 2014). A handful of studies have also focused specifically on women batterers. This literature has indicated that women batterers display a) increased activity in the cingulate cortices and middle prefrontal cortex, and decreased activity in the superior prefrontal cortex when observing images of interpersonal violence (Bueso-Izquierdo et al., 2016), b) reduced DMN engagement when making moral decisions in intimate partner violent scenarios (Marín-Morales et al., 2020), c) increased activity in the hippocampus, fusiform gyrus, posterior cingulate gyrus, thalamus and occipital cortex in response to threat, and d) increased activity in bilateral precuneus when viewing images of aggression against women (Lee et al., 2009). Finally, a number of studies have investigated the neural integrity of white-collar criminals. In one study, white-collar criminals (compared to non-offenders) presented increased cortical thickness in the ventromedial prefrontal cortex, inferior frontal gyrus somatosensory cortex and temporal-parietal junction (Raine et al., 2012). In another study, having committed more severe white-collar crimes (e.g., embezzlement, forgery) was associated with increased gray matter volume in the frontal lobe (Ling et al., 2019). Thus, a variety of neural disruptions appear associated with having committed specific types of crimes, including crimes of a violent or sexual nature.

Work investigating relationships between the number of crimes committed and neural integrity in offenders is sparse. However, what little work exists suggests that those who commit more antisocial behaviours tend to show more significant functional and

structural neural disruptions. For instance, one study conducted among non-offenders showed that individuals who report committing more criminal behaviour show increased activity in parietal regions, insula, anterior cingulate cortex, right supramarginal gyrus and right angular gyrus when making risky choices (Reyna et al., 2018). In another study, Hyde and colleagues investigated amygdala reactivity in response to emotional faces in a group of low-income urban men. They identified that participants who self-reported having been arrested more times presented a heightened amygdala activity when presented with fearful and angry faces (Hyde et al., 2016). Thus, previous work has demonstrated a relationship between the quantity of antisocial behaviour and aberrant task-related activity (Hyde et al., 2016; Reyna et al., 2018).

1.2.5. Inter-relationship Between Risk Factors of Offending

Of import, variation in the quantity and nature of crimes reported in offenders may also relate to their level of psychopathic traits and substance use. For instance, offenders with higher psychopathic traits tend to commit different types of crimes and a greater number of crimes compared to offenders with lower psychopathic traits (Williamson et al., 1987). Indeed, individuals with heightened psychopathic traits are more likely to commit violent assaults and property crimes, to not be a stranger to their victim and to be more motivated to commit a crime out of monetary gain than emotional arousal, in comparison to those with lower psychopathic traits (Williamson et al., 1987). Similarly, increased substance use has been shown to be more predictive of future violent crimes than non-violent crimes (although all models were strong predictors; Dowden & Brown, 2002). Moreover, inmates with polydrug disorders tend to commit more severe crimes than those without polydrug disorders (Bennett & Holloway, 2005; DeLisi et al.,

2015). Finally, higher lifetime use of more harmful drugs, such as crack, is associated with higher odds of committing crimes than the use of recreational drugs, such as cannabis (Bennett et al., 2008; Innes, 1988; Nurco et al., 1991), suggesting a relationship between the type of drug used and criminal behaviour. Consequently, it is important to consider the concurrent effects of the quantity and quality of criminal history, psychopathic traits and substance use disorders when investigating the risk factors of offending as this relationship could potentially relate to underlying neural mechanisms in offenders.

1.3. Research Aims

As noted above, several important knowledge gaps remain regarding our understanding of the neural underpinnings of offending. First, it remains unclear how resting-state baseline neural activity or task-based neural activity differ across adult offenders compared to non-offenders. To this end, Studies 1 and 2 compared whole-brain resting-state power spectra between offenders and non-offenders, while Study 3 performed a meta-analysis of task-based neural activity differences between offenders and non-offenders. Second, work investigating neural activity specific to subgroups of offenders compared to non-offenders remains sparse, and this literature needs consolidation. In Study 1, those subgroups were constructed based on the presence or absence of cocaine substance use disorders. In Study 2, those subgroups were constructed based on crime type. In study 3, I undertook a meta-analysis that looked at subgroup differences in task-based neural activity based on crime and task paradigm types (i.e., cognitive/emotional). Thus, with this dissertation, I aim to better understand the neural markers of offending and inquire into whether this altered neural activity is common

across offenders or specific to certain subtypes of offenders. Moreover, I aim to assess the degree to which these aberrant neural processes are influenced by trait-based (i.e., psychopathy traits, drug dependence status) and lifestyle (i.e., criminal history, drug use) components of individual risk-factors of offending.

Chapter 2: Study 1

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Altered power spectra in antisocial males during rest as a function of cocaine dependence: A network analysis

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ABSTRACT

Abnormalities in the spectral power of offenders' neural oscillations have been noted within select Resting-State Networks (RSNs); however, no study has yet evaluated the influence of cocaine dependence, drug use severity, and psychopathic traits on these abnormalities. To this end, the present study compared rest-related power spectral characteristics between two groups of offenders (with and without a DSM-IV-TR cocaine-dependence diagnosis) and a non-offender control group. Results indicated that both offender groups presented with lower *low frequency power ratio* (LFPR) scores (i.e. across all RSNs) than non-offenders. These differences in LFPR scores were due to both higher high-frequency power (0.15-0.25 Hz; within seven (in non-dependent offenders) and five (in cocaine-dependent offenders) of eight investigated networks) and decreased low-frequency power (0.01-0.10 Hz; within six (in non-dependent offenders) and one (in cocaine-dependent offenders) of eight investigated networks) compared to non-offenders. Thus, both cocaine-dependent and non-dependent offenders displayed abnormal neural oscillations, suggesting that these oscillatory abnormalities could exist as neurobiological features associated with offender status. Offenders' LFPR levels correlated with lifetime years of cocaine use, but not with the level of psychopathic traits. These findings supplement our knowledge regarding the influence of substance use on resting-state activity in offenders; moreover, they provide further indication of the importance of evaluating shared/unique variance associated with drug use and pyschopathic personality traits.

1. Introduction

The spectral dynamics of resting-state networks (RSNs), which can be assessed through the evaluation of frequency oscillations across the power spectrum, have been hypothesized to underlie the synchronicity of rest-related activity in RSNs (Baria et al., 2011; Thompson & Fransson, 2015; Yaesoubi et al., 2017), and to provide generalized insights into the development and maintenance of healthy brain function (Pizoli et al., 2011). A typical normalized resting-state spectral profile consists of peak activity within low-frequencies (i.e. $<$ 0.10 Hz), with sharply decreased activity occurring within mid- (i.e. 0.10-0.149 Hz), and particularly within high (i.e. 0.15-0.25 Hz; Allen et al., 2011; Kalcher et al., 2014) frequencies. A higher proportion of low-frequency activity at rest is believed indicative of a more stable, functional network (Biswal et al., 1996), whereas a higher proportion of high-frequency activity at rest has been linked to instability of network activity (Buzsáki & Draguhn, 2004; Salvador et al., 2008; but see DeRamus et al., 2020). Consistent with this view, considerable work has associated decreased low-frequency activity (Cauda et al., 2009; Garrity et al., 2007; Han et al., 2011; Malinen et al., 2010; Wang et al., 2016; Xu et al., 2014; Yu et al., 2014) and/or increased high-frequency activity (Cauda et al., 2009; Malinen et al., 2010; Otti et al., 2013; Sambataro et al., 2017) with numerous neurological and psychiatric disorders, including schizophrenia, chronic pain, bipolar disorder, major depressive disorder and amnestic mild cognitive impairment. The consistency and breadth of this body of work suggests that aberrant spectral patterns may serve as a common underlying feature of a wide variety of psychiatric disorders.

To date, only a handful of studies have reported on spectral dynamics within offender populations, and that work has almost exclusively been conducted within adolescent samples (Liu et al., 2014; Thijssen et al., 2017; Zhou et al., 2015). Zhou and colleagues (2015) reported decreased *fractional amplitude of low frequency fluctuation* (fALFF) within adolescent offenders' bilateral amygdala/hypothalamus, right lingual gyrus, left cuneus and right insula. More recently, Thijssen and colleagues (2017) demonstrated that more prolonged cannabis use related to decreased low-frequency power within the Default-Mode Network

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(DMN), Executive Control Network (ECN) and sensory networks of adolescent offenders (more extended history of alcohol use also related to decreased low-frequency power within the right frontoparietal, salience, dorsal attention and sensory networks). Finally, the only study to have investigated spectral abnormalities within an adult offender sample to date (though the mean age of the sample was still only 20; Liu et al., 2014) reported reduced low-frequency fluctuations within the right orbitofrontal cortex, left temporal pole, right inferior temporal gyrus and left cerebellum of 32 offenders with antisocial personality disorder compared to 35 healthy controls. The consistency of these findings does provide preliminary support for the existence of spectral abnormalities in offending populations. However, the relative sparseness of the field, the small sample sizes employed, and the predominant focus on adolescent offenders, does limit the ability to form firm conclusions.

One distinct possibility is that offenders' osscilatory abnormalities are due to their generally higher levels of substance use disorders (SUD), which can reach prevalence rates as high as 74% (Peters et al., 1998) or even 95% (Kouri et al., 1997), and which are well-established to impart long-lasting changes to neural integrity (e.g. Barrós-Loscertales et al., 2011; Cisler et al., 2013; Ma et al., 2015; Beard et al., 2019). Indeed, a growing body of work has begun reporting spectral disturbances within substance-dependent populations (Ide et al., 2016; Jiang et al., 2011; Wang et al., 2013), including evidence of increased thalamic fALFF (Ide et al., 2014) and decreased power spectrum scale invariance (i.e. weaker autocorrelation and signal persistence) in various frontoparietal regions (Ide et al., 2016) in cocaine dependent individuals. Similar results have been reported in heroin-dependent individuals within a variety of frontal, temporal, occipital and parietal regions (Jiang et al., 2011; Wang et al., 2013). It should be noted, however, that the vast majority of this work has not been conducted within offender populations. Thus, important gaps remain regarding the extent to which dependence status underpins spectral abnormalities in offenders.

To this end, the present study sought to evaluate the integrity of resting-state power spectral dynamics within two groups of offenders (those who do and do not meet DSM-IV diagnosis for cocaine-dependence) and within a control group without offending behavior. Resting-state networks were identified using Independent Component Analysis (ICA), and power spectral dynamics were evaluated within and between the three study groups. Based on the handful of studies conducted within offender samples, and on work conducted to date within substance-using populations, we predicted decreased low-frequency activity and increased high-frequency activity (resulting in decreased *low frequency power ratio* (LFPR; the ratio of the integral of low-frequency activity to the integral of high-frequency activity, see Allen et al., 2011) in both offender groups compared to the non-offender controls. Identifying such power spectral disruptions in offenders could indicate incoherence in baseline neural activity in this population. Additionally, we sought to explore the extent to which network-related disruptions in offenders were characteristic of all RSNs, or were instead specific to only certain resting-state networks. To this end, exploratory analyses compared/contrasted oscillatory patterns within each identified RSN, to evaluate for the existence of global versus network-specific effects.

Another possibility is that offenders' abnormal spectral patterns relate to their generally higher levels of antisocial traits, which have also been linked to substantive neural abnormalities, including decreased amygdalar and frontal activity (Blair, 2010; Poeppl et al., 2019; Umbach et al., 2015), as well as increased fronto-insular cortex activity (Poeppl et al., 2019). As a complicating factor, antisociality and substance abuse are themselves highly comorbidity (as high as 90% comorbidity by some estimates; Smith & Newman, 1990), making parcellation of SUD/antisociality distinctions extremely difficult. As this parcellation is a particular focus of work in our laboratory, we also conducted subsequent exploratory regression analyses to evaluate the extent to which SUD participants' spectral patterns could be explained as a function of either their drug-use severity or their psychopathic personality. To our knowledge, no work has yet investigated these issues as they pertain to spectral power in adult offenders; thus, while exploratory, results may provide preliminary insights that can help guide future progress in this underrepresented area.

2. Methods

2.1. Participants

One hundred and twelve right-handed male participants were recruited as part of a larger project focused on identifying various biomarkers of cocaine use disorders. Offenders were recruited through active recruitment at New Mexico probation/parole offices, temporary employment agencies, alcohol/drug treatment centers, and through print/online ads. Non-offenders were recruited solely through print/online ads. Offenders met initial screening criteria if they were on probation/parole, were 18-55 years of age, had a felony-level conviction history, did not self-report psychotic disorders or use of antipsychotic medications, and matched fMRI acquisition criteria (e.g. no metal in the body, no history of major head trauma, no pregnancy, right-handedness). Subsequent screening excluded for lifetime history of a psychotic disorder (one exclusion), major depressive disorder within the last six months, current use of antipsychotic medications (9 exclusions), and full IQ estimates < 70. Non-offenders were matched on important demographic variables to the extent possible, but self-reported having no criminal record. Following all screening mechanisms, 102 participants were included in final analyses (37 Cocaine-Dependent, 47 Non-Dependent and 18 non-offenders; see Table 1 for full demographics). This study was approved by the Research Ethics Board of the University of New Mexico. Participants provided written informed consent in accordance with the Code of Ethics of the World Medical Association (Declaration of Helsinki).

2.2. Clinical/Forensic assessments

2.2.1. SCID-I/P

The Structured Clinical Interview for DSM-IV-TR (SCID-I/P; First, Spitzer, Gibbon, & Williams, 2002) was used to evaluate participants for Axis I and II disorders. Interviews were videotaped and conducted by trained Master's level research personnel, under the guidance of a senior SCID trainer (R.C.; see acknowledgements).

2.2.2. Psychopathic traits

Offenders were assessed for psychopathic traits via a Psychopathy Checklist-Revised (PCL-R) interview (Hare, 2003), which were videotaped and conducted by trained research personnel (trained by M.S.). Subsequent file reviews were not possible; thus, participants were scored 0 to 2 on each of the 20 PCL-R items based on the clinical interview alone (see Denomme et al., 2018, 2020; Forth et al., 1996; Kosson et al., 1997 for evidence of the validity of this approach).

2.2.3. Cocaine use

The number of years of regular drug use was evaluated using a modified Addiction Severity Index - Expanded (ASI-X; McLellan et al., 1992), which was administered orally by a trained examiner. As offenders were recruited based on cocaine dependence diagnoses, total years of lifetime cocaine use (LCU) was included as a covariate in relevant analyses.

2.2.4. Other Drug use

Following administration of the ASI-X, composite scores of total drug use (other than cocaine) were calculated by summing the total years of use of drugs that corresponded to two categories: 'major drugs' (i.e. methamphetamines, opiates/analgesics, heroin) and 'minor drugs' (i.e. alcohol, cannabis, nicotine, hallucinogens and inhalants). Thus, for

Table 1
Participant demographics.

	O (N = 84)	DO (N = 37)		NDO (N = 47)		NO (N = 18)	Group differences (t scores)			
	Mean	N	*Mean*	N	*Mean*	*Mean*	O vs NO	DO vs NO	NDO vs NO	DO vs NDO
	Range		Range		Range	Range				
	SD		*SD*		*SD*	*SD*				
Age	33.44		35.11		32.13	30.87	2.63*	3.32*	1.90	-1.52
	20-59		22-56		20-59	18-50				
	8.97		*8.36*		*9.30*	*9.27*				
IQ	105.76		106.03		105.55	112.60	-1.99*	-1.77	-1.93	-0.18
	77-137		80-137		77-131	89-131				
	11.84		*11.74*		*12.04*	*12.50*				
ASI-X										
LCU	3.35		7		0.47	0.33	4.48**	5.89**	0.31	-5.82**
	0-24		0-24		0-10	0-5				
	5.61		*6.66*		*1.67*	*1.29*				
Major Drugs	4.51		6.38		3.04					1.58
	0-31		0-31		0-29					
	6.87		*7.24*		*6.26*					
Minor Drugs	21.55		27.05		17.21					1.05
	0-74		1-61		0-74					
	16.25		*14.82*		*16.15*					
PCL-R										
Total	20.79		23.19		18.90					-2.76*
	4-34		12-34		4-34					
	7.34		*6.57*		*7.43*					
Factor 1	7.32		8.09		6.71					-1.81
	2-15		2-14		2-15					
	3.51		*3.21*		*3.64*					
Factor 2	12.09		13.38		11.08					-2.66*
	2-20		3-20		2-19					
	4.09		*3.83*		*4.05*					
SCID										
GAD		1		1						
MDD		7		4						

Demographic data on age, IQ, lifetime cocaine use (LCU), major drug use (combining years of use of methamphetamines, opiates/analgesics, heroin), minor drug use (combining year of use of alcohol, cannabis, nicotine, hallucinogens and inhalants), PCL-R scores, and SCID diagnosis (i.e. General Anxiety Disorder (GAD) and Major Depression Disorder (MDD)). Demographics are reported separately for Offenders (O), Dependent Offenders (DO), Non-Dependent Offenders (NDO) and Non-Offenders (NO). Group differences were evaluated via between-group t-tests (two-tailed).

* $p < 0.05$

** $p < 0.001$

example, if a participant used cannabis for 3 years, hallucinogens for 5 years and nicotine for 5 years, their minor drug use score was calculated as 13 years. In addition, a 'total drug use' score was calculated as the sum of major + minor drug use. These drug use scores were included as covariates in supplementary regression analyses, to examine whether reported results were specific to cocaine use, or generalized to other drugs of abuse.

2.2.5. WAIS-III

Participants' full-scale intelligence quotients were approximated using the vocabulary and matrix reasoning scales of the Wechsler Adult Intelligence Scale 3rd edition (WAIS-III).

2.3. fMRI Data acquisition

Resting-state data was acquired using a 3T Magnetom Trio Tim Siemens scanner. Participants kept their eyes open and directed at a fixation cross. The 5.5 minutes acquisition was performed using a fast gradient-echo EPI sequence. Imaging parameters were as follows: TR = 2000ms, TE = 29ms, FA = 75°, matrix = 63 × 63, slices = 33 acquired in an ascending interleaved fashion, slice thickness = 3.5 mm, FOV = 240mm x 240mm and voxel size = 3.8 × 3.8 × 3.5mm.

2.4. fMRI Preprocessing

Resting-state data was preprocessed using SPM12. Data was motion-corrected using INRIAlign (Freire et al., 2002). Realignment was handled using a distance cut-off of 2.5, with data coregistered with a full-width-as-half-maximum (FWHM) of 8mm. Slice timing correction was applied using the 16th slice as reference. Data was then normalized using the SPM5 EPI template; the mean image was used for parameter estimation. Finally, data was smoothed using a 10mm Gaussian kernel. No participant presented movement above 5mm. Hence, after pre-processing, all participants were included in analyses.

2.5. Independent Component Analysis (ICA)

Following preprocessing, Independent Component Analysis (ICA) was performed on all 102 participants using the GIFT toolbox (GroupICAT v4.0b, https://trendscenter.org/software/) following well-established methodology (Allen et al., 2011). The spatial group ICA (GICA) was set to identify a high number of components (75) using the Infomax algorithm, and repeated in Icasso 20 times to ensure stability of the estimation. Two GICA back-reconstruction steps were performed to produce subject-specific spatial maps (SMs) and time courses (TCs); results were subsequently scaled to z-scores.

The ICA analysis yielded 75 components, constructed by grouping individual voxels that were synchronously activated during rest. These components were manually classified into two groups: resting state and noise. Components were classified into resting-state based on the methodology recommended by Allen et al. (2011): component t-maps showed activity in grey matter areas, activity peaks between 0.01-0.08 Hz followed by flat activity in high frequencies, a low to high-frequency ratio higher than 4, and a cluster stability index (I_q) of at least 0.80. Components displaying spectral activity non-typical of resting-state networks (i.e. activity outside of grey matter, a flat power

spectrum, or power spectrum with an activity peak in high-frequency) were classified as noise. Following this component selection process, 34 components were classified as representing valid BOLD activity. One-sample t-tests were performed using SPMstats in GIFT to create SPM12 compatible t-maps. Activity peaks in each component were visually inspected, identified using PickAtlas 3.0.5 (Maldjian et al., 2003), matched with key regions of known resting-state networks using the 90 fROIs Atlas (Shirer et al., 2012), and classified into one of DMN, ECN, Posterior Salience (PS), Basal Ganglia (BG), Sensorimotor (SMN), Language (LG), Visual, or Auditory (AU) networks (see Figure 1).

2.6. Spectral analyses

Power spectra were computed for each participants' RSNs using spectral group comparison analyses in GIFT. Following widely used process for categorizing frequency bands (Allen et al., 2011; Bryant et al., 2016), power spectra data were pooled into three bins representing low- (< 0.10 Hz), mid- (0.10 to 0.149 Hz) and high-frequency (0.15 to 0.25 Hz) activity. Analyses focused exclusively on the low and high frequency bands, as both the function and dynamics of mid-frequency activity are significantly less characterized in the broader neuroscientific literature. Spectral data for each participant was then extracted for further analysis in SPSS 22.

2.7. Data analytic strategy

A 3 (Group) x 2 (Bins) x 8 (Network) omnibus ANOVA was used to evaluate higher-order effects, followed by targeted comparisons used to evaluate specific hypothesized effects. This analytic strategy provided the greatest granularity with regard to the nature of spectral distributions in offenders, and thus served as our primary analytic pipeline. However, because the literature tends to focus predominantly on low-frequency centered metrics (e.g. ALFF, fALFF, LFPR), we also report differences in LFPR scores, which have previously been found predictive in clinical settings (e.g. see Yu et al., 2013).

In addition, hierarchical regression models were employed using interview-based PCL-R scores, LCU, and their interaction as regressors to explore the potential influence of psychopathic traits and lifetime cocaine use on power spectra integrity. An additional model also included lifetime use of other substances as a covariate in the model, which confirmed that reported effects were specific to lifetime cocaine burden rather than to the burden of other comorbid substances (see supplementary material Sections 7 to 12).

3. Results

3.1. Demographics

As can be seen in Table 1, rates of comorbid psychiatric disorders were generally low, and chi-square analyses confirmed no prevalence differences in either GAD or MDD between offender groups, χs = GAD: 1.97; MDD: 0.03). Interview-based PCL-R scores (M = 20.79) were closely in line with previously published norms collected through interview + file review methods in offender populations (Hare, 2003).

One-way ANOVA identified group differences in LCU, ($F = 28.79$, $p < 0.001$), with the Dependent group ($M = 7.00$, $SD = 6.66$) showing higher LCU than either the Non-Dependent ($M = 0.47$, $SD = 1.67$; $t = -5.82$, $p < 0.001$) or Non-Offender ($M = 0.33$, $SD = 1.29$; $t = 5.89$, $p < 0.001$) groups. As expected, the Dependent group ($M = 23.19$, $SD = 6.57$) also manifested higher PCL-R scores than the Non-Dependent group ($M = 18.90$, $SD = 7.43$; $t = -2.76$, $p < 0.05$; Non-Offenders did not receive the PCL-R interview). No main effect of IQ was identified; however, the Dependent group ($M = 35.11$, $SD = 8.36$) was somewhat older than the Non-Dependent ($M = 32.13$, $SD = 9.3$; $t = -1.52$, $p > 0.05$), and Non-Offender ($M = 30.87$, $SD = 9.27$; $t = 3.32$, $p < 0.05$) groups. Analyses reported within were reported without controlling for age, as the well-established collinearity between age and LCU (Wagner & Anthony, 2002) may influence validity/interpretability of age-controlled models. However, we also provide age-controlled results in Section 1 of Supplementary Materials.

Correlational analyses indicated that PCL-R scores were negatively correlated with IQ ($r = -0.26$, $p = 0.02$), and uncorrelated with age ($r =$

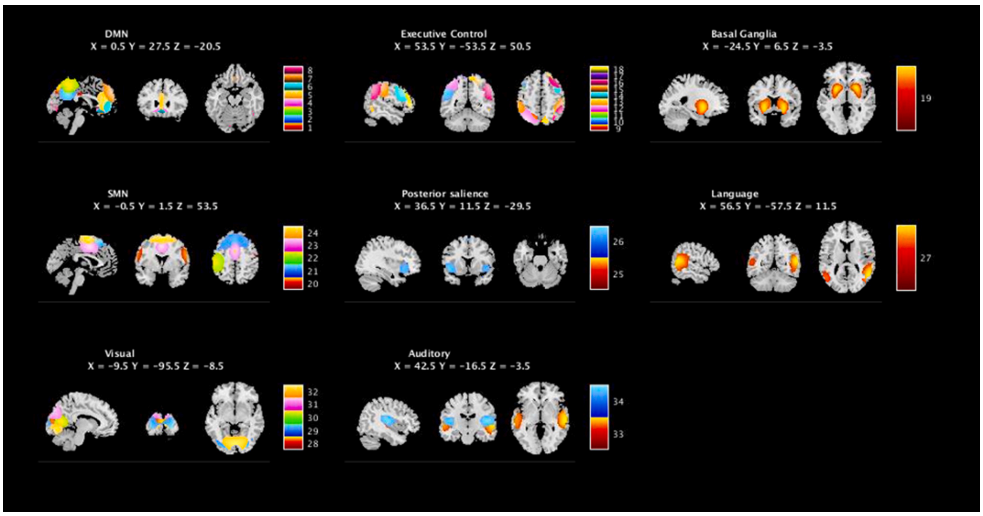

Fig. 1. Network classification of valid resting-state components. Resting-state component classification yielded 34 components of interest. RSN classification, using the peak activity of each component, was categorized into 8 RSNs: Default mode (DMN), Executive Control (ECN), Basal Ganglia, Sensorimotor (SMN), Posterior Salience, Language, Visual and Auditory networks. p(FWE) < 0.05

-0.01, $p = 0.95$), whereas LCU was positively correlated with age ($r = 0.45$, $p < 0.001$), but uncorrelated with IQ ($r = -0.11$, $p = 0.27$). LCU and PCL-R scores showed a modest positive relationship ($r = 0.26$, $p = 0.02$).

3.2. Spectra analyses

The omnibus ANOVA identified a main effect of Group, $F(2, 99) = 9.92$, $p < 0.001$, with pairwise comparisons indicating that Dependent ($M = 0.83$; $SD = 0.05$) and Non-Dependent ($M = 0.83$; $SD = 0.05$) groups showed similar overall power spectra, $p = 1.00$, that were both higher than Non-Offenders ($M = 0.79$; $SD = 0.07$; both $ps < .05$). Main effects of Network, $F(7, 99) = 8.13$, $p < 0.001$, and Bin $F(1, 99) = 1116.57$, $p < 0.001$, were also observed, with the low frequency bin ($M = 1.15$; $SD = 0.08$) showing higher overall power than the high-frequency bin ($M = 0.56$; $SD = 0.10$). These main effects were influenced by significant Group x Bin, $F(2, 99) = 6.19$, $p = 0.003$, and Network x Bin, $F(7, 99) = 13.20$, $p < 0.001$, interactions. The Group x Network, $F(14, 99) = 1.39$, $p = 0.15$, and Group x Bin x Network, $F(14, 99) = 0.94$, $p = 0.63$, interactions did not reach significance.

As no interactions with Network were found, we collapsed mean spectral values across all networks and evaluated for group differences in low-/high-frequency bins via Bonferroni-corrected pairwise comparisons. As can be seen in Fig. 2 (see also Section 2 of Supplementary materials), while the two offender groups did not present with differences across either low or high frequency bins, several differences were observed between these groups and the Non-Offender group. Specifically, both offender groups showed significantly decreased low-frequency power spectra compared to Non-Offenders (Dependent Offenders: $t = -3.29$, $p((FWE)) = 0.002$; Non-Dependent Offenders: $t = -2.26$, $p(FWE) = 0.03$), and significantly increased high-frequency power compared to Non-Offenders (Dependent Offenders: $t = 4.25$, $p(FWE) < 0.001$; Non-dependent Offenders: $t = 3.24$, $p(FWE) = 0.002$). As may be expected, given the pattern of low/high frequency activity reported, both Dependent Offenders ($M = 5.83$, $SD = 2.94$; $t = -2.29$, $p(FWE) = 0.03$) and Non-Dependent Offenders ($M = 5.64$, $SD = 3.13$; $t = -2.38$, $p(FWE) = 0.03$) presented with significantly decreased LFPR in comparison to Non-Offenders ($M = 10.76$, $SD = 8.91$; see Fig. 3).

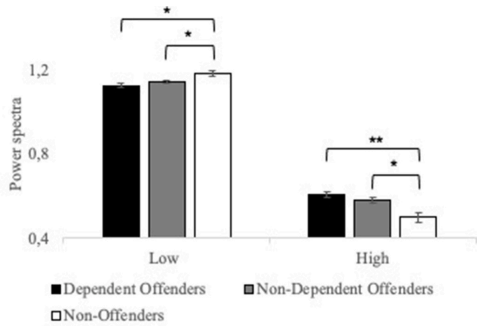

Fig. 2. Mean power spectra differences between groups in overall resting-state activity. Mean power spectra differences in the low-and high-frequency bins between Dependent Offenders (white), Non-Dependent Offenders (black), and Non-Offenders (grey) in overall resting-state activity. Dependent and Non-Dependent Offenders exhibited overall lower peak amplitude in low-frequency activity and higher peak amplitude in high-frequency activity in comparison to Non-Offenders. However, no significant differences were observed between the two Offender sub-groups in either frequency bin. * $p(FWE) < 0.05$ ** $p(FWE) < 0.001$, error bars = SEM

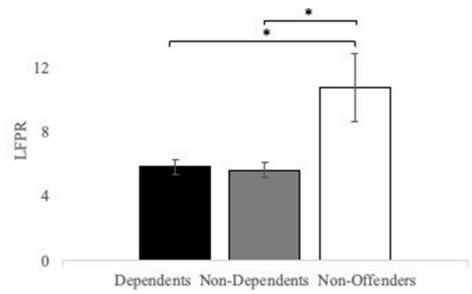

Fig. 3. LFPR differences between groups across all networks. Mean differences in LFPR between Dependent Offenders, Non-Dependent Offenders and Non-Offenders across all networks. Both Offender sub-groups displayed significantly lower LFPR in comparison to Non-Offenders; however, no differences were observed between Dependent and Non-Dependent Offenders. * $p(FWE) < 0.05$, error bars = SEM

3.3. Exploratory network analyses

The non-significant interactions with Network suggested that observed differences in spectral power occurred similarly across all RSNs. To more formally evaluate the extent to which this was true, we compared LFPR between our three participant groups within each of the eight RSNs. As displayed in Fig.4, the two offender groups presented with highly similar spectral profiles within each RSN (all $ps > 0.05$), which differed from the Non-Offender group's frequency profiles within several RSNs. Most consistent was an increase in high-frequency power, which was characteristic of five of the Dependent groups' RSNs, and seven of the Non-Dependent groups' RSNs. On the other hand, reductions in low-frequency power were identified within six of the Non-Dependent group's RSNs, but only one of the Dependent group's RSNs (the DMN; see Section 3 of Supplementary materials for results of higher order effects). Together, this led to significantly reduced network-specific LFPR scores compared to Non-Offenders in a highly overlapping set of four of the Dependent group's RSNs (DMN, ECN, LG and AU), and four of the Non-Dependent group's RSNs (DMN, ECN, LG and BG; see Section 4 of Supplementary materials).

3.4. Exploratory contributions of LCU and psychopathy

Exploratory regression models evaluating the influence of LCU and/or interview-based PCL-R scores on LFPR were conducted separately for Dependent and Non-Dependent groups, as LCU levels showed a quite restricted range within the Non-Dependent group. In the Dependent group, only LCU negatively predicted total LFPR, while PCL-R and the LCU x PCL-R interaction were unpredictive (see Section 5 of Supplementary materials for detailed results). Importantly, this negative relationship with LCU was identified within seven of the eight investigated RSNs (see Section 6 of Supplementary materials); moreover, these findings were only minimally affected by the inclusion of 'major', 'minor' or 'total' substance use metrics as model covariates (see Section 2.2.4 for definitions of major/minor/total substance use). Thus, LCU appeared to serve as a broad-spectrum marker of spectral imbalance in the cocaine-dependent offenders. Supplementary analyses including total use of other substances confirmed that this effect could not be attributed to the use of comorbid substances (see Sections 7 to 12 of Supplementary materials).

Fig. 5 displays the relationship between LCU and overall LFPR within the Dependent offenders. As can be seen, this relationship was curvilinear and negatively-skewed, $r = -0.39$, $p = 0.016$; see the best-fit line in Fig. 5), with data populating only three of four quadrants. Visual

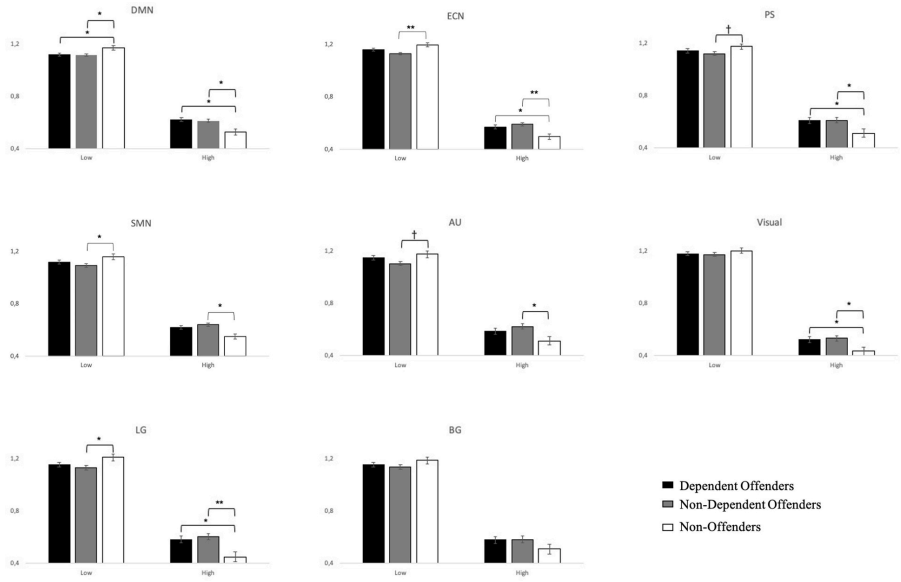

Fig. 4. Pairwise comparisons in power spectra between groups in each RSN. Mean power spectra differences between Dependent Offenders, Non-Dependent Offenders and Non-Offenders in the low- and high-frequency bins in each RSN . † *p(FWE)* < 0.10 * *p(FWE)* < 0.05 ** *p(FWE)* < 0.001, error bars = SEM

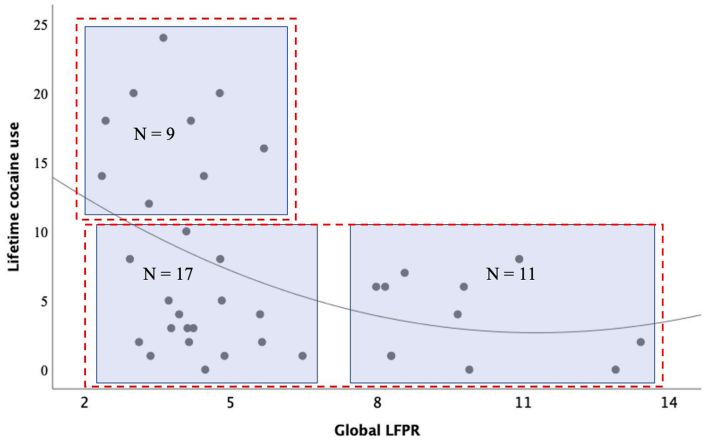

Fig. 5. Relationship between lifetime cocaine use and global LFPR in the Dependent Offender group. The negative curvilinear relationship between lifetime cocaine use and LFPR during overall resting-state activity in Dependent Offenders. The red dotted-lined rectangles represent a 2-cluster representation of the relationship between LCU and LFPR with 9 Dependent Offenders presenting LCU > 10 and LFPR <7, as well as 28 with LCU <10 and ranging LFPR. The blue rectangles represent a 3-cluster representation between LCU and LFPR with 9 Dependent Offenders presented LCU > 10 years with a LFPR < 7, 17 with LCU < 10 years and LFPR < 7, 11 with LCU < 10 years and LFPR > 7, while no Dependent Offenders presented a LCU > 10 years and LFPR > 7.

inspection of the data suggests that all offenders who presented with LCU levels > 10 years showed lower LFPR scores (i.e. less than 6), while all offenders with LFPR > 8 reported LCU levels < 8 years. To formalize these observations, we used k-means clustering to evaluate the efficacy of both 2-cluster and 3-cluster models. Results indicated that the 2-cluster model (Silhouette coefficient = 0.62, Calinski-Harabasz index = 78.76, Davies-Boulding index = 0.50) performed better than the 3-cluster model (Silhouette coefficient = 0.45, Calinski-Harabasz index = 66.58, Davies-Boulding index = 0.82). Given the modest sample size, the precision of this solution should be considered preliminary; however, previous guidance on the use of clustering techniques suggests that a minimum of 30-40 participants per variable (in line with the present study) is sufficient to provide adequate reliability (Clatworthy et al., 2007; Dalmaijer et al., 2020; Dolnicar et al., 2014).

Similar regression models undertaken within the Non-Dependent group showed no significant relationships. However, two caveats:

First, it should be reiterated that the range of LCU scores in the Non-Dependent group were significantly restricted, making interpretation of these null results with cocaine difficult. Second, supplementary analyses indicated that 'minor' and 'total' drug use metrics (see Section 2.2.4 for definitions) did correlate negatively with LFPR in the Non-Dependent participants (see Sections 7 to 12 of Supplementary materials). No relationships were revealed when regression models were conducted across the entire offender sample either, however, this model would be difficult to interpret given the high collinearity between dependence status and LCU.

4. Discussion

The primary aim of our study was to investigate the integrity of rest-related spectral power in antisocial offenders with and without DSM-IV diagnoses of cocaine dependence. Our results indicated broadly disrupted neural oscillations within both offender groups compared to a group of non-offender controls. Indeed, both offender groups showed reduced low-frequency power, and increased high-frequency power, compared to the Non-Offender group. In total, these spectral disruptions led to lower global LFPR levels in both offender groups within four of the eight investigated RSNs. Work of this nature is currently sparse; however, these results are consistent with a handful of studies that have reported spectral abnormalities in a variety of offender populations, including adult offenders with antisocial personality disorder (Liu et al., 2014), and adolescent offenders with antisocial tendencies (Thijssen et al., 2017; Zhou et al., 2015). Considered together with this previous work, the observed abnormalities may be viewed as a) representing a pervasive disturbance characteristic of several RSNs, and b) be largely independent of cocaine dependence status.

4.1. Interpretations of spectral abnormalities in offenders

An understanding of the role of low- and high-frequency activity during rest remains in its infancy; however, several interpretations have been offered. The traditional view posits low-frequency activity during rest as a marker of neural efficiency, and high-frequency activity during rest as indicative of increased 'neural noise' (Biswal et al., 1995). In line with this view, the decreased LFPR levels in both offender groups may be interpreted as indicative of neural inefficiencies. This is in line with a growing body of work linking neural efficiencies to a variety of psychiatric populations including schizophrenia, chronic pain, bipolar disorder, major depressive disorder, and amnestic mild cognitive impairment (Cauda et al., 2009; Malinen et al., 2010; Otti et al., 2013; Sambataro et al., 2017), and suggests that neural inefficiencies may exist as a neurobiological feature within offender populations.

Other research, relying more on task-related activity (i.e. non-rest), suggests that high-frequency activity may play an active, functional role in network dynamics (see Baria et al., 2011; Craig et al., 2017; Gohel & Biswal, 2015). For instance, Baria and colleagues (2011) reported that high-frequency activity often related to increased BOLD response within cortical regions involved in higher-level cognitive processes (i.e. OFC, temporal cortex). In contrast, the low-frequency activity often related to BOLD response within subcortical regions involved in lower-level processes (i.e. posterior precuneus, posterior cingulate). Moreover, Craig and colleagues (2017) observed that high-frequency activity in the DMN was strongly engaged during cognitively demanding tasks. These results suggest that in the DMN, at least, high-frequency activity may play an as-yet-undetermined functional role (Craig et al., 2017). Though speculative, through this lens, our results could suggest the increased engagement of resource-demanding activity during rest in offenders.

Interestingly, some research has linked variation in spectral activity to variation in the synchrony/coherence across disparate neural regions (Anderson et al., 2014; Biswal et al., 1995; Di et al., 2013). For instance, Anderson and colleagues (2014) found a positive relationship between low-frequency amplitudes and local connectivity across the brain. Similarly, low-frequency fluctuations have been associated with connectivity within the motor network during rest (Biswal et al., 1995) and ALFF values (calculated between 0.01 and 0.08 Hz) were strongly related to connectivity strength within and between various RSNs/ROIs (Di et al., 2013). One possibility then is that our result of altered power dynamics in offenders could underpin disruptions in functional connectivity. This interpretation is consistent with numerous reports of connectivity disruptions in offenders (Contreras-Rodríguez et al., 2015; Hoppenbrouwers et al., 2014; Jiang et al., 2017; Leutgeb et al., 2016) and cocaine users (Geng et al., 2017; Ide et al., 2016; Martins et al., 2018; McCarthy et al., 2017; Prichep et al., 2002).

4.2. Implications for dependence

Interestingly, both offender samples showed similar oscillatory differences compared to the Non-Offender controls; moreover, no between-group differences were observed between the Cocaine-Dependent and Non-Dependent Offenders. Thus, all offenders showed similar atypicality in neural oscillations that were largely independent of their drug dependence status. While these results could be seen as running somewhat counter to reports of spectral disruptions in cocaine-dependent (Ide et al., 2016, 2014) and heroin-dependent (Jiang et al., 2011; Wang et al., 2013) individuals, it should be noted that these previous studies were conducted within non-offender samples (and showed a combination of increased and decreased fALFF levels). This distinction may be important, should an aspect of the offender lifestyle itself underpin the observed spectral abnormalities. For instance, we note a handful of studies that have connected neural/cognitive abnormalities to environmental factors such as early life adversity (Dismukes et al., 2015; Kolla et al., 2014), and incarceration itself (Gendreau et al., 1972; Johnson et al., 2015; Umbach et al., 2018). It would be interesting for future research to evaluate the effects of these environmental factors on spectral integrity and/or other neural features.

The results from the present findings will require additional confirmation, optimally with larger samples of participants. However, if the results hold, it would be interesting to consider the potential for targeting abnormal oscillatory patterns in offender populations as treatment/rehabilitative targets. To our knowledge, no fMRI-based power spectra treatment protocols have been tested within offending populations, however, an EEG-based protocol has shown preliminary efficacy reducing aggression and impulsivity in violent offenders through regulation of frequency dynamics (Konicar et al., 2015). Clearly only so much should be made of this at present; however, the finding does serve as an early demonstration that oscillatory disruptions can be remedied, and that this change can have an impact on downstream behavioral/cognitive constructs in offenders.

4.3. Influence of LCU and/or PCL-R scores on spectral abnormalities in offenders

While we identified no differences in spectral dysfunction between the Dependent and Non-Dependent offender groups, the length of lifetime cocaine use (but not interview-based PCL-R scores) was negatively predictive of spectral dysfunction within the Dependent group. One possibility is that it is not dependence status, but rather the lifetime drug burden, that is most relevant to the degree of spectral dysfunction in offenders. These findings partially replicate a recent report regarding the detrimental influences of drug use on spectral integrity in antisocial adolescents (Thijssen et al., 2017), but serve as the first demonstration of this effect within adult offenders. In contrast, neither LCU nor PCL-R scores predicted the magnitude of spectral dysfunction in the Non-Dependent group. Given the restricted range of LCU in the Non-Dependent group, it would be premature to read too much into these group differences. However, one possibility deserving of future consideration is that drug use severity may serve as an important

predictor of dysfunction only after it reaches a certain threshold, whereas other (as of yet unidentified) factors (e.g. personality traits, early life adversity, length of incarceration) could serve more explanatory roles in individuals with less substance use/abuse problems. Consistent with this interpretation, supplementary analyses indicated that some metrics of non-cocaine drug use (ie. 'minor' and 'total' drug use), which the Non-Dependent group did show measurable levels of, did correlate with LFPR. At the least, these results further highlight the need for increased work that treats the offender population as a heterogenous group, with unique, if overlapping, risk factors.

5. Limitations

Several limitations are important to note. First, as the highest identifiable frequency in a power spectrum is equal to ½ the sampling rate (Weik, 2001), the upper-bound limit for the observable frequencies of the time series was limited to < 0.25 Hz given the 2 sec TR. However, high-frequency activity has been shown to contribute to network activity up to 1.4 Hz when using a shorter TR of less than 500 ms (Boubela et al., 2013). A faster TR should be used in future work to investigate the contribution of all ranges of high-frequency activity to resting-state activity in offenders. Second, while our reliance on male-only data will provide the greatest comparison to the extant literature, generalization to female offenders should be undertaken with caution. Particularly given reports of significant differences between male/female offenders (Colins et al., 2017; Rogstad & Rogers, 2008), a future evaluation within a sample of offending women will be crucial. Third, in light of the non-significant effects with PCL-R scores, we should note that our assessment of psychopathy entailed an interview-only process that did not include subsequent file review. While this assessment process has been previously accepted (e.g. Forth et al., 1996; Kosson et al., 1997), and has elicited valid, significant findings in several previous imaging studies (Denomme et al., 2018, 2020; (Arbuckle and Shane, 2017); Shane & Groat, 2018), it does not follow official PCL-R scoring guidelines (Hare, 2003). Thus, the possibility that differences in scoring methodologies are responsible for the null findings should not be completely overlooked. Finally, we note that participants' LCU scores correlated moderately-to-highly with their age in our sample. This is hardly unexpected, however, the inherent collinearity between substance use burden and age does complicate the ability to dissect their influence on rest-related neural activity (see supplementary analyses for results that control for age, but these should be interpreted with caution given due to this collinearity). That said, the fact that LCU only correlated with LFPR in the Dependent group (where LCU variance was sufficient to reliably interrogate the effect) suggests that LCU, and not age, wielded the important influence in our dataset.

Declaration of Competing Interest

None of the material included in our research article has been published previously, and it is not under consideration in another journal. All the coauthors have contributed significantly to this study and agree on the content and authorship of the manuscript. Isabelle Simard conceptualized and designed the study, analyzed and interpreted the data, as well as drafted the article and gave final approval of the version hereby submitted. William J. Denomme and Matthew S. Shane both contributed to the analysis and interpretation of data, critically revised the manuscript and gave final approval of the version submitted, additionally Dr. Shane acquired the data. None of us have conflicts of interest related to this study.

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Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.pscychresns.2020.111235.

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Chapter 3. Study 2

3.1. Introduction

In Simard et al., 2021 (Study 1 of this dissertation), I compared the integrity of rest-related power spectra in offenders with cocaine dependence, offenders without cocaine dependence, and non-offenders and further evaluated the extent to which lifetime cocaine use and/or psychopathic traits related to observed group differences. Results showed that the two offender groups did not present differences in power spectra characteristics; however, both offender groups displayed decreased low-frequency power spectra and increased high-frequency power spectra compared to non-offenders. These disruptions were unrelated to psychopathic traits, but were related to the length of lifetime cocaine use, in those diagnosed with cocaine dependence. Nonetheless, even cocaine use only explained 16% of the variance in power spectra disruptions and only did so within those with a cocaine dependence diagnosis. Thus, other factors beyond those tested in Study 1 may remain unidentified at present. To this end, Study 2 aimed to evaluate the extent to which the number, and the type, of criminal convictions may further explain power spectra dynamics between offenders and non-offenders.

3.1.1. Relationship between criminal history and neural integrity in offenders

Offenders do not necessarily exist as a singular entity but instead differ in both the type and the severity of their criminal histories (Carson et al., 2020). One possibility is that resting-state disruptions may be more characteristic of certain groups of offenders (with certain criminal history characteristics) than others. Past research has put a particular emphasis on differences between offenders with violent versus non-violent criminal histories, and a fair amount of work has identified brain-based differences

between these groups (da Cunha-Bang et al., 2017, 2019; Schiffer et al., 2011), as well as differences in neurocognitive abilities (Hoaken et al., 2007; Meijers et al., 2017) that may reflect brain-based abnormalities (Meijers et al., 2017). However, no work has investigated differences between violent and non-violent offenders in terms of rest-related power spectra.

On the other hand, a fair amount of work has investigated differences in rest-related neural integrity between violent offenders and non-offender controls. One study has suggested that violent offenders show increased resting-state functional connectivity (rs-FNC) between the dorsolateral prefrontal cortex (DLPFC) and both the cerebellum and amygdala, and decreased rs-FNC between the orbitofrontal cortex and the cerebellum, compared to non-offenders (Leutgeb et al., 2016). A second study, which compared violent juvenile offenders and non-offender controls, reported reduced rest-related regional homogeneity (i.e., a measure of the consistency of the spontaneous neuronal activity between a brain area and its adjacent neighbours) in the right caudate, right medial prefrontal cortex and left precuneus, as well as higher regional homogeneity in the right supramarginal gyrus (Chen et al., 2015). Other work has investigated the integrity of resting-state neural activity within violent offender groups. For instance, work investigating within-group disruptions in rest-related *electroencephalogram* (EEG) activity in violent offenders identified asymmetric alpha activity between their right and left hemispheres, as well as stronger right-frontal alpha activity in those with higher levels of aggression (Keune et al., 2012). Overall, this work consistently demonstrates disrupted rest-related activity in offenders. However, because violent offenders were either only compared to non-offenders, or investigated within-group, it is not possible to

know whether observed effects were due to the violent nature of the offender group or instead to something characteristic of all offenders. Clarifying this distinction may be important to determine if differences in rest-related neural activity between violent and non-violent offenders could reflect distinct or common neural mechanisms across groups. Thus, the first aim of Study 2 was to reanalyze the data from Study 1 to specifically evaluate for potential differences between those offenders who did/did not have a violent criminal history.

As reviewed in the General Introduction, previous work has also investigated the relationship between neural activity and specific types of criminal convictions. For instance, pedophilic offenders, compared to non-pedophiles, present aberrant FNC in various neural regions associated with sexual arousal (Poeppl et al., 2015), while, according to a meta-analysis, they might not differ in terms of task-based neural activity (Polisois-Keating & Joyal, 2013). Other work has focused on non-pedophilic sexual offenders of children and reported that, compared to non-offenders, they present decreased rest-related FNC between the amygdala and elements of the frontal cortex (Kärgel et al., 2015; Kneer et al., 2019). Some work has focused on sexual abusers of adults and found that, compared to non-offenders, they present disruptions in white matter connectivity tracts in various neural regions (Chen et al., 2016) and that non-sadistic sexual offenders show increased activity in the temporal cortex when judging moral transgressions as being more severe while sadistic sexual offenders do not show this relationship (Cazala et al., 2020). Other work has investigated homicidal offenders and identified that adult and juvenile homicidal offenders, compared to non-homicidal offenders, present decreased grey matter volume in various neural regions (Cope, Ermer,

et al., 2014; Sajous-Turner et al., 2019). Work has also focused on domestic batterers and observed that, compared to offenders who have not been convicted of violence against women, women batterers present disrupted activity in various neural regions (Bueso-Izquierdo et al., 2016; Lee et al., 2009) and reduced DMN engagement (Marín-Morales et al., 2020) when confronted to violent interpersonal scenarios and images. Finally, some work has investigated white-collar offenders and found that, compared to non-offenders, they present increased cortical thickness in frontal and temporal-parietal regions (Raine et al., 2012) and increased gray matter volume in the frontal lobe (Ling et al., 2019). Thus, while previous work has identified a relationship between having been convicted of certain crimes and neural disruptions, this work remains sparse, and few studies have investigated this relationship in the context of resting-state activity. Consequently, as a secondary goal, Study 2 aimed to assess whether rest-related power spectra related to the different types of criminal convictions. To this aim, I explored the integrity of rest-related power spectra within specific crime types.

Finally, offenders with more prolific criminal histories may have distinct or more severe neural insults than those with less prolific criminal histories. The only two studies to have investigated this relationship to date focused on task-based neural activity. In a first study, participants recruited from the general population who reported having committed more criminal behaviour presented increased activity in the temporal and parietal cortices and the insula when making risky choices compared to participants who reported having committed fewer criminal behaviours (Reyna et al., 2018). In the second study, Hyde et al. (2016) found that offenders who self-reported a greater number of arrests showed heightened amygdala activity when presented with fearful and angry

faces. While these studies used self-reported arrest and criminal behaviour measures rather than convicted crimes, they nonetheless indicated a relationship between neural activity and the number of crimes perpetrated. The extent to which a similar relationship may occur with power spectra remains unknown. However, if power spectra serves as a valid metric of baseline neural activity, it may provide evidence of underlying core abnormalities associated with the severity of an offender's antisocial history. Therefore, a second goal of the study was to evaluate whether there would be a relationship between baseline neural activity and the number of crimes that offenders were convicted of. To this aim, Study 2 explored the relationship between rest-related power spectra and the number of convictions received.

3.1.2. Relationship between future crimes and neural integrity in offenders

Recent work has established that differences in neural features can (to some extent, at least) also predict who is more at risk for re-offending (Aharoni et al., 2013, 2014; Coppola, 2018; Gaudet et al., 2016; Kiehl et al., 2018; Poldrack et al., 2018; Steele et al., 2015). For instance, Pardini and colleagues (2014) found that decreased amygdala volume accurately predicted aggression and violence in a mixed sample of non-offenders, and offenders with a history of chronic or transient violence. Additionally, Aharoni et al. (2013) showed that offenders with a reduced anterior cingulate cortex (ACC) response during an inhibitory task were two times more likely to be re-arrested than their counterparts. In a follow-up study, Aharoni et al. (2014) demonstrated that including inhibitory task-related ACC activity in predictive models of re-arrest could predict overall crime, as well as violent crimes, over and above the combination of the traditional risk factors of age, drug use and psychopathic traits with an accuracy of 68%. In one of the

few studies to use multimodal neuroimaging metrics, Steele and colleagues (2015) reported that combining fMRI (ACC activity) and EEG (error-related negativity (ERN/Ne) and the error positivity (Pe) event-related potentials (ERPs)) during Go/NoGo performance increased the predictive accuracy of the model presented by Aharoni and colleagues in 2014 to 78.05% (Steele et al., 2015). Finally, Kiehl and colleagues (2018) assessed the validity of different machine learning models on a longitudinal cohort using brain age of grey matter volume in the amygdala to obtain a model which would be most predictive of recidivism. Their baseline risk-assessment model included age, PCL-R Factor 1 and Factor 2 scores, the interaction between Factor 1 and Factor 2, drug and alcohol dependence and false alarm rate from a Go/NoGo task. Machine learning models combining grey matter age measures and behavioural risk factors of offending were more predictive than neural age or behavioural risk factors alone (Kiehl et al., 2018). Together, this work suggests that task-based neural activity and grey matter volume, combined with non-brained based predictors, can increase the accuracy of recidivism predictions.

However, to my knowledge, only one study has investigated whether adding resting-state neural data to traditional risk-assessment models can further improve the prediction of future crimes. In this study, Delfin and colleagues (2019) used a comprehensive risk assessment model (which included current age, gender, age of the first offence, psychopathic traits, substance use disorder, cluster B personality disorder (i.e., ASPD, borderline personality disorder (BPD), histrionic personality disorder and narcissistic personality disorder), educational attainment, mental disorder of first-degree relative, and the number of crimes committed as predictors of recidivism). They then examined whether adding rest-related regional cerebral blood flow (rCBF) data from within eight

distinct brain regions (i.e., left/right frontal lobe, left/right parietal lobe, left/right temporal lobe and left/right cerebellum) to this model could further improve the prediction of 10-year recidivism rates. Results showed that adding resting-state rCBF increased the predictive model's accuracy from 0.64 to 0.82 (Delfin et al., 2019). However, it is important to note that this study was conducted using a relatively small sample (44 participants) and a rather complex machine learning algorithm (i.e., random forest classifier). Generally, random forest classifiers can require upwards of 200 participants *per variable* to offer appropriate levels of power, stability and generalizability (van der Ploeg et al., 2014). Thus, while Delfin and colleagues' (2019) study provided an important proof of concept, definitive conclusions require additional work with more robust predictive pipelines. Hence, the third goal of Study 2 was to evaluate whether combining rest-related neural oscillations to a traditional risk assessment model could improve prediction of recidivism. To this aim, I conducted several machine learning models to predict re-offending: one model that included individual and lifestyle risk factors of offending and another model that also added rest-related power spectra activity as an additional predictor.

3.2. Methods

3.2.1. Participants

The sample was the same as in Study 1. However, criminal history data could not be obtained for five participants; thus, 97 participants were included in final analyses (46 Violent offenders, 33 Non-Violent offenders and 18 Non-offenders; see Table 3.1 for complete demographics, or Simard, Denomme & Shane (2021) for complete details on recruitment and screening procedures). Offenders and non-offenders were matched on

important demographic variables (e.g., age, gender, IQ) to ensure that groups did not differ on these variables by more than .5 standard deviations. Offenders were classified as Violent Offenders if they had any of the following violent crimes in their criminal history (up to the date of their fMRI scan): homicide, murder, manslaughter, attempted murder, assault/battery, sexual assault, rape, robbery, or kidnapping. Offenders were classified as Non-violent if they had not been convicted of any of these violent crimes. The Research Ethics Board of the University of New Mexico approved this study. Participants provided written informed consent in accordance with the Code of Ethics of the World Medical Association (Declaration of Helsinki).

Table 3.1

Participant Demographics

	Violent Offenders (N = 46)	Non-Violent Offenders (N = 33)	Non-Offenders (N = 18)	*t*-tests		
	Mean *sd*	Mean *sd*	Mean *sd*	VO vs N-O	N-V O vs N-O	VO vs N-V O
Age	34.91 *9.54*	31.12 *7.71*	27.50 *7.14*	2.98*	1.64	1.88
IQ	105.17 *11.39*	106.94 *12.96*	111.72 *9.98*	-2.14	-1.36	-0.64
Convictions	9.78 *5.96*	9.94 *6.39*				0.11
PCL-R	21.58 *7.47*	19.26 *7.05*				1.390
Drug use (years)						
Total	42.16 *29.43*	32.06 *23.57*	8.17 9.46	6.98**	5.12**	1.64
Major	7.72 *8.77*	8.36 *10.06*	0.61	5.23**	4.31**	-0.30
Minor	24.43 *17.58*	17.06 *10.79*	7.56	5.08**	3.19*	2.30*
Recidivism	N = 6	N = 4	-			

Note. Violent Offenders (VO), Non-violent Offenders (N-V O), Non-Offenders (N-O), Standard Deviation (*sd*), Number (N). 'Convictions' represents the total number of criminal convictions received by offenders. 'Major' represents the years of lifetime use of drugs such as methamphetamines, opiates/analgesics, heroin, and cocaine. 'Minor' represents the years of lifetime use of drugs such as alcohol, cannabis, nicotine, hallucinogens and inhalants.

* $p < 0.05$ ** $p < 0.001$

3.2.2. Clinical/Forensic Assessments

3.2.2.1. SCID-I/P. The Structured Clinical Interview for DSM-IV-TR (SCID-I/P; First et al., 2002) was used to evaluate participants for Axis I and II disorders, including ASPD and substance use disorders. Interviews were videotaped and conducted by highly trained Master's level research personnel, under the guidance of a senior SCID trainer (R.C.; see acknowledgements in Study 1). Participants were excluded if they met diagnostic criteria for a lifetime history of psychotic disorder, bipolar disorder, major depressive disorder within the last six months or current use of antipsychotic medications. This resulted in the exclusion of 10 recruited participants (one due to a history of psychosis; nine due to the use of current antipsychotic medication).

3.2.2.2. WAIS-III. Participants' full-scale intelligence quotients (IQ) were approximated using the vocabulary and matrix reasoning scales of the Wechsler Adult Intelligence Scale 3rd edition (WAIS-III).

3.2.2.3. Criminal history. A comprehensive background check was performed on all offenders in November 2020 to obtain a record of all criminal offences for which offenders were convicted a) prior to their fMRI scan and b) one-year post-study completion. This information was obtained by collating information across the New Mexico courts online offence database (which provides a public record of all criminal convictions in the state) and www.instantcheckmate.com (a paid database that provides a broader search of publicly held criminal records). Five participants could not have their criminal data located in either search process and were excluded from subsequent analyses.

3.2.2.3.1. Classification of crime types. Convictions (as reported on background check data; see above) were classified as violent or non-violent following the classification system described in the previous section. In addition, convictions were further classified into distinct crime types using crime classification from the Crime Inventory Questionnaire, which was developed in-house at the Mind Research Network (MRN) (see Appendix B; e.g., theft, drug-related offences, assault/battery, driving while intoxicated). Next, to reduce collinearity between crime types and to increase cell sizes in each crime category, crimes were categorized into specific crime categories: assaulting a police officer, assault/battery, domestic assault/battery and child abuse/neglect were combined to form an assault category; drug possession and drug distribution were combined to form the category of drug-related crime; theft <$250, theft >$250 and burglary were combined to form a theft category; escape, failure to appear and resisting arrest were combined to create a resisting/escape category; rape/sexual assault and child sexual assault/incest were grouped to form a sexual assault category. Cell sizes were still somewhat low for these crime-specific analyses, so all analyses pertaining to crime types were undertaken for exploratory purposes. The relationship between crime category data was evaluated both categorically (to evaluate for neural differences between those who were/were not convicted of a specific type of crime) and continuously (to evaluate for neural differences as a function of the number of criminal convictions).

3.2.2.3.2. One-year Post-fMRI Recidivism Rates. One-year post-scan recidivism rates were calculated as a binary measure (i.e., yes/no), based on whether participants had or had not been convicted of an offence within that one year. This binary data was then

used to undertake exploratory analyses into the extent to which neural characteristics could be used to predict future criminal activity.

3.2.2.4. Psychopathic traits. Offenders were assessed for psychopathic traits via a semi-structured Psychopathy Checklist-Revised (PCL-R) interview (Hare, 2003), which was videotaped and conducted by highly-trained research personnel (trained by M.S.). Criminal file reviews were not possible at the time of scoring. Thus, participants were scored 0 to 2 on each of the 20 PCL-R items based on the clinical interview alone (see Denomme et al., 2018, 2020; Forth et al., 1996; Kosson et al., 1997 for evidence of the validity of this approach). Psychopathy was not included in primary models as the influence of psychopathic traits on power spectra activity has already been investigated in this sample (see Simard et al., 2021).

3.2.2.5. Drug use. The number of regular drug use years was evaluated using a modified Addiction Severity Index - Expanded (ASI-X; McLellan et al., 1992), administered orally by a trained examiner. Following administration of the ASI-X, composite scores of total drug use were calculated by summing the total years of use of drugs that corresponded to two categories: Major drugs (i.e., methamphetamines, opiates/analgesics, heroin, cocaine) and Minor drugs (i.e., alcohol, cannabis, nicotine, hallucinogens and inhalants). Thus, for example, if a participant used cannabis for three years, hallucinogens for five years and nicotine for five years, their Minor drug use score was calculated as 13 years. In addition, a 'Total drug use' score was calculated as the sum of major + minor drug use. Drug was not included in primary models as the influence of drug use on power spectra activity has already been investigated in this sample (see

Simard et al., 2021). However, drug use is provided as demographic information (see Table 3.1).

3.2.3. Data acquisition

Resting-state data was acquired using a 3T Magnetom Trio Tim Siemens scanner at the Mind Research Network (MRN). Participants kept their eyes open and directed at a fixation cross. The 5.5-minute acquisition was performed using a fast gradient-echo EPI sequence. Imaging parameters were as follows: TR = 2000ms, TE = 29ms, FA = 75°, matrix = 63 x 63, slices = 33 acquired in an ascending interleaved fashion, slice thickness = 3.5 mm, FOV = 240mm x 240mm and voxel size = 3.8 x 3.8 x 3.5mm.

3.2.4. Preprocessing

Resting-state data was preprocessed using SPM12. Data was motion-corrected using INRIAlign (Freire et al., 2002). Realignment was handled using a distance cut-off of 2.5, with data coregistered with a full-width-as-half-maximum (FWHM) of 8mm. Slice timing correction was applied using the 16th slice as reference. Data was then normalized using the SPM5 EPI template; the mean image was used for parameter estimation. Finally, data was smoothed using a 10mm Gaussian kernel. No participant presented movement above 5mm. Hence, after preprocessing, all participants were included in the analyses.

3.2.5. Independent Component Analysis (ICA) and Spectral analysis

All ICA processes and spectral analyses were as described in Simard et al. (2021).

3.2.6. Data Analytic Strategy

A 3 (Group) x 2 (Bins) x 8 (Network) omnibus ANOVA was used to evaluate all higher-order effects, followed by targeted comparisons used to evaluate specific

hypothesized effects. This analytic strategy provides the greatest granularity with regard to the nature of spectral distributions in offenders and thus served as our primary analytic pipeline. However, because the literature focuses predominantly on single low-frequency centred metrics (e.g., ALFF, fALFF, low-frequency power ratio (LFPR)), I also report differences in LFPR scores, which have previously been used as a predictor in a clinical setting (see Yu et al., 2013).

In addition, correlations were employed to explore the relationship between criminal history (i.e., number of convictions) and power spectra integrity (i.e., LFPR).

Next, *support vector classifier* (SVC) with Leave One Out cross-validation (LOOCV) models were performed using binarized recidivism after a one-year follow-up as a dependent variable to explore if adding global power spectra to a traditional predictive model could improve our ability to predict future crimes. Thus, two SVC models were performed: one model combining age, PCL-R score, the number of past convictions and total drug use as predictors and a second model in which global LFPR was added to the predictors from the first model. As there was a steep class imbalance between those who had been convicted of a crime after a one-year follow-up (N = 10) and those who had not (N = 69), I used a *Synthetic Minority Oversampling Technique* (SMOTE; Chawla et al., 2002) to oversample the cases of the minority class (i.e., recidivism) during the training loop of the model, to obtain an equal distribution between classes. These analyses were performed in the Python 3.7.4 environment using the Scikit-learn library (Pedregosa et al., 2011). The final sample size of 79 offenders falls within recommendations (between 75 to 100 participants) to obtain good (albeit potentially

somewhat underpowered) classification with reasonable precision and validation when performing SVC modelling (Beleites et al., 2013).

3.3. Results

3.3.1. Demographics

One-way ANOVAs were used to evaluate for potential group differences in relevant demographic/clinical/forensic variables. As in Study 1, a main effect of Age was identified, ($F = 5.30$, $p < 0.05$), with subsequent t-tests this time indicating that the Violent Offender group ($M = 34.91$, $SD = 9.54$) was significantly older than the Non-Offender group ($M = 27.50$, $SD = 7.14$; $t = 2.98$, p (FWE) < 0.05) and non-significantly older than the Non-Violent Offender group ($M = 31.12$, $SD = 7.71$; $t = 1.88$, p (FWE) > 0.05). No main effect of IQ was identified (see Table 3.1 for detailed demographics).

The Violent Offender group also presented with higher Minor, $t = 2.30$, $p < 0.05$, but not Major or Total, ts < 1.64, $p > .05$, drug use than the Non-Violent Offender group, and both the Violent and Non-Violent Offender groups presented with higher Total, Major and Minor drug use compared to the Non-Offender group (see Table 3.1 for detailed results). No significant differences were observed in the total number of criminal convictions or total PCL-R scores between the Violent Offender group and the Non-Violent Offender group, ts < 1.39, ps $> .05$ (the Non-Offender group was not assessed for criminal activity or psychopathy). Correlational analyses were reported in Simard et al. (2021) but are reported again in Table 3.2 for the reader, with the addition of the number of criminal convictions.

Table 3.2

Correlation matrix between demographic variables in offenders

	PCL-R	All Drugs	Minor Drugs	Major Drugs	Number of Convictions
PCL-R	--				
All Drugs	0.43**	--			
Minor Drugs	0.43**	0.90**	--		
Major Drugs	0.31*	0.64**	0.44**	--	
Number of Convictions	0.39**	0.42**	0.38**	0.38**	--

* $p < 0.05$ ** $p < 0.001$

3.3.2. Spectra analyses

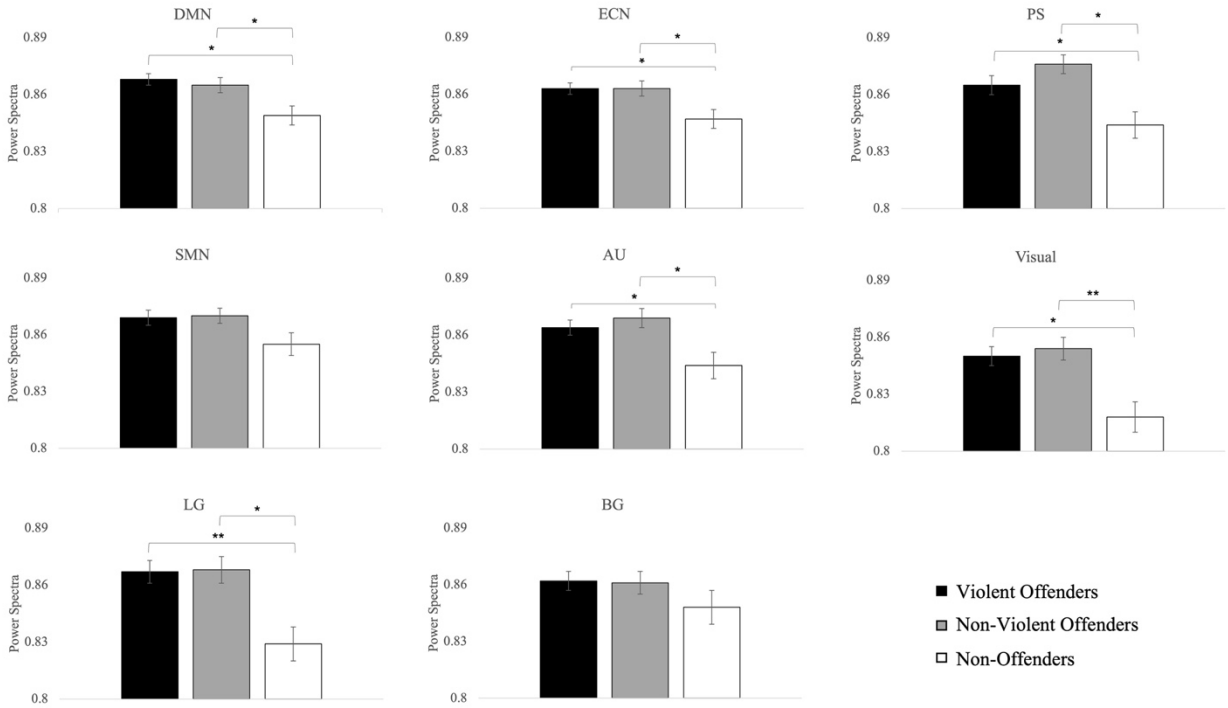
The 3 (Group) x 2 (Bins) x 8 (Network) omnibus ANOVA identified main effects of Group, Network and Bin, which were very similar to those reported in Simard et al. (2021; see Appendix C.1 for detailed results, and Simard et al., (2021) for analyses investigating the effect of cocaine dependence status). To reduce duplication, results reported here focus primarily on potential interaction effects associated with the Violent/Non-Violent group designation. To this end, a Bin x Group, F (2, 94) = 5.32, p = 0.006, and a near significant Group x Network, F (14, 94) = 1.53, p = 0.094 interaction were identified. The 3-way Group x Bin x Network interaction did not reach significance, F (14, 94) = 1.39, p = 0.148).

Bin x Group interaction: I collapsed across networks to dissect the Bin x Group interaction and evaluated spectral differences in each Bin/Group cell. Overall, Violent and Non-Violent Offenders showed significantly decreased low-frequency power, ts = -2.67, -2.51, ps (FWE) < 0.05, and significantly increased high-frequency power, ts = 3.45, 3.69, ps (FWE) < 0.001, compared to Non-Offenders. Additional results of this analysis can be found in Appendix C.2.

Group x Network interaction: To explore the nature of the Group x Network interaction, mean spectral power within each RSN was evaluated separately for Violent, Non-Violent and Non-Offender groups. As displayed in Figure 3.1, both Violent and Non-Violent Offender groups presented with similar spectral profiles in all RSNs (all *p*s > 0.05), with mean power spectra values higher than that of the Non-Offender groups' spectral profiles in six of the eight RSNs (i.e., DMN, ECN, PS, AU, Visual and LG networks; see Table 3.3 for results of higher-order effects).

Figure 3.1

Mean power spectra differences between groups in each resting-state network



Note. Global power spectra was increased in both Violent Offenders and Non-Violent Offenders groups compared to Non-Offenders in the Default-Mode Network (DMN), Executive Control Network (ECN), Posterior Salience (PS), Auditory (AU), Visual and Language (LG) networks. However, global power spectra did not significantly differ between groups in the Sensorimotor Network (SMN) and Basal Ganglia (BG) network.

* *p(FWE)* < 0.05 ** *p(FWE)* < 0.001

Table 3.3

Main effects of 2 (Bin) x 3 (Group) ANOVAs in each resting-state network

	DMN	ECN	BG	SMN	PS	LG	Visual	AU
Within-Subject Effects								
Bin $F(1, 87)$	837.48**	1221.52**	559.64**	487.48**	605.49**	481.52**	973.03**	414.98**
Bin x Group $F(2, 87)$	5.41*	6.42*	2.37†	3.66*	3.63*	5.59*	2.43†	3.09*
Between-Subjects Effects								
Group $F(2, 87)$	4.85*	4.85*	0.99	1.98	4.76*	6.56*	7.72**	3.84*

Note. F-score (*F*), Default-Mode Network (DMN), Executive Control Network (ECN), Basal Ganglia (BG), Sensorimotor Network (SMN), Posterior Salience (PS), Language (LG), Visual, Auditory (AU).

† $p(FWE) < 0.10$ * $p(FWE) < 0.05$ ** $p(FWE) < 0.001$

3.3.3. Relationship between past convictions and power spectra

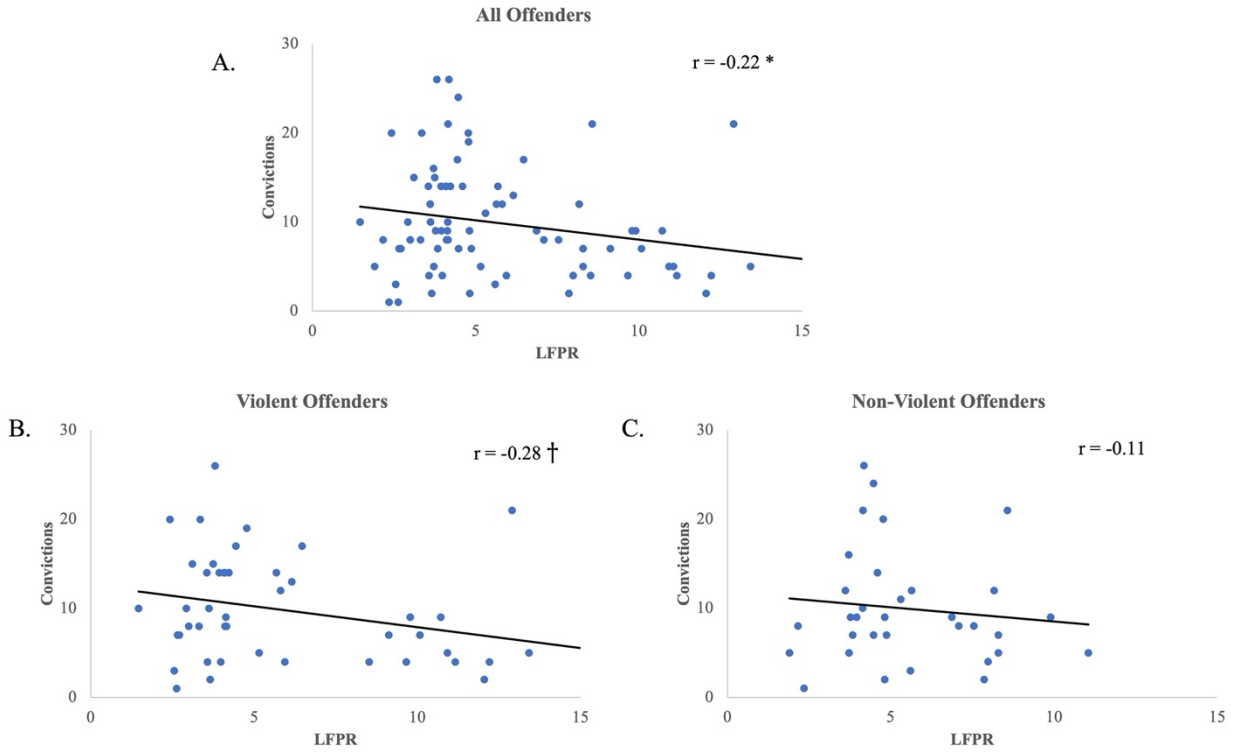
3.3.3.1. Number of past convictions. The total number of criminal convictions was first assessed for outliers. This involved inspecting the data to identify data points that were more than three standard deviations from the mean, and considering Cook's D leverage values for values greater than one. No data points reached these thresholds, and thus all data was retained in analyses reported below.

Correlation analyses revealed a significant negative relationship between number of convictions and LFPR across the whole offender sample (r = -0.22, $p = 0.05$) and within each of the Violent (r = -0.28, $p = 0.06$), and Non-Violent (r = -0.114, $p = 0.53$) offender groups (see Figure 3.2). A Fisher's r-to-z transformation showed that the

magnitude of this relationship did not statistically differ between the Violent and Non-Violent groups (z = -0.74, p = 0.23).

Figure 3.2

Scatterplots of number of convictions and global low-frequency power ratio across all Offenders and within Violent and Non-Violent Offenders



Note. Low-Frequency Power Ratio (LFPR) was significantly negatively related to the number of convictions across all offenders (A) and near significantly within Violent Offenders (B), while this relationship was not significant in Non-Violent offenders (C).

† $p < 0.10$ * $p < 0.05$

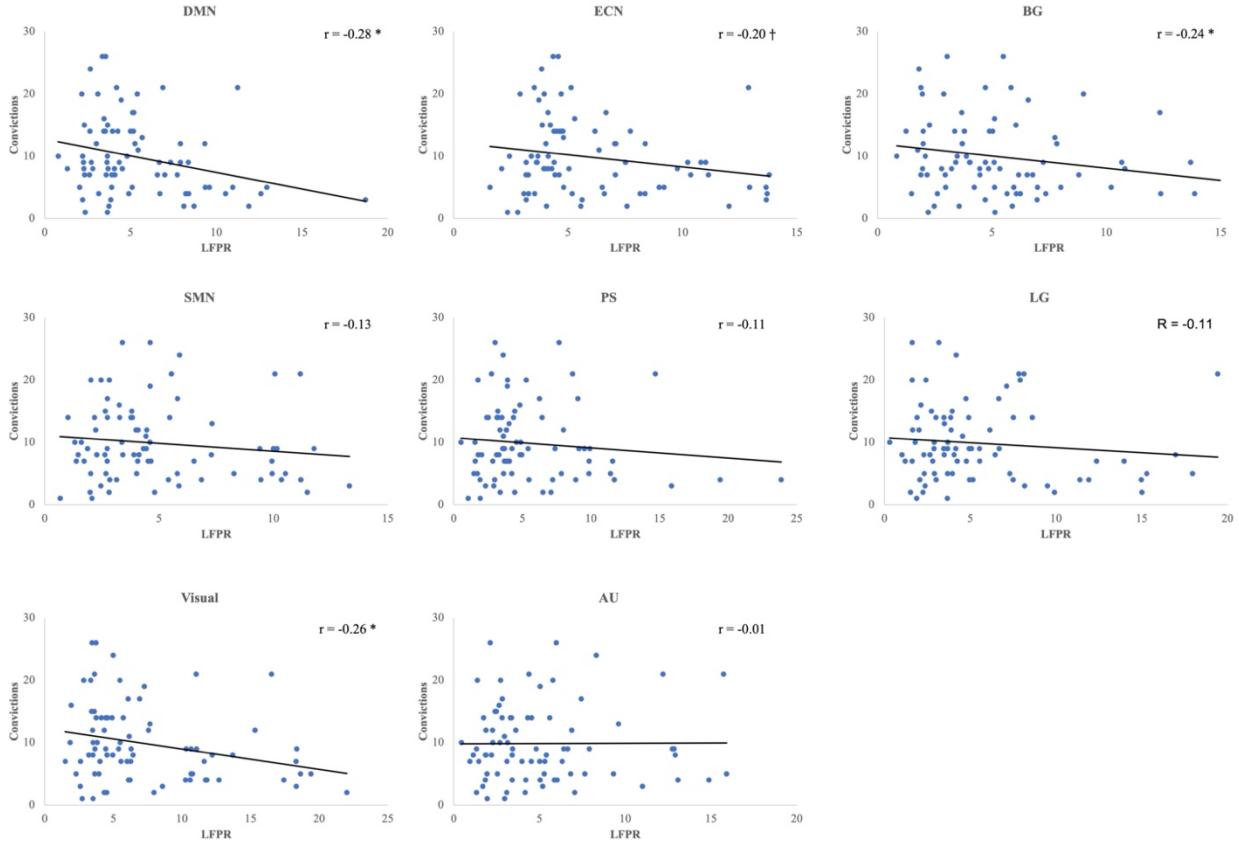
To further explore the effect of the total number of convictions on LFPR, I performed a 2 (Group: Violent, Non-Violent) x 8 (Network) ANCOVA, using the number of convictions as a covariate. Results showed a significant main effect of

Network F (7, 75) = 6.17, $p < 0.001$, and a near significant main effect of Number of Convictions, F (1, 75) = 3.01, $p = 0.09$, such that offenders who were convicted of more crimes showed decreased LFPR. These effects were influenced by a significant Network x Number of Convictions interaction, F (7, 75) = 2.28, $p = 0.03$, and a near significant Network x Group interaction, F (7, 75) = 2.84, $p = 0.08$. The main effect of Group, F (1, 75) = 1.46, $p = 0.23$, the Group x Number of Convictions, F (1, 75) = 1.35, $p = 0.25$, and the Network x Group x Number of Convictions, F (1, 75) = 1.32, $p = 0.24$, did not reach significance.

Network x Number of Convictions Interaction: To explore the nature of the Network x Number of convictions interaction, I evaluated the correlation between the number of convictions and LFPR across all offenders within each RSN. These analyses indicated a negative relationship between the number of convictions and LFPR in 7 of the 8 RSNs (all except the Auditory network), which reached significance in the DMN, BG and Visual networks, and nearly reached significance in the ECN; see Figure 3.3 and Appendix C.4).

Figure 3.3

Scatterplots of number of convictions and global low-frequency power ratio in each resting-state network across offenders



Note. Across offenders, the number of convictions were significantly related to low-frequency power ratio (LFPR) in the Default-Mode Network (DMN), Basal Ganglia (BG) and visual networks, while this relationship was near significant in the Executive Control Network (ECN) and non-significant in the Sensorimotor Network (SMN), Posterior Salience (PS), Language (LG) and Auditory (AU) networks.

† $p < 0.10$ * $p < 0.05$

Interestingly, the relationship between convictions and LFPR visually resembled the curvilinear relationship between cocaine use and LFPR in cocaine-dependent offenders from Simard et al. (2021). It is possible that these variables may explain similar variance in LFPR levels in offenders; or it could be that they explain independent or even interactive effects on LFPR. To evaluate these possibilities, I conducted a hierarchical linear regression model across all offenders (i.e., Violent and Non-Violent groups) that included number of convictions (level 1), cocaine use (level 2), and the number of convictions x cocaine use interaction (level 3) as potential predictors of LFPR. This model did not significantly contribute to LFPR (Number of convictions ($\beta = -0.18$, SE $\beta = 0.06$, $p > 0.05$), cocaine use ($\beta = -0.12$, SE $\beta = 0.06$, $p > 0.05$), number of convictions x cocaine use interaction ($\beta = 0.07$, SE $\beta = 0.01$, $p > 0.05$); see Appendix C.5 for detailed results). Importantly, cocaine use and number of convictions ($r = 0.32$, $p < 0.05$) were moderately significantly correlated with each other. However, these variables met the assumptions of collinearity (i.e., Tolerance higher than 0.1 and VIF value lower than 10), thus collinearity was not a concern (Number of convictions, Tolerance = 0.88, VIF= 1.13; Cocaine use, Tolerance = 0.75, VIF= 1.34; Number of convictions x Cocaine use interaction, Tolerance = 0.78, VIF= 1.28). Thus, the relationship between these variables makes it hard to disentangle the individual contributions of the number of convictions and years of cocaine use on LFPR but may suggest that these variables have a parallel negative effect on LFPR.

3.3.3.2. Types of past convictions. Although no group effects were identified, I wished to explore whether power spectra activity disruptions related to having been convicted of specific types of crimes. It is important to note that the following analyses

are very exploratory as the number of participants within certain crime types is relatively low, and interpretations must thus be undertaken with caution (see Appendix C.6. for the distribution of individuals who were convicted in each crime category).

Thus, I wished to explore whether LFPR related to the number of convictions in each crime category. To this aim, I performed separate logistic regressions for individual crime types to assess whether a conviction of that crime (assessed as a binary yes/no measure), related to offenders' global LFPR levels. For transparency, these analyses did not control for multiple comparisons, as they were administered for purely exploratory purposes. These analyses indicated that global LFPR was negatively associated with having been convicted of drug-related crimes ($\beta = -0.19$, SE $\beta = 0.08$, OR = 0.83, $p < 0.05$), driving while intoxicated (DWI; $\beta = -0.17$, SE $\beta = 0.10$, OR = 0.84, $p = 0.09$) and 'other driving offences' ($\beta = -0.19$, SE $\beta = 0.08$, OR = 0.83, $p < 0.05$).

3.3.4. Exploratory prediction of future crimes

Support vector classifier (SVC), a type of supervised learning algorithm, was used to assess the ability of individual risk-factors of offending and global LFPR to accurately classify offenders as having been convicted or not of a crime after a one-year follow-up. Model 1, which included age, PCL-R, the number of past convictions and total drug use as predictors, was 48% predictive of one-year recidivism rates (detailed classification metrics of the models are presented in Appendix C.7). SVC Model 2, which added global LFPR to the predictors of Model 1, showed slight but non-significant predictive improvement (51%). Thus, combining global LFPR with a traditional recidivism prediction model did not significantly improve the prediction accuracy of recidivism. As shown in Table 3.4, the number of past convictions was the strongest

predictor of recidivism in both models. Global LFPR was also predictive of recidivism in Model 2, albeit to a lesser extent. Age, PCL-R score and total drug use, on the other hand, were negatively associated with recidivism, with age being the strongest predictor.

Table 3.4

Coefficients of the contribution of each predictor to the support vector classifier models

	Coefficients	
	Model 1	Model 2
Age	-0.056	-0.108
PCL-R	-0.006	-0.051
Past Convictions	0.171	0.173
Drugs	-0.037	-0.027
LFPR		0.019

Note. Low-Frequency Power Ratio (LFPR).

3.4. Discussion

The primary goal of this study was to assess for differences in rest-related power spectra activity between Violent Offenders, Non-Violent Offenders and Non-Offenders. To this end, I re-analyzed data from Simard et al. (2021) to focus more specifically on the potential role of a violent criminal history on power spectra. As in Simard et al. 2021, both offender groups showed decreased low-frequency activity and increased high-frequency activity in comparison to non-offenders; as a result, both offender groups displayed increased power spectra compared to non-offenders in six of the eight investigated RSNs. As in Study 1 (Simard et al., 2021), there were no differences in rest-related power spectra between offender groups (in Study 1 between cocaine-dependent groups; in Study 2 between violent/non-violent groups). Thus, while the integrity of resting-state neural oscillations differed between offenders and non-offenders, it again did not differ between different types of offender groups.

3.4.1. Violent versus Non-violent Offenders

To my knowledge, this work is the first to compare power spectrum differences between violent and non-violent offenders. While the results remain preliminary, they nonetheless suggest that disruptions in rest-related neural oscillations do not differ as a function of offenders' violent history. This suggests that power spectra abnormalities may not be a neural feature related to violent offending.

This finding contrasts with a small amount of previous work that has been conducted on other neural differences between violent/non-violent offenders. For instance, previous studies have identified task-related (i.e., hemodynamic activity) differences in the amygdala and the striatum in reaction to provocation (da Cunha-Bang et al., 2017), in cingulate and middle/superior prefrontal cortices when observing images of interpersonal violence (Bueso-Izquierdo et al., 2016), in the hippocampus, fusiform gyrus, posterior cingulate gyrus, thalamus and occipital cortex in response to threat (Lee et al., 2009), in bilateral precuneus when viewing images of aggression against women (Lee et al., 2009), and in reduced DMN-relevant circuitry when making moral decisions in intimate partner violent scenarios (Marín-Morales et al., 2020). It should be noted that there are several differences between these previous studies and the present study. First, the previous studies all measured task-based activity, while I measured rest-based power spectra. Second, the previous studies all measured activity in the context of violence-related scenarios. It may be that violent/non-violent offenders *do* show abnormal neural responses when placed in violent contexts. Nevertheless, the results of my study suggest that these abnormalities do not occur during less engaging rest-related contexts.

Consequently, this could indicate that the underlying neural mechanisms of violent offending are context-dependent rather than associated with baseline neural functioning.

3.4.2. Relationship between the number of previous convictions and power spectra

A secondary goal of this study was to assess the relationship between the number of convictions received by offenders and neural disruptions in resting-state power spectra. Results identified a negative relationship between the number of convictions and LFPR. As higher levels of low-frequency activity have been associated with stable RSN activity (Biswal et al., 1996), these results suggest that an increased criminal history is associated with instability in resting-state activity. To my knowledge, only one other study has investigated the relationship between neural activity (albeit not during rest) and the number of crime-related legal interventions. In this study, the number of arrests was positively associated with increased activity in the amygdala in reaction to fearful and angry faces in a sample of low-income men (Hyde et al., 2016). While I could not identify previous work investigating the relationship between the number of convictions and resting-state neural activity, a rich literature has established a relationship between neural activity and antisocial/criminal behaviour (Dugré & Potvin, 2021; Reyna et al., 2018). Indeed, in their 2021 meta-analysis, Dugré & Potvin showed that those with a high level of lifestyle and antisocial behaviour traits (across studies using offenders and non-offenders) showed decreased resting-state functional connectivity within the ventromedial prefrontal cortex. Another study conducted among non-offenders showed that individuals who report committing more criminal behaviour show increased activity in parietal regions, insula, anterior cingulate cortex, right supramarginal gyrus and right angular gyrus when making risky choices (Reyna et al., 2018). Thus, while previous work

has demonstrated a relationship between antisocial behaviour and aberrant task-related activity (Hyde et al., 2016; Reyna et al., 2018) and with rest-related connectivity (Dugré & Potvin, 2021), my results further indicate that the number of past convictions in adult offenders also relates to disruptions in baseline neural oscillations.

Of potential import, these results showed a negative curvilinear relationship between offenders' LFPR levels and the number of crimes they were convicted of. Indeed, decreased LFPR was consistently associated with having been convicted of more crimes. However, LFPR showed more variability in offenders who had fewer criminal convictions. Study 1 (Simard et al., 2021) also showed a negative curvilinear relationship with LFPR (with regard to lifetime cocaine use within cocaine-dependent offenders). Thus, both cocaine-dependent offenders with more cocaine use, and offenders who have been convicted of more crimes, showed lower levels of LFPR. This could suggest overlapping influences between cocaine use metrics and criminal history metrics on LFPR. Alternatively, it could be that these curvilinear influences are supported by different underlying mechanisms, such that they independently have a negative curvilinear relationship with LFPR. Indeed, further analyses revealed that these variables were moderately correlated and that, when combined, did not have a significant additive relationship with LFPR. This correlation is not surprising, as drug use, drug dependence, and criminal behaviour have previously been reported to be highly associated with each other (DeLisi et al., 2015; Innes, 1988; Makkai & Payne, 2003). Notably, offenders using more severe drugs, such as cocaine, tend to commit more crimes than those using less severe drugs, such as cannabis (Bennett et al., 2008; Innes, 1988; Nurco et al., 1991), as do those with polydrug abuse and dependence (DeLisi et al., 2015). Although the

individual contributions of drug use, drug dependence and criminal behaviour to neural activity in offenders are hard to disentangle given their high collinearity, these results suggest that these factors should be taken into consideration when investigating the neural underpinnings of offenders, especially in offenders who present with more severe drug use and who have been convicted of more crimes.

Additionally, results showed that having been convicted of more crimes was particularly associated with decreased LFPR within the DMN, BG and Visual networks. These results align with previous reports associating disruptions within the DMN with antisocial behaviour. Indeed, offenders present various DMN disruptions, such as a lack of DMN de-engagement during tasks (Freeman et al., 2015), reduced DMN white matter diffusivity (Sethi et al., 2015) and decreased DMN connectivity during rest (Philippi et al., 2015; Pujol et al., 2012). Moreover, disruptions in white matter tract microstructures of the DMN (Waller et al., 2017) and decreased connectivity within the ventromedial prefrontal cortex (a central hub of the DMN; Raichle, 2015) have been related to heightened antisocial behaviour (Dugré & Potvin, 2021). As rest-related DMN activity is generally thought to support a self-referential and introspective state (Greicius et al., 2003; Raichle, 2015), the present finding of DMN oscillatory disruptions as a function of the number of criminal convictions could indicate disruptions in their introspective and self-referential abilities. The possibility that these disruptions could be associated with a decreased self-awareness of the impact of their antisocial behaviour is something that future research would be warranted to consider.

While activity within the basal ganglia (BG) network itself has not specifically been associated with antisocial behaviour, different structures of the BG network (e.g.,

striatum; Glenn & Yang, 2012, amygdala; Blair, 2007) present functional and anatomical disruptions in offenders that are directly related to heightened antisocial behaviour. Notably, offenders present an increased striatum volume which is associated with heightened antisocial behaviour (Korponay et al., 2017). In the same vein, the number of arrests has been positively associated with increased activity in the amygdala in reaction to fearful and angry faces in a sample of low-income men (Hyde et al., 2016), while in juvenile offenders, amygdala reactivity to angry faces has been shown to be related to the severity of their antisocial behaviour (i.e., increased frequency of aggressive and delinquent behaviours; Dotterer et al., 2017). Additionally, decreased amygdala volume has been associated with past and future antisocial behaviour (Pardini et al., 2014). The striatum and the amygdala are two structures of the BG network that are part of the limbic system, and both are involved in emotion and motivation (Cardinal et al., 2002; Satterthwaite et al., 2011). Therefore, the increased BG oscillatory disruptions could relate to increased disruptions in offenders' emotions processing, leading offenders with abnormal emotional reactions to commit more crimes.

Activity within the visual network, on the other hand, is not typically associated with antisocial behaviour. While some work has identified disruptions in neural activity in occipital regions in offenders and associated these disruptions with a reduced ability to identify emotions when viewing faces (Contreras-Rodriguez et al., 2014; Decety et al., 2014), to my knowledge, no direct link has previously been established between activity within the visual network and criminal behaviour. However, the implications of low-level processing disruptions in antisocial individuals have been increasingly discussed in recent years. Notably, offenders with heightened psychopathic traits present decreased activity

in the visual cortex when passively viewing emotion-laden stimuli (Anderson et al., 2017). Interestingly, this effect is not accompanied by variations in amygdala activity, thus suggesting that it represents disruptions in visual perception processes, which might disrupt attention processes, rather than a disruption of the emotional reaction to the stimulus (Anderson et al., 2017). Moreover, offenders with heightened psychopathic traits present a disrupted shift between rest-mode (i.e., DMN activity) and engagement on a task (i.e., salience network activity; Freeman et al., 2015) even when performing baseline task conditions (Anderson et al., 2018). This effect has been suggested to indicate disruptions in low-level attention processing in offenders with heightened psychopathic traits, which could undermine their ability to pay attention to stimuli that would typically be salient, such as emotionally relevant information (Anderson et al., 2018). Thus, my result of decreased LFPR in the visual network in offenders who had been convicted of more crimes provides further support to this literature by suggesting that disruptions in low-level visual processing are associated with increased criminal activity in offenders. Combined with results from Contreras-Rodriguez et al. (2014) and Decety et al. (2014), this deficit could be linked with a decreased ability to perceive emotional facial expressions. However, future work should explore the relationship between rest-related neural oscillation disruptions in the visual network and antisocial behaviour in offenders.

3.4.3. Relationship between the types of previous convictions and power spectra

Another secondary goal of the study was to assess the potential relationship between the different types of crimes that offenders had been convicted of and disruptions in rest-related power spectra. It is important to reiterate that these results should be interpreted

with caution as cell sizes for certain crime types were relatively small, and no main effect of group was obtained. Nonetheless, results showed that decreased levels of global LFPR were associated with a history of drug-related convictions, driving while intoxicated (DWI) and other driving offences in offenders. Of note, these three types of crimes are all non-violent. Thus, offenders with decreased rest-related LFPR were more likely to have been convicted of non-violent offences such as drug-related offences, DWI and other types of driving offences. It may be that these effects relate to the relationship between these types of crimes and substance use. Indeed, drug-related offences (i.e., drug possession and drug distribution) and DWI offences are often consequences of substance use. Moreover, risky driving and a history of driving offences are associated with the use of substances such as cigarettes, marijuana and alcohol (Shope et al., 2001). Thus, it could be that the relationship observed between decreased LFPR and having been convicted of these crimes might be associated with the substance use in these offenders. Although it is complex to disentangle drug use and criminal history's contributions to offenders' neural activity, substance use's influence on the integrity of rest-related power spectra in offenders convicted of these crimes should be explored in future studies.

3.4.4. Prediction of recidivism

A tertiary goal of this study was to assess whether rest-related power spectra could improve the prediction of recidivism compared to traditional risk assessment metrics. The machine learning analyses showed that global rest-related neural oscillations only modestly improved recidivism prediction after one year when added to a model that included age, PCL-R, number of previous criminal convictions and total drug use. Thus, these results suggest that although global LFPR somewhat improved the traditional

predictive model, it might not be the strongest predictor of recidivism. Metrics of neural activity other than rest-related power spectra might be stronger additions to our ability to predict future crimes.

These results differ somewhat from Delfin et al.'s (2019) study, which identified that combining SPECT rest-related neural activity in eight ROIs with a traditional risk-assessment model improved recidivism prediction accuracy from 0.64 to 0.82. However, it is important to note that various methodological differences exist between my study and Delfin and colleagues' (2019), which could explain this difference in outcome. First, both studies measured a different signal of rest-related neural activity, with Delfin et al. (2019) using SPECT to measure regional cerebral blood flow, while I measured the magnitude of electric activity. Thus, it could be that rest-related regional cerebral blood flow, but not neural oscillations, are predictive of recidivism. Additionally, Delfin et al. (2019) combined a small dataset with a complex decision tree algorithm which reduces the power and generalization of their results. While I also did not use a large dataset, I chose an algorithm and cross-validation technique that have demonstrated adequate power and reliability considering the size of my sample (Beleites et al., 2013). Finally, Delfin et al.'s (2019) included neural activity covering most of the brain, except for occipital and insular lobes and basal ganglia regions, while I used global power spectra activity. Thus, it could be that rest-related occipital, insular and basal ganglia activity might show a reduced relationship with recidivism than other regions of the brain. However, this relationship should be further investigated in future studies.

While I found a moderate relationship between rest-related neural activity and recidivism in offenders, other work has identified strong relationships between task-based

neural activity and recidivism (Aharoni et al., 2014; Steele et al., 2015). For instance, Aharoni et al. (2014) demonstrated that offenders' ACC response during an inhibitory task could predict re-arrest after a one-year follow-up with an accuracy of 0.68 *area under the curve* (AUC) using a *receiver operating characteristic* (ROC) curve analysis. In the same vein, Steele and colleagues (2015) have shown that combining fMRI measured activity of the ACC during a Go/NoGo task with event-related potentials (ERPs) measured by EEG during the same task increased the predictive accuracy of the model presented by Aharoni and colleagues (2014). The improved model classified whether or not offenders would be re-arrested after a one to four-year follow-up with an overall accuracy of 78.05%, albeit on a modest sample (N = 45; Steele et al., 2015). Thus, our results, combined with previous research, suggest that rest-related power spectra might not be the best predictor of recidivism and that inhibition-related neural activity might be more strongly predictive of recidivism in offenders. Alternatively, it could be that other baseline measures of neural activity, such as regional activity (Delfin et al., 2019), or the yet untested rest-related FNC activity, could be more accurate predictors of recidivism than rest-related neural oscillations. However, the relationship between alternative metrics of rest-related neural activity and criminal recidivism should be further explored in future work.

Nonetheless, a few caveats should be noted to understand these results better. First, it is important to note that recidivism variability was low in the offender group, with only ten offenders convicted of a crime within the follow-up year. Thus, it could also be that my offender group did not present enough recidivism variability to assess power spectra's ability to predict future crimes accurately.

Also, it is important to note that PCL-R did not show a strong predictive accuracy for recidivism. Indeed, PCL-R presented a low and non-significant relationship with recidivism in my sample (r = -0.03, p = 0.83). In contrast, previous accounts have indicated that PCL-R presents a positive and significant relationship with recidivism. Indeed, a meta-analysis identifies an R-value of 0.28 (Gendreau et al., 1996), while other work shows that PCL-R is predictive of general recidivism with an AUC of around 0.60 (Olver & Wong, 2015). Thus, another possibility is that there are issues with the validity of my recidivism measure itself. This could be explained by the restrictive nature of my recidivism measure, as I limited recidivism to criminal convictions to mirror my measure of past convictions. However, previous measures of recidivism have used a wider scope by including either arrest, conviction, incarceration, parole violation or a combination of those criteria (Aharoni et al., 2014; Gendreau et al., 1996; Olver & Wong, 2015; Steele et al., 2015). Thus, it is possible that the lower predictive ability demonstrated in my predictive models in comparison to previous work using neural activity and/or measures of antisocial traits could be attributed to a difference in recidivism measure, by which others have used a wider definition of recidivism leading to more variability in their sample.

3.4.5. Limitations

A broader discussion of limitations pertaining to fMRI-based power spectra and the use of a male sample can be found in Simard et al. (2021). Some additional limitations pertaining to the collection of criminal history data should be noted, however. These background checks were performed using two public databases that may or may not provide a complete reflection of each participant's total criminal activity. The first

database (i.e., New Mexico courts online offence database) offered official conviction records, but was restricted to criminal convictions within the state of New Mexico. The second database (i.e., www.instantcheckmate.com) providing criminal convictions from other states; however, it may or may not entirely reflect official governmental records. Moreover, none of the databases allowed me to search for federal convictions. Thus, while the use of two corresponding methods was a strength of the study design, there may nonetheless be some limitations to the exhaustiveness or accuracy of the conviction data used.

Another limitation pertains to the one-year follow-up period utilized in this study (which was the maximum duration of time we had ethical clearance to follow participants for). Indeed, during the one-year follow-up, 12.7% of the offenders included in this sample were convicted of a crime. The variability of the recidivism data may consequently not have been sufficient to assess a relationship with power spectra activity. Indeed, a recent meta-analysis has estimated that reconviction rates can oscillate between 20 to 63% after a two-year follow-up (Yukhnenko et al., 2019). Thus, a longer follow-up could have yielded more variability in recidivism data and a potentially stronger relationship with our predictors.

Finally, I should note that power spectra is only one possible neural integrity metric. As such, the extent to which these results could be used to generalize about overall neural integrity in offenders may be preliminary for now. Indeed, violent offenders may differ in other neural features not investigated in this study. Candidates include functional connectivity or task-based activation patterns. Thus, future work

should investigate offenders' neural integrity as a function of violent offending history in additional neural features.

Chapter 4: Study 3

4.1. Introduction

Studies 1 and 2 together suggest that offenders present disrupted resting-state neural oscillations, illustrated by both decreased low-frequency activity and increased high-frequency activity. This is broadly consistent with other work identifying neural abnormalities associated with offending (see, for instance, Contreras-Rodriguez et al., 2014; Jiang et al., 2017; Juarez et al., 2013; Kiehl et al., 2006), but extends evidence of these dysfunctions to rest-related spectral power. Evidence of differential neural features in offenders has also been reported in task-based neural activity. Moreover, there is some suggestion that subtypes of offenders may be characterized by unique neural patterns at times (see the General Introduction for a review of this work). However, these literatures still require further consideration. For instance, while there is some evidence of neural disruptions in offenders, it remains unknown whether these observed task-based neural markers are features of all offenders, or are more characteristics of certain offender subtypes. Additionally, it remains largely unknown whether these abnormalities are more likely to manifest in certain processing domains than others (e.g., cognitive versus emotional domains). Establishing such distinctions could help more precisely characterize the nature of neural abnormalities within offending populations, allowing for a better focus of research efforts on the neural underpinnings of offending.

A consolidation of the task-based neural abnormalities associated with offending may be achieved through meta-analytic methods. Individual empirical studies can have a variety of methodological characteristics that limit their interpretability and reproducibility, which meta-analytic methods can help address. Indeed, fMRI studies

often use small sample sizes, which can cause the study to have low statistical power and, potentially, low reproducibility (Button et al., 2013). There are additional concerns regarding the interpretation of individual fMRI studies as well. First, small sample sizes increase the propensity for false-positive results (Wager et al., 2007); second, the high heterogeneity between study pipelines makes it difficult to compare results across studies (Carp, 2012). Meta-analysis can remedy these concerns by considering the results of individual empirical studies as a unified whole to identify regions of the brain that show the most consistent activation across studies. This can protect against the low power of individual studies and increase the overall reliability of results compared to the findings from any one individual study (Hedges & Olkin, 1985). In the context of neural activity, meta-analytic methods may help confirm whether certain neural markers are reliably observed across specific groups or subgroups (Tahmasian et al., 2019). To this end, Study 3 aimed to conduct a meta-analytic study of task-based neural activity in offenders that could help identify task-based markers that most reliably present in offenders and/or offender subgroups.

While no previous work has attempted to consolidate knowledge regarding task-based fMRI activity in offenders, several recent meta-analyses have come close by examining neural activity in a variety of (adult and/or child) populations known to exhibit heightened antisocial *traits* (Deming & Koenigs, 2020; Dugré et al., 2020; Nickerson, 2014; Poeppl et al., 2019; Yang & Raine, 2009). However, as reviewed in the General Introduction, these meta-analyses almost unanimously included studies that recruited based on antisocial *traits,* rather than antisocial *behaviour*, and thus included heterogeneous samples of offending and non-offending individuals. For instance, Yang

and Raine (2009) compared anatomical (i.e., MRI) and functional (i.e., SPECT and PET) prefrontal metrics across studies that compared a broad spectrum of individuals, recruited from both general and forensic populations, who held low versus high antisocial characteristics. Results indicated that heightened antisocial traits were associated with reduced anatomical volume and reduced functional activity within the right orbitofrontal, right anterior cingulate and left dorsolateral prefrontal cortices (Yang & Raine, 2009). Nickerson (2014) updated Yang and Raine's (2009) by adding additional work published between 2009 and 2014 and largely replicated the functional and anatomical findings. However, it should be noted that Yang & Raine (2009) and Nickerson (2014) both limited their search space to prefrontal regions; thus, their meta-analytic findings preclude the ability to offer a whole-brain representation of antisocial traits.

More recently, Dugré et al. (2020) used a *Seed-based d-Mapping with Permutation of Subject Images (SDM-PSI)* meta-analytical algorithm to assess the neural activity associated with conduct problems and ASPD in individuals recruited from both general and forensic populations. ROI-based results corrected for multiple comparisons indicated that *callous-unemotional* (CU) traits were negatively related to activity in the right amygdala. Whole-brain analyses identified additional negative associations between a) antisocial traits and activity in the anterior thalamic nuclei (bordering on the mammillary body) and the right amygdala, and b) callous-unemotional traits and activity in the right superior temporal gyrus (however, these whole-brain results did not survive multiple correction thresholding; Dugré et al., 2020). Importantly, 86% of the studies included in Dugré et al. (2020) used children and/adolescents as participants (14% of Yang & Raine's (2009) and 3% of Nickerson's (2014) included samples were of children and/or

adolescents). Thus, it is difficult to know if these results (and to a lesser extent those reported by Yang & Raine (2009) and Nickerson (2014)) would generalize to an adult offender population.

Finally, two additional meta-analyses have investigated neural activity specifically associated with heightened psychopathic traits (Deming & Koenigs, 2020; Poeppl et al., 2019). As presented in more detail in the General Introduction, these meta-analyses reported that heightened psychopathic traits were associated with neural activity in various cortical and subcortical regions. While both studies identified a relationship between heightened psychopathic traits and neural activity in the amygdala and dorsomedial cortex, this relationship was reported to be negative by Poeppl et al. (2019) and positive by Deming & Koenigs (2020). This difference in the directionality of the effects reported could be attributable to various methodological differences between these studies. For instance, Poeppl et al. (2019) used an Activation Likelihood Estimation (ALE) algorithm, which is thought to have the best implementation of the Monte Carlo test null distribution used to create inferences among coordinate-based meta-analysis methods (Samartsidis et al., 2017), while Deming & Koenigs (2020) used Multilevel Kernel Density analysis (MKDA) methods. Both methods use peak coordinates to assess the statistical convergence of neural activity across paradigms (Samartsidis et al., 2017). However, while in MKDA, a sphere size of a fixed diameter is used to weigh each focus, in ALE, foci are weighted using each experiment's sample size. It should also be noted that ALE and MKDA were not primarily designed to evaluate the directionality of effects. Moreover, although there is substantial overlap between the studies included in both meta-analyses (i.e., 23 studies in common), these differences in outcome could be

attributable to differences in the selection of experiments, as coordinate-based meta-analysis methods are highly sensitive to this choice (Samartsidis et al., 2017). Indeed, Deming & Koenigs (2020) included two studies for which experiments were not included in Poeppl et al. (2019), while Poeppl et al. (2019) included five studies and 68 experiments which were not included in Deming & Koenigs (2020). Nonetheless, these studies focused specifically on individuals with high psychopathic traits; thus, the extent to which their results will generalize to the offending population, in general, is unclear.

Taken together, these meta-analytic studies suggest that antisocial traits are most consistently related to activity disruptions in the prefrontal cortex (Deming & Koenigs, 2020; Nickerson, 2014; Poeppl et al., 2019; Yang & Raine, 2009) and the amygdala (Deming & Koenigs, 2020; Dugré et al., 2020; Poeppl et al., 2019), although there seem to be some inconsistencies regarding the directionality of these effects. However, it is important to keep in mind that, as presented in the General Introduction, these meta-analyses used samples from heterogeneous groups recruited from community/offender populations based on personality diagnoses/characteristics (i.e., ASPD, CD, psychopathic traits). On the other hand, only a minority of the samples recruited active offenders. This distinction is important, as antisocial *traits* and antisocial *behaviour* are only moderately related (Black et al., 2010; Forsman et al., 2010), indicating that antisocial traits are not always linked to offending behaviour. Consequently, the results of these meta-analyses do not fully inform of the specific contribution of antisocial behaviour to these task-based neural markers.

To my knowledge, only one meta-analytic study has focused specifically on the neural underpinnings of antisocial behaviour (albeit within the context of resting-state

functional connectivity (rs-FNC); Dugré & Potvin, 2021). Results of this meta-analysis indicated that individuals with higher levels of antisocial behaviour showed decreased rs-FNC within limbic, frontal, parietal and occipital areas, but increased rs-FNC in the ventral posterior cingulate. Moreover, the severity of the individuals' antisocial traits (as assessed via scores on scales measuring either hyperactivity/impulsivity, affective/interpersonal or antisocial behaviours/lifestyle traits) was negatively associated with rs-FNC in the ventromedial prefrontal cortex. This study provides valuable information regarding the integrity of baseline co-activation of brain regions in relation to antisocial behaviour. However, this meta-analysis included a large number of samples that were recruited based on non-criminal antisocial behaviour, more representative of behaviour observed in children/juveniles with a conduct disorder and which might not generalize to adult offenders. Thus, these results also cannot fully inform on the integrity of task-based neural activity associated with antisocial behaviour in adult offenders.

Thus, a whole-brain meta-analytic investigation of offenders' task-based neural activity, using studies that recruited exclusively from offender populations, is still lacking. To fill this gap, the primary goal of this study was to perform a meta-analysis of fMRI studies that previously investigated task-based neural activity differences between offender and non-offender populations. Doing so will provide a more direct and comprehensive mapping of neural markers related to *antisocial behaviour,* which can only be inferred by investigating the neural markers related to *antisocial traits.* Identifying such antisocial *behaviour*-relevant neural markers could further inform on the neural integrity of offender populations and may offer useful targets for considering novel treatment and prevention opportunities.

4.1.1. Influence of processing domains

Previous meta-analyses using populations with heightened antisocial traits have identified relationships between neural activity and antisocial traits in the context of specific cognitive and emotional processing domains (Dugré et al., 2020; Poeppl et al., 2019). Indeed, Dugré et al. (2020) investigated the neural activity associated with conduct problems and ASPD within five specific processing domains: acute threat response, cognitive control, social cognition, punishment, and reward processing (albeit using a sample of studies mostly representative of children and adolescents). ROI-based analysis revealed that CU traits were negatively related to activity in the right amygdala during social cognition, and negatively related to activity in both the left and right amygdala during threat detection (additional whole-brain analyses found further relationships associated with acute threat, cognitive control, punishment processing and social cognition; however, these results did not survive correction for multiple comparisons; Dugré et al., 2020). Additionally, Poeppl et al. (2019) compared the neural clusters identified in their meta-analysis with BrainMap meta-data. They found that activity negatively associated with psychopathic traits tended to be associated with action execution (i.e., right lateral prefrontal cortex), language processing (i.e., left lateral prefrontal cortex) and emotion processing (i.e., amygdala) domains (Poeppl et al., 2019). This work suggests that heightened antisocial traits relate to aberrant neural activity in both emotional and cognitive processing domains. However, the heterogeneous nature of the samples makes it difficult to assess whether these neural abnormalities relate most closely to heightened antisocial *traits* or heightened antisocial *behaviours*.

Consequently, I wished to assess if offenders' neural abnormalities occur more reliably in one processing domain than the other (i.e., emotional and cognitive). To this end, I undertook ALE analyses separately for experiments that evaluated neural responses in the context of emotional and cognitive processing domains. Performing this analysis allowed me to explore whether neural inefficiencies in offenders are specific to emotional/cognitive processes. Doing so may help hone in on the processing inefficiencies associated with offending behaviour and thus establish more precise targets for future research and interventions with offenders.

4.1.2. Influence of crime type

As previously reviewed above (see section 1.2.4.1 of Chapter 1), while a modest literature has identified neural differences associated with specific types of offenders, very little is currently known about brain structure/function differences associated with different criminal acts. Literature on the neural underpinnings associated with specific types of crimes has generally focused on violent versus non-violent offenders, and has generally reported differences in neural activity (da Cunha-Bang et al., 2017) and grey matter volume (Schiffer et al., 2011) in various limbic system regions. Several other studies have investigated more specific crime types. For instance, work has identified aberrant specific neural features in pedophilic offenders (Poeppl et al., 2015; Polisois-Keating & Joyal, 2013), non-pedophilic sexual abusers of children (Kärgel et al., 2015; Kneer et al., 2019), sexual abusers of adults (Cazala et al., 2020; Chen et al., 2016), homicidal offenders (Cope, Ermer, et al., 2014; Sajous-Turner et al., 2019), women batterers (Bueso-Izquierdo et al., 2016; Lee et al., 2009), and white-collar criminals (Ling et al., 2019; Raine et al., 2012). Thus, specific neural disruptions appear associated with

having committed specific types of crimes. However, no clear or consistent pattern has yet emerged, and considerably more work could thus be undertaken in this area. Thus, the current meta-analysis also aimed to assess the extent to which neural dysfunctions in offenders relate to their propensity to commit specific crimes. To account for the limited work to date and the restricted power associated with specific subtypes of crime, I primarily utilized a high-level classification system, classifying crimes into violent and non-violent subtypes. Additional exploratory analyses investigated more specific crime types, which may offer preliminary insights into more subtle neural/crime relationships.

4.2. Methods

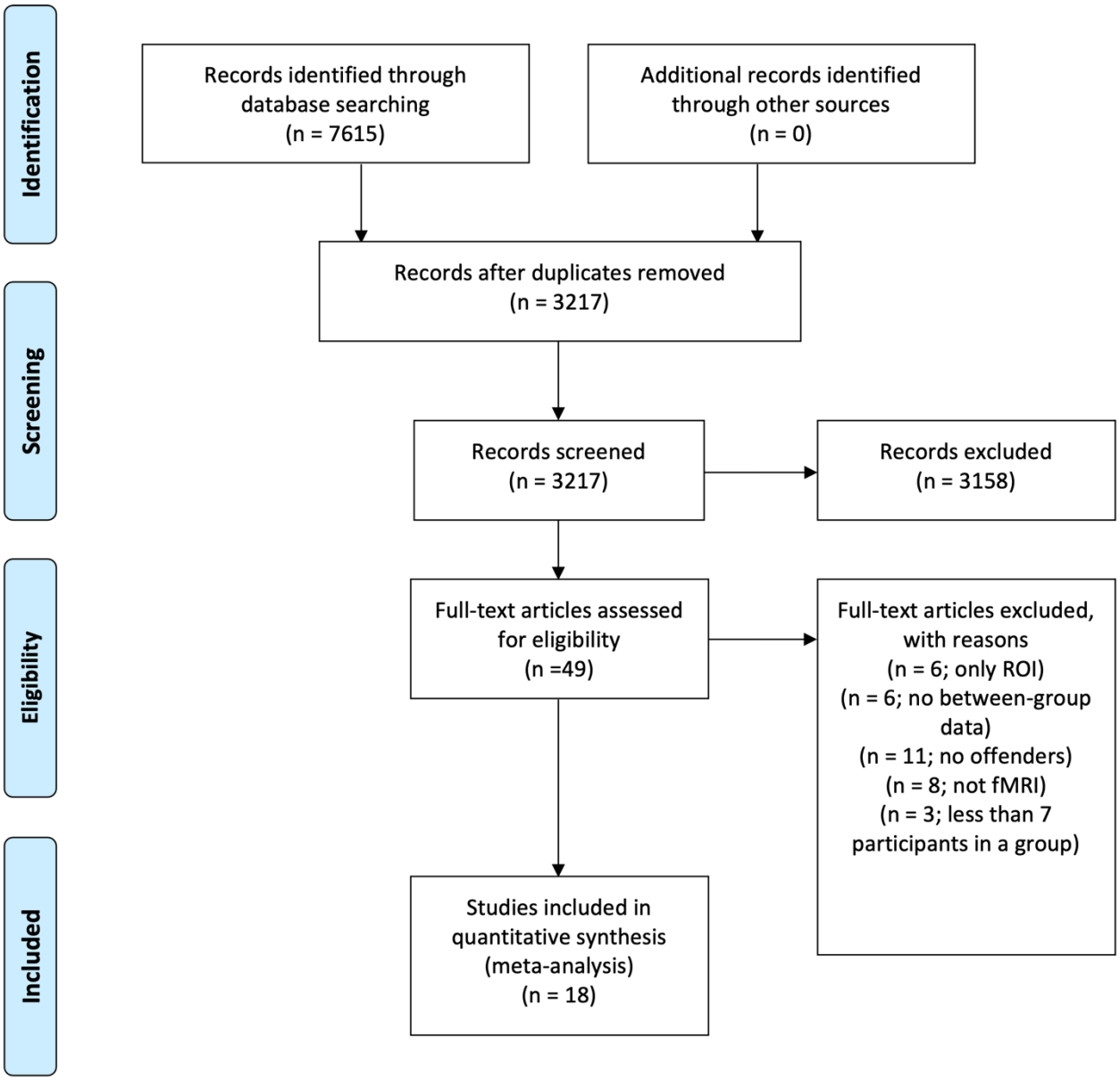
4.2.1. Data Selection

I performed literature queries on PubMed and PsycINFO databases with the following combination of keywords [‘Psychopathy’ OR ‘Antisocial’ OR ‘Offenders’ OR ‘Psychopathic traits’ OR ‘Offend*’ OR ‘Inmate*’ OR ‘Probation*’ OR ‘Pedophil*’ OR ‘Antisocial*’ OR ‘Parole*’ OR ‘Crime’ OR ‘Criminal*’ OR ‘Psychopaths’ OR ‘Psychopathic’ OR ‘Prisoner*’ OR ‘Felon*’ OR ‘Convict*’ OR ‘Prison*’ OR ‘Jail*’ OR ‘Imprison*’ OR ‘Detention’ OR ‘Correctional’ OR ‘Forensic’ OR ‘Sentenced’ OR ‘Detainee*’ OR ‘Penal’ Or ‘Incarcerate*’] AND [‘fMRI’ OR ‘functional MRI’ OR ‘functional magnetic resonance’ OR ‘PET’ OR ‘positron emission’ OR ‘ASL’ OR ‘Arterial spin labelling’ OR ‘MEG’ OR ‘magnetoencephalography’ OR ‘neuroimaging’] on December 9th, 2020, in search of articles that used fMRI to compare neural activity between offender and non-offender samples. After eliminating duplicates, I identified 3217 relevant articles (see Figure 4.1 for PRISMA workflow chart). Abstract screenings

were performed and double-rated using Rayyan software (Ouzzani et al., 2016) by myself and a trained research assistant.

Figure 4.1

Study flow diagram of the meta-analysis' literature screening process based on PRISMA model (Moher et al., 2009)



Abstract screening inclusion criteria included a) recruitment of at least one adult offender sample and one adult non-offender sample, b) reporting of fMRI analysis, and c) English language articles. Articles went through a two-stage screening process, with articles first screened at the abstract level, followed by a full-text review of all remaining articles. Articles were excluded based on the abstract if a) they were not written in English (N = 99), b) they did not recruit adult offender and non-offender samples (N = 1536), c) they did not report empirical fMRI results (e.g., meta-analysis, theory-based articles, other fields of research; N = 2072) or d) they were not peer-reviewed research articles (e.g., letter to the editor, dissertation; N = 40). It is important to note that some articles were excluded for multiple reasons (thus, total exclusions could exceed 100% of all articles reviewed). Based on these criteria, I selected 49 articles for full-text screening.

Full-text inclusion criteria screened out studies that a) did not report group-level contrasts; N = 6), b) did not report fMRI results (e.g., only FNC; N = 8), c) recruited a heterogeneous participant group of offenders and non-offenders (N = 11), d) did not meet minimum sample size recommendations for inclusion in meta-analytic studies (Tahmasian et al., 2019; N = 3). Additionally, only studies reporting whole-brain analyses with coordinates presented in a standard reference space (Talairach-Tournoux or Montreal Neurological Institute (MNI)) were included; studies only reporting Region of Interest (ROI) analyses (N = 6) were excluded. This was necessary to ensure that all experiments were analyzed in the same search space, without biasing findings towards the smaller search spaces created by ROI analyses (Müller et al., 2018). Articles presenting more than one exclusionary characteristic are represented in more than one category.

After the full-text screening, I selected 18 articles for between-group analyses totalling 22 samples (one task contrast per sample) of direct group comparisons (i.e., offenders > non-offenders and non-offenders > offenders), 146 foci and 766 participants (368 offenders and 399 non-offenders). The number of samples included falls within the recommended guidelines to afford sufficient power for an ALE analysis (i.e., a minimum of 17-20 samples; Eickhoff et al., 2016). A list of the articles, contrasts, number of foci and number of participants included in the meta-analysis is presented in Table 4.1.

Table 4.1

Characteristics of the articles and contrasts included in the meta-analysis

Article	First author	Year	Participants		Task	Subject-Level analysis
			O	Ctls		
1	Contreras-Rodriguez	2014	22	22	Emotional face-matching task	Emotional faces > shapes
2	Gregory	2015	12	18	Probabilistic response-reversal task	Punished reversal errors > rewarded correct responses
			12	18	Probabilistic response-reversal task	Rewarded correct responses > punished reversal errors
3	Joyal	2007	24	12	Go/NoGo	Go/NoGo > baseline
			12	12	Go/NoGo	Go/NoGo > baseline
4	Kargel	2017	40	37	Go/NoGo	NoGo>Go
5	Lee	2009	10	13	Picture viewing task	Aggressive-threat pictures > Neutral pictures
6	Massau	2017	16	19	Moral judgement task	Sexual offence against children > Sexual offence against adults
7	Meffert	2013	18	26	Observation of hand interactions	Empathy > Rest
8	Mier	2014	11	18	Social cognitive paradigm	Theory of Mind
9	Prehn	2013a	12	13	Financial decision-making task	Increasing uncertainty
			11	13	Financial decision-making task	Loss > bond
10	Prehn	2013b	15	17	Emotional n-back task	High saliency negative emotional pictures
11	Pujol	2012	22	22	Moral processing + Stroop	Moral dilemma task
12	Ristow	2019	13	13	Viewing sexually arousing images	Sexual adult picture viewing > sexual child picture viewing
13	Schiffer	2008a	8	12	Viewing sexually arousing images	Nude girls > Rest
14	Schiffer	2008b	11	12	Viewing sexually arousing images	Nude boys > dressed boys
15	Schiffer	2014	21	23	Non-verbal Stroop task	Error-related activity
16	Schiffer	2017	13	18	Mental state decoding from visual stimuli	Mental state attribution > Gender Discrimination
			16	18	Mental state decoding from visual stimuli	Mental state attribution > Gender Discrimination
17	Tonnaer	2017	16	18	Provocation and regulation task	Anger engagement > Neutral engagement
18	Vollm	2010	25	25	Go/NoGo	NoGo>Go

Note. Offenders (O), Non-Offenders (Ctls)

Table 4.1 (cont.)

Characteristics of the articles and contrasts included in the meta-analysis

Article	First author	Year	Group-Level Analysis	Task category	Violence category	Foci
1	Contreras-Rodriguez	2014	O > Ctls	Emotion	Violent	5
2	Gregory	2015	O (ASPD + Psychopath) > Ctls	Cognition	Violent	6
			O (ASPD) < Ctls	Cognition	Violent	3
3	Joyal	2007	O (Sz + ASPD + SUD) vs Ctls	Cognition	Violent	10
			O (Sz) vs Ctls	Cognition	Violent	6
4	Kargel	2017	Ctls > O	-	Pedophilia	3
5	Lee	2009	O vs Ctls	Emotion	Violent	14
6	Massau	2017	O < Ctls	Cognition	Pedophilia	3
7	Meffert	2013	O (Psychopath) vs Ctls	Cognition	-	33
8	Mier	2014	O (Psychopath) vs Ctls	Cognition	Violent	2
9	Prehn	2013a	O vs Ctls	Cognition	Violent	5
			O vs Ctls	Cognition	Violent	2
10	Prehn	2013b	O (ASPD + BPD vs Ctls	Emotion	-	2
11	Pujol	2012	O (Psychopath) > Ctls	Cognition	Violent	5
12	Ristow	2019	O vs Ctls	-	Pedophilia	7
13	Schiffer	2008a	Ctls > O	-	Pedophilia	6
14	Schiffer	2008b	O > Ctls	-	Pedophilia	4
15	Schiffer	2014	O vs Ctls	Cognition	Violent	9
16	Schiffer	2017	O (Sz + CD/ASPD) > Ctls (Sz)	Cognition	Violent	6
			O (Sz) > Ctls (Sz)	Cognition	Violent	2
17	Tonnaer	2017	O vs Ctls	Emotion	Violent	3
18	Vollm	2010	O vs Ctls	Cognition	-	5

Note. Offenders (O), Non-Offenders (Ctls), Antisocial Personality Disorder (ASPD), Schizophrenia (Sz), Substance Use Disorder (SUD), Borderline Personality Disorder (BPD)

4.2.2. Activation likelihood estimation (ALE)

I performed ALE analyses using GingerAle 3.0.2 (Turkeltaub et al., 2012) following guidelines by Eickhoff (2012). ALE is a coordinate-based meta-analysis algorithm, which identifies brain regions showing a consistent response across experiments. It seeks to refute the null hypothesis that the foci of experiments are spread uniformly throughout the brain. ALE is the most widely used method of coordinate-based meta-analysis (Samartsidis et al., 2017) and provides qualitatively similar results as other types of kernel-based meta-analysis (i.e., Multilevel kernel density analysis (MKDA), Signed differential mapping (SDM); Radua et al., 2012; Samartsidis et al., 2017) and model-based methods (e.g., Bayesian models; Samartsidis et al., 2017).

In my ALE analyses, I included foci resulting from a whole-brain search space corrected for multiple comparisons and the number of participants included in each sample. I included foci thresholded using a small volume correction if they also reached whole-brain thresholds. When required, foci were converted from Talairach to MNI space as I conducted all analyses in the MNI space. During ALE analysis, for each experiment, a Gaussian distribution was applied to each focus with an FWHM weighted by the number of participants included in each sample to create Modeled Activation (MA) maps. Next, ALE maps were created by the union of all MA maps and ALE scores were attributed to each focus based on union-derived probabilities. Finally, a histogram-based permutation procedure tested the difference between true convergence of foci and noise-attributed signal. All analyses were cluster-thresholded using a cluster forming threshold of $p < 0.001$, p (FWE) < 0.05 corrected, with 1000 permutations.

4.2.3. Main effects of offenders vs. non-offenders

To assess the neural activity associated with offending behaviour, I selected contrasts comparing offenders and non-offenders. Most articles only reported task-contrasts for which significant results were available in one direction (e.g., Offenders > Controls or Controls > Offenders). However, when an article reported a task-contrast for which significant results were available in both directions, the foci of both between-group contrasts were combined to form a single sample (11 such instances were detected and are marked as Offenders vs Controls in the "Group-level Analysis" column of Table 4.1). Only one task contrast was selected per sample to reduce the risks of within-sample dependency. This selection was made by ensuring that the most representative contrast was included in the meta-analysis. For example, in a Go/NoGo task, if both NoGo > Go and NoGo > Rest contrasts were performed in an article, only the NoGo > Go contrast was included in the meta-analysis as it is a more direct measure of the neural activity related to NoGo than the other contrast and including both contrasts would be repetitive (see Table 4.1 for a list of the contrasts included; consensus agreement regarding the contrasts included was established between myself and Dr. Shane). I also wished to assess the directionality of any activity differences observed between offenders and non-offenders. To do so, I pooled the between-group contrasts/foci into two groups representing the Offender > Controls contrast (18 samples, 86 foci, 300 offenders vs 335 controls) or the Controls > Offenders contrast (16 samples, 78 foci, 268 offenders vs 283 controls).

4.2.4. Relevance of processing domain.

To assess the extent to which neural abnormalities were more reliably identified within either emotional or cognitive processing domains, I grouped contrasts according to the processing domain of the task performed. Hence, I separated contrasts into two groups that evaluated neural responses within emotional contexts (e.g., emotion recognition; 5 samples, 57 foci, 81 offenders vs 96 controls) and within cognitive contexts (e.g., Go/NoGo; 15 samples, 70 foci, 270 offenders vs 282 controls; see Table 4.1 for a list of contrasts included in each group).

4.2.5. Relevance of the violent nature of crimes.

To assess the extent to which neural abnormalities differed depending on the violent nature of the committed crime, I separated studies into those that a) included samples within which all participants had committed violent offences (e.g., murder, sexual offences, grievous bodily harm; 19 samples, 101 foci, 310 offenders vs 330 non-offenders) and b) did not explicitly select participants based on the presence of a violent offence (which formed a mixed offence group; 3 samples, 40 foci, 58 offenders vs 68 non-offenders). Interestingly, five experiments exclusively recruited pedophiles. As previous studies have reported neural markers associated with pedophilic offending (Poeppl et al., 2015; Polisois-Keating & Joyal, 2013) and sexual offending (Cazala et al., 2020; Chen et al., 2016; Kargel et al., 2015; Kneer et al., 2019), I sought to explore the neural activity related to sexual offending and non-sexual offending, respectively. Consequently, I also ran exploratory analyses to compare samples that recruited pedophile offenders (5 samples, 23 foci, 88 offenders vs 92 non-offenders) and samples that recruited non-sexual violent offenders (14 samples, 78 foci, 222 offenders vs 238

non-offenders). A summary of the sample characteristics of each study included in the meta-analysis is provided in Table 4.1.

4.2.6. Exploratory: Functional characterization

Two different but complementary techniques, each with its advantages and drawbacks, can be used to glean information related to the behavioural functions that may underlie specific neural abnormalities: forward-inference and reverse-inference. Forward-inference can be used to infer a context-specific indication of the behavioural domain associated with a particular brain area (Henson, 2005). For instance, based on observations that fearful face expressions relate to increased activity in the amygdala, it can be inferred that amygdala activity may be associated with a fear response to the perception of specific facial expressions. While this inference can provide considerable insight, forward inference cannot indicate if the observed association generalizes to other contexts. For instance, we cannot conclude from amygdala activity to fearful faces that the amygdala is involved in all types of fear responses or triggered by all fear-inducing stimuli.

On the other hand, reverse-inference can help estimate the behavioural domains with which a specific neural activity cluster is more generally associated by comparing it to results obtained by previous studies stored in a database (Poldrack, 2006, 2011). For instance, through data-driven approaches with databases such as BrainMap and Neurosynth, we can statistically estimate the behavioural domains with which a specific amygdala cluster has been shown to be associated using meta-data taken from thousands of publications (Poldrack, 2011). However, reverse inference can also be misused when conclusions are drawn from a small set of literature (Poldrack, 2006). Indeed, it is

relatively common to conduct a literature search to identify which behavioural functions a particular neural area underserves when interpreting results. This can lead researchers to draw conclusions from a few studies, which might not capture the full scope of the literature available and does not necessarily reflect the full array of behavioural functions in which this specific neural region can be involved. However, this issue can be circumvented with the use of data-driven databases (e.g., BrainMap and Neurosynth). Additionally, reverse-inference results should be interpreted with caution as a specific brain area is often involved in many behavioural domains and reverse-inference analysis only presents a statistical probability of the relationship between specific brain areas and behavioural domains, rather than an absolute relationship.

Given both approaches' strengths and weaknesses, I undertook both forward- and reverse-inference functional characterization of the activity clusters identified during my ALE analyses. To assess forward inference, I listed the meta-data experiments that statistically contributed to the activity clusters identified in the ALE analysis within GingerAle outputs. To assess reverse-inference, I used BrainMap meta-data (which is less error-prone than the Neurosynth method; Omlor et al., 2019) through the Behavioral Analyses plugin v3.1 (Lancaster et al., 2012) in MANGO image viewer v4.1 (Research Imaging Institute, UT Health Science Center at San Antonio, TX, USA; http://ric.uthscsa.edu/mango/). First, I transformed ALE maps in Talairach space using the MNI-to-Tal transform option in MANGO. Second, I shrink-wrapped an ROI on each cluster of activity obtained in the ALE results. Each ROI was statistically compared to foci stored in the BrainMap database using the Behavioral Analyses plugin. This plugin allowed me to make statistical inferences on the behavioural domain associated with the

neural regions identified through the ALE analysis. I considered only the behavioural domains showing a probability higher than z > 3 ($p < 0.05$ Bonferroni correction for multiple comparisons) significantly related to the neural region identified.

4.3. Results

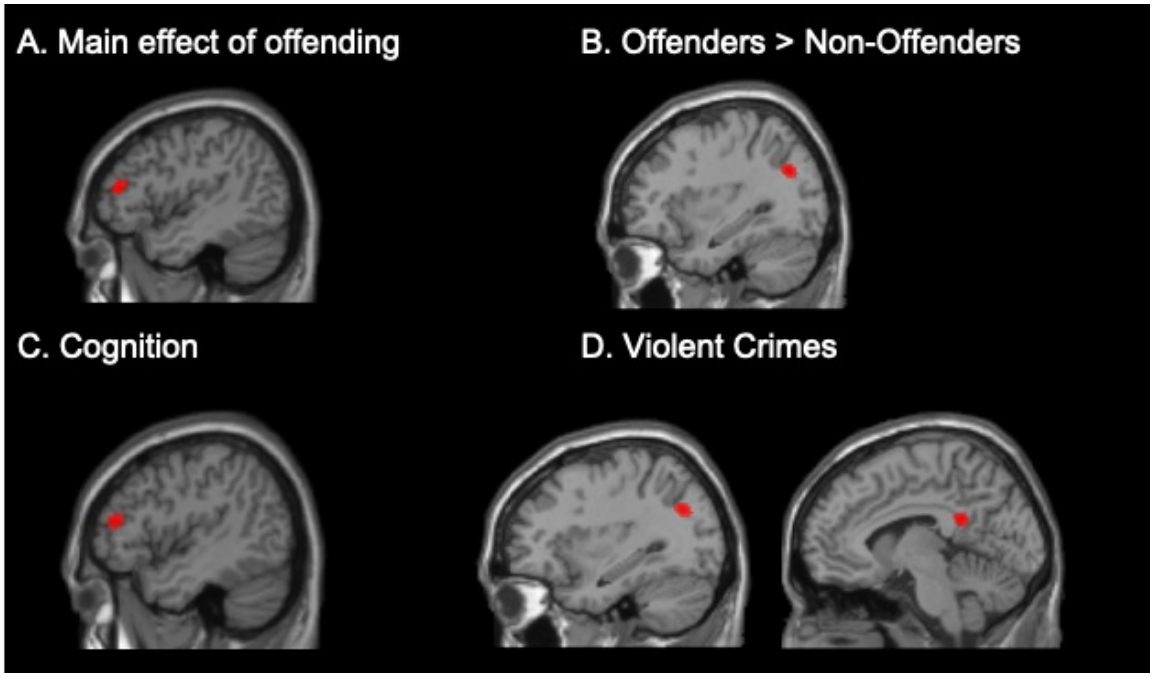
4.3.1. Offenders vs non-offenders

Mean age of offenders and non-offenders across all studies included in the meta-analysis was 38.5 and 38, respectively ($t = 0.97$, $p = 0.34$). All participants were male; 8 offenders were on medication (1.90%), while only one non-offender was medicated (0.25%). Among offenders, 85 (23%) were diagnosed with psychopathy, 153 (38%) with antisocial personality disorder, 27 (7%) with a borderline personality disorder, 65 (18%) with schizophrenia, 24 (6.5%) with a substance use disorder and 88 (24%) with pedophilia. It is important to note that some offenders presented more than one diagnosis.

A main effect of offending was identified within a cluster that included the left inferior frontal gyrus (see Table 4.2 and Figure 4.2.A.). The offender > control analysis indicated that offenders presented increased activity in the left middle occipital gyrus compared to non-offenders (see Figure 4.2.B.). The control > offender analysis yielded no clusters with significant differences.

Figure 4.2

Neural activity clusters identified in the activation likelihood estimation analyses



Note. Results of the Activation Likelihood Estimation (ALE) analysis showed that offending behaviour (A) and cognitive processing (C) were associated with a cluster of activity in the left inferior frontal gyrus, while an increase of activity in offenders compared to non-offenders (B) was associated with a cluster of activity in the left middle occipital gyrus. Violent crimes (D) were associated with clusters of activity in the left middle occipital gyrus and the left posterior cingulate cortex.

$p < 0.05$ *FWE*, cluster threshold $p < 0.001$ unc., 1000 permutations

Table 4.2

Main results of the meta-analysis

Anatomical Location	Cytoarchitectonic location	Cluster size in mm^3	MNI coordinates			ALE score	Z score
			x	y	z		
Offenders vs non-offenders:							
Left inferior frontal gyrus	BA 46	776	-44.8	41.2	15.6	0.017	4.77
Offenders > Non-Offenders:							
Left middle occipital gyrus	BA 19	760	-31.9	-65.8	30.2	0.015	4.71
Non-Offenders > Offenders:							
No significant results							
Emotion:							
No significant results							
Cognition:							
Left inferior frontal gyrus	BA 46	872	-44.8	41.6	15.2	0.017	5.17
Violent + Pedophilic crimes:							
Left middle occipital gyrus	BA 19	736	-32	-65.8	30.1	0.015	4.58
Left posterior cingulate cortex	BA 23	688	-6.3	-43.4	24.5	0.016	5.78
Violent crimes:							
No significant results							
Pedophilic crimes:							
No significant results							

Note. Brodmann Area (BA), Montreal Neurological Institute (MNI), Activation Likelihood Estimation (ALE).

$p < 0.05$ *FWE*, cluster threshold $p < 0.001$ unc., 1000 permutations

4.3.2. Relevance of processing domain

Studies that targeted cognitive-related processes were associated with a cluster of activity in the left inferior frontal gyrus (see Figure 4.2.C.). No significant activity pattern was identified for experiments that targeted emotion-related processes.

4.3.3. Relevance of the violent nature of crimes

Having a history of violent offences was associated with activity within the left middle occipital gyrus and the left posterior cingulate cortex (see Figure 4.2.D.). No

significant clusters were identified when separately investigating mixed-violence offenders, pedophilic offences, and non-sexual violent crimes.

4.3.4. Functional characterization: Forward-inference

GingerAle produces a list of the experiments with foci inside the boundaries of the significant clusters identified during the ALE analysis. This list may offer some insight into the neurocognitive processes that are associated with this brain region. This method indicated that the IFG (where activity was associated with having a history of offending, and most specifically when cognitive processes were interrogated) is often related to error processing (Schiffer et al., 2014), *Theory of Mind* (TOM; Schiffer et al., 2017), and response inhibition (Vollm et al., 2010). Similarly, this method indicated that the left MOG (where activity was higher in offenders compared to non-offenders, and particularly so for violent offenders) is often related to TOM (Schiffer et al., 2017) and threat perception (Lee et al., 2009). Finally, the left PCC (which was specifically associated with the commission of a violent crime) has been related to response inhibition (Kargel et al., 2017) and threat perception (Lee et al., 2009).

4.3.5. Functional characterization: Reverse-inference

The reverse-inference analyses did not uncover any significant relationship between the activity clusters identified in the ALE analyses and specific neurocognitive processes.

4.4. Discussion

This study is the first ALE-based meta-analysis of task-based neural activity in offenders. Results revealed that offender status was associated with differential activity in several regions, including the left IFG and left MOG. Interestingly, previous work

suggests that left IFG is involved in the salience (Uddin et al., 2019) and language (Hertrich et al., 2020) networks, both of which presented decreased power spectra in offenders compared to non-offenders in Studies 1 and 2. Moreover, task-based disruptions in the left IFG have previously been reported in offenders (Schiffer et al., 2014, 2017; Vollm et al., 2010). The IFG is part of the mirror neuron system and is involved in observing and executing action (Hamzei et al., 2016; Kilner et al., 2009). For instance, an rTMS study demonstrated that disrupting activity in the left IFG leads to disruptions in the perception of social stimuli and associated task responses (Keuken et al., 2011). If the left IFG activity associated with offending does relate to deficits in the observation and execution of action, then it may explain how offenders could react inappropriately in certain contexts (e.g., commit illegal acts).

Offenders also showed increased activity in the left middle occipital gyrus (MOG) in comparison to non-offenders. Specifically, the Offender > Non-Offender contrast showed aberrant activity in the left MOG. Activity in this region has been associated with disruptions in TOM (Schiffer et al., 2017) and threat perception (Lee et al., 2009) in offenders. More generally, the MOG is part of the dorsal visual stream and has been associated with empathic face processing (Del Casale et al., 2017) and unconscious face/tool processing (Tu et al., 2013) in the general population. This result of increased activity in the left MOG in offenders compared to non-offenders, combined with the results from Study 1 (i.e., decreased LFPR in the visual network) and Study 2 (i.e., a negative association between the number of crimes committed and power spectra activity in the visual network), suggest that offenders may indeed show disruptions in low-level processing compared to non-offenders. While this has not been commonly reported in the

literature, as discussed in Study 2's discussion, some reasons support such speculation. Furthermore, the consistency across Studies 1, 2 and 3 suggests the value of additional consideration. One possibility is that offenders may present reduced visual acuity compared to non-offenders. Indeed, juvenile offenders have been reported to present a higher prevalence of disruptions in visual acuity and are less likely to wear corrective lenses when needed than non-offenders (Harrie & Harrie, 2016). Another possibility is that offenders present a reduced ability to send *peripheral information* (outside one's goal-directed focus) to cognitive areas of the brain. For instance, it has been demonstrated that offenders present disruptions in their attention processes (Baskin-Sommers et al., 2011; Newman et al., 2010). This disruption in attention has been theorized to limit offenders' ability to process information peripheral to their focus of attention and, in turn, to modulate their ability to experience fear in fear-inducing situations (Baskin-Sommers et al., 2011; Newman et al., 2010). Nevertheless, another possibility is that the increased MOG activity could be a compensatory mechanism for aberrant visual processing, by which offenders over-engage activity in the left MOG during visual perception. As abnormal activity in this region is also associated with disruptions in TOM (Schiffer et al., 2017) and threat perception in offenders (Lee et al., 2009), it could be that disruptions in left MOG activity in offenders could have a negative impact on their social interactions. Indeed, such disruptions in visual processing could have important implications for offenders, as they might misperceive the visual components of social interactions, potentially engaging a disproportionate antisocial behaviour in response. For example, offenders tend to show reduced ability to identify emotions when viewing faces, and this visual processing aberrance is accompanied by disrupted neural activity in

occipital regions, among other neural disruptions (Contreras-Rodriguez et al., 2014; Decety et al., 2014).

I did not identify any clusters of decreased activity in offenders compared to non-offenders. This null result could be attributable to the heterogeneity of the control and offender groups combined in this analysis. Consequently, future meta-analytical work should explore the specific contexts in which neural activity is decreased between offenders and non-offenders or among which sub-groups of offenders this decrease might happen.

Previous meta-analytic work found that heightened antisocial traits were primarily associated with activity in the prefrontal (Deming & Koenigs, 2020; Nickerson, 2014; Poeppl et al., 2019; Yang & Raine, 2009) and amygdalar regions (Deming & Koenigs, 2020; Dugré et al., 2020; Poeppl et al., 2019), as well as parietal and temporal regions (Deming & Koenigs, 2020). Thus, the present results are at some odds with these previous meta-analyses. This difference in results could possibly be explained by the differing sample selection criteria used in this study compared to those used in the other meta-analyses. Indeed, while previous meta-analyses have included articles in which participants were recruited based on their antisocial *traits*, I selected articles in which samples were recruited based on their antisocial *behaviour*, as rigorously defined by offender status. It is important to highlight that despite my focus on including samples characteristic of antisocial behaviour, 61% of the offenders included in my meta-analysis presented a diagnosis of either psychopathy or ASPD. Thus, the majority of this sample presented heightened antisocial traits and, thus, my results could potentially also be representative of antisocial traits. However, while my results are representative of

heightened antisocial *traits* to a certain extent, they provide a more homogeneous representation of the neural activity associated with antisocial *behaviour* than previous meta-analyses, indicating that *antisocial traits* and *antisocial behaviour* seem to present both an overlapping and distinct neural signature.

Alternatively, these divergent results could be attributable to other methodological differences between the present meta-analysis and previous work. For instance, some meta-analyses included both juvenile and adult participants (Dugré et al., 2020; Nickerson, 2014; Yang & Raine, 2009), while I focused only on adults. Also, the previous meta-analyses, except for Poeppl et al. (2019), used algorithms other than ALE to perform their analyses. Given these various methodological differences, conclusions regarding the comparison between my results and those obtained by these meta-analyses should be interpreted with caution. Nonetheless, my results suggest the value of further investigating differences between neural mechanisms associated with *antisocial behaviour* and *antisocial traits* in future studies.

4.4.1. Neural disruptions associated with cognitive and emotional-processing domains

IFG-related abnormalities were specifically identified within studies that targeted the cognitive processing domain. Given previous evidence of left IFG participation within the mirror neuron system (Hamzei et al., 2016; Kilner et al., 2009), offenders' aberrant left IFG activity could relate to disruptions in cognitive processing during the observation of and/or response to stimuli. This finding is in line with previous research, which has shown that offenders are indeed characterized by cognitive processing deficits that can interfere with their social skills (Lahat et al., 2015; Meijers et al., 2015; Serin & Kuriychuk, 1994). For instance, offenders have shown reduced performance, and less

reliance on executive systems, during performance of a moral-conventional judgement task and show less reliance on executive functioning when performing the task, compared to non-offenders (Lahat et al., 2015).

Of potential import, some overlap exists between the present methodology and the methodology employed by Dugré et al. (2020). Notably, some samples that I included in the cognitive domain analysis overlapped with the task domains used by Dugré et al. (2020), more precisely with cognitive control (Joyal et al., 2007; Kargel et al., 2017; Schiffer et al., 2014, 2017; Vollm et al., 2010), punishment/reward processing (Gregory et al., 2015; Prehn, Schulze, et al., 2013), risk-taking (Prehn, Schlagenhauf, et al., 2013) and social cognition (Massau et al., 2017; Mier et al., 2014; Pujol et al., 2012; Schiffer et al., 2017). Similarly, some overlap was observed between the samples included in the emotional domain analysis and the task domains used by Dugré et al. (2020), notably with acute threat response (Contreras-Rodriguez et al., 2014; Lee et al., 2009; Prehn, Schulze, et al., 2013) and social cognition (Meffert et al., 2013). However, despite these similarities, I did not observe the relationship between amygdala activity and threat detection/social cognition reported by Dugré et al. (2020). This difference in outcome could be attributable to some fundamental differences between the two studies. Indeed, Dugré et al.'s (2020) meta-analyses focused primarily on youth, with 86% of samples being under 18 years of age, while the present study focused exclusively on adult samples. Thus, it could be that amygdala abnormalities are more characteristic of children and adolescents, or may rectify by adulthood. Moreover, Dugré et al. (2020) aimed to assess the influence of *antisocial traits* on neural activity, with 63% of the samples included in their study having not committed a criminal act. In contrast, the present study

focused exclusively on individuals who *had* committed an antisocial act. Thus, an important distinction between *antisocial traits* and *antisocial behaviour* may be being teased apart between the results of the two meta-analyses. Thus, while some intersect is present, both studies provide valuable but distinct contributions to our understanding of neural activity in antisocial populations.

4.4.2. Violent crime-related activity

Violent criminal behaviour was associated with clusters of activity in the left MOG and the left posterior cingulate cortex (PCC). While previous meta-analytic work has not tended to identify an association between violent behaviour and neural activity in occipital regions (Raschle et al., 2015), a reasonable body of work has, in fact, noted this relationship (Calzada-Reyes et al., 2013; Drexler et al., 2000; Raine et al., 1998; Tiihonen et al., 2008). For instance, bilateral occipital lobes in violent offenders present increased beta activity (Calzada-Reyes et al., 2013), increased glucose metabolism (Raine et al., 1998) and increased white matter volume (Tiihonen et al., 2008). Moreover, during a competitive reaction time task, non-offenders who responded aggressively to their opponent by administering an aversive stimulus in retaliation showed increased activity in bilateral occipital lobes (Lotze et al., 2007). Thus, my result of aberrant activity in the left MOG in violent offenders adds to a growing literature that suggests that aberrant occipital activity could be an important marker of violent behaviour in offenders. Future research could investigate if decreasing activity in the left MOG could relate to a decrease in violent behaviour.

The second neural cluster where activity was associated with violent offending was in the left posterior cingulate cortex (PCC). Within the general population, the PCC,

a key node of the default-mode network (Gusnard & Raichle, 2001), is thought to be involved in the retrieval of autobiographical memories (Maddock et al., 2001; Summerfield et al., 2009), the planning of future actions (Pearson et al., 2011), the focusing of attention (Gusnard & Raichle, 2001; Hahn et al., 2007; Leech & Sharp, 2014) and, more globally, self-referencing processing (Brewer et al., 2013; Whitfield-Gabrieli et al., 2011). Within violent offenders, PCC activity has been associated with disruptions in response inhibition (Kargel et al., 2017) and threat perception (Lee et al., 2009). Moreover, perpetrators of domestic violence present aberrant neural activity in the PCC (Bueso-Izquierdo et al., 2016; Romero-Martínez & Moya-Albiol, 2013), and violent offenders present with decreased grey matter volumes in the left PCC compared to non-offenders (Tiihonen et al., 2008). Interestingly, Deming & Koenigs, in their (2020) meta-analysis, reported that heightened activity in the posterior cingulate cortex was associated with heightened psychopathic traits. Thus, activity in the left PCC could relate to violent behaviour in offenders; however, as activity in the PCC is associated with various functional processes, the exact nature of the processing disruptions associated with aberrant activity in the left PCC in violent offenders should be explored in future studies.

Analyses conducted separately for non-sexually violent crimes and sexually violent crimes did not yield significant results. This null result suggests that neural mechanisms underlying violent offending do not relate specifically to the sexual nature of the crimes.

4.4.3. Limitations

For this meta-analysis, I purposely focused on neural differences between offenders and non-offenders in order to gain an understanding of neural features specific to

offenders. However, this specific focus limited the number of samples that could be included in the meta-analysis, as more studies have performed within-group investigations of neural activity in offenders. This limitation could be circumvented in future meta-analytic work by including within-group analyses of offender samples.

Another potential limitation of this study is that I included exclusively articles that investigated task-based neural activity. While the literature on task-based neural activity in offenders is limited, a rich literature has investigated other functional and anatomical neural metrics associated with offending, such as FNC, resting-state activity, cortical thickness, and white-matter integrity. Thus, future work of this nature could benefit from also including neuro-anatomical studies and studies investigating other metrics of neural activity in offenders to gain an even broader understanding of the neural differences associated with offending behaviour and further consolidate our functional and anatomical knowledge of neural inefficiencies in this population.

Some of the analyses performed in this study were well under the number of samples recommended to achieve good power when using ALE methods. Indeed, current ALE standards recommend using between 17 and 20 samples to achieve well-powered results (Eickhoff et al., 2016), and in some analyses (i.e., those investigating emotional processing contexts), the current meta-analysis incorporated as few as three observations. With this in mind, analyses pertaining to mixed offences (three samples), emotion processing and pedophilic offenders (five samples each) were underpowered, which could potentially explain their null results. Indeed, during ALE analysis, each focus is attributed an ALE score representing the union of the probabilities from each experiment for which this focus was reported. This ALE score increases when more experiments are

added to the model, with this increase being linear for a specific focus when the experiments added also report it (Eickhoff et al., 2016). Thus, when too few experiments are included in an ALE analysis, the ALE value might not be high enough to reach statistical significance (i.e., to present an ALE value significantly higher than other foci in the brain). To a lesser extent, analyses comparing offenders to non-offenders (16 samples), evaluating the cognitive processing domain (15 samples) and evaluating non-sexual violent offending (14 samples) were also somewhat underpowered. However, they contained enough samples that it is unlikely that the results of these analyses were driven by one specific sample (Eickhoff et al., 2016). Consequently, these analyses could be reproduced in future meta-analytic work when more fMRI studies investigating neural integrity in offenders will be available. Alternatively, given the current limited availability of such research, future meta-analytic work could use image-based meta-analytic methods, which are less sensitive to the number of samples included and less prone to data loss than coordinate-based methods (Samartsidis et al., 2017). Indeed, image-based meta-analytic methods involve the statistical combination of fMRI statistical maps rather than the combination of foci. Thus, they depend on the willingness of researchers to share their data. However, although data sharing is becoming more common, this practice is still not frequent enough to allow large-scale image-based meta-analyses yet (Samartsidis et al., 2017).

Chapter 5. General discussion

This dissertation comprised three studies, each of which aimed to further knowledge regarding potential neural markers of offending. Studies 1 and 2 both focused on resting-state metrics to evaluate whether offenders showed differential resting-state characteristics than non-offenders. Results of both studies were consistent in indicating that offenders were characterized by decreased spectral power in the majority of evaluated RSNs compared to non-offenders. Importantly, these disruptions in spectral power occurred as a result of both decreased low-frequency activity and increased high-frequency activity compared to non-offenders. This result is important as low-frequency disruptions are thought to indicate decreased neural efficiency (Fransson, 2005; Greicius et al., 2003), while high-frequency disruptions have been related to noisier baseline neural activity (Biswal et al., 1995). Thus, as offenders showed disruptions in both low- and high-frequency activity compared to non-offenders, these results suggest that resting-state activity disruptions in offenders could represent decreased neural efficiency, 'noisier' activity or a combination of both. Future research should investigate these possibilities.

Study 3 took a complementary approach by undertaking a meta-analysis to summarize results regarding differential task-based neural activity between offenders and non-offenders. This meta-analysis indicated that there were indeed regions that showed differential neural activity in offenders; moreover, some of these regions appeared to be particularly prominent in those with violent offence histories (i.e., left MOG and left PCC). These results, combined with the results from Study 1 (i.e., decreased LFPR was associated with increased cocaine use in cocaine-dependent offenders) and Study 2 (i.e.,

decreased LFPR was associated with increased criminal convictions across offenders), suggest that both resting- and task-based abnormalities related to specific criminogenic features of the offender.

One specific goal of this dissertation was to evaluate whether disruptions would be seen across various neuroimaging modalities and functional domains, or whether they would be more prominent in certain modalities/domains than others. To this end, I found evidence for both patterns in different neural regions. As an example of a pattern of disruption that spanned across neuroimaging modalities, dysfunctions within, or in RSNs encompassing, the left IFG and left MOG were observed in all three studies (and thus both resting-state and task-based metrics). Indeed, the left IFG is involved in the salience (Uddin et al., 2019) and language (Hertrich et al., 2020) networks, while the left MOG is involved in the visual network (Yang et al., 2015) and offenders presented power spectra disruptions in these RSNs in Studies 1 and 2 in comparison to non-offenders. Interestingly, offenders also show anatomical disruptions in these brain regions (e.g., increased white matter volume in occipital regions and decreased grey matter volume in the left IFG; Tiihonen et al., 2008). While there may not be a one-to-one relationship between the number of modalities that abnormalities are seen in and the severity of those abnormalities, the pervasiveness of these abnormalities nonetheless suggests that they underlie a particularly stable difference in neural integrity between offenders and non-offenders. On the other hand, studies 1 and 2 indicated that rest-related neural activity disruptions in offenders were specific to certain RSNs. Indeed, offenders presented power spectra disruptions in five of the eight investigated RSNs in Study 1, and six of the eight investigated RSNs in Study 2, compared to non-offenders. Thus, offenders presented

broad but specific RSN disruptions, which encompassed a larger area of the brain than those observed in their task-based activity.

Study 3 found that aberrant activity in the left IFG was most apparent when offenders were asked to perform tasks requiring cognitive processes. This coincides with the results of Studies 1 and 2, where ECN and salience networks - which are heavily involved in cognitive processing (Menon, 2010) - were disrupted in offenders. The ECN is involved in highlighting task-relevant information, inhibiting information that is not relevant to the task and using relevant information to produce an appropriate behavioural response (Gratton et al., 2017), while the salience network is involved in the detection of behaviorally relevant information and the coordination of neural resources (Uddin, 2015). The neural mechanisms underlying these cognitive dysfunctions, and the nature of the relationship between these dysfunctions and offending behaviour, should be further investigated.

Studies 1 and 2 also identified neural disruptions within RSNs associated with other processing domains. Specifically, offenders showed neural oscillatory disruptions in the DMN, language and visual networks. While some elements of the DMN, language and visual networks can be involved in cognitive processes (Fedorenko & Thompson-Schill, 2014; Smallwood et al., 2021; Yang et al., 2015), these networks mainly sustain other processing domains. The DMN is involved in emotional processing, self-referential mental activity and memory recollection (Raichle, 2015) and tends to desynchronize when we are focused on a task (Fox et al., 2015; Raichle, 2015). The language network sustains the ability to perceive, produce and understand language (Fedorenko & Thompson-Schill, 2014), while the visual network is mainly involved in the perception

and recognition of visual information and visual imagery (Wang et al., 2008). The previous literature has often characterized offenders as presenting with both cognitive and emotional processing deficits, with little consensus regarding which, if any, might be predominant in antisocial individuals. The results of my dissertation suggest that cognitive deficits may, in fact, be more characteristic of offender populations than emotional deficits. The nature of the relationship between these functional deficits and offending behaviour should be explored in future work.

An additional goal of the dissertation was to evaluate whether offenders' neural disruptions related more closely to trait-based (i.e., psychopathy traits, drug dependence status) or lifestyle (i.e., criminal history, drug use) factors of individual risk-factors of offending. To this end, Study 1 evaluated the influence of cocaine dependence, cocaine use and psychopathic traits on resting-state, Study 2 evaluated the influence of different aspects of offenders' criminal histories (e.g., presence of a violent criminal history, number of convictions) on resting-state, and Study 3 considered violence history on task-related activity at a meta-analytic level. Across studies, I found some evidence for an influence of lifestyle, but not of trait-based, factors. Specifically, in Studies 1 and 2, cocaine use and the number of criminal convictions predicted LFPR disruptions, but psychopathy and cocaine-dependence status did not; in Study 3, left MOG and left PCC activity was associated with a violent criminal history. These results indicate that neural disruptions are not uniform across offenders and that the neural disruptions identified in certain sub-groups of offenders varied as a function of specific lifestyle features. The underlying neural mechanisms associated with cocaine use and criminal proficiency in offenders should be further investigated.

Given the strong relationship between major drug use and crime (DeLisi et al., 2015; Innes, 1988; Makkai & Payne, 2003), it is unclear whether drug use is the cause or the effect of the participants' criminal behaviour. Similarly, it is difficult to know if heightened drug use is the cause or the effect of the neural disruptions observed. Indeed, my results present a chicken and egg situation, one that has been very difficult for research in this area to crack. It could be that individuals characterized with decreased rest-related integrity may be predisposed towards higher cocaine use and/or criminal tendencies. Alternatively, it could be that this pattern of disrupted resting-state activity is a consequence of the compounding effects of these two factors over the years. The results of this dissertation cannot provide additional clarity to this quandary, but do further highlight its importance. Indeed, while increased cocaine use and criminal proficiency were independently related to decreased global LFPR in offenders, they were also moderately correlated. Thus, it may be that individuals who are convicted of more crimes tend to use more drugs, or that individuals who use more drugs tend to be convicted of more crimes. Similarly, it could be that increased drug use and/or criminal behaviour may negatively affect LFPR integrity, or that individuals who present decreased LFPR tend to be convicted of more crimes and/or use more drugs. It is important to recognize this limitation in interpreting my results because much work remains to be done to understand the complex relationship between drug use, criminal behaviour and aberrant neural activity. For instance, future work should compare LFPR integrity in drug users who have been convicted of crimes and those who have not. Nevertheless, my results do highlight the complicated nature of these effects and the importance that future studies aim to disentangle these potentially complicated relationships.

Finally, I observed inconsistent results regarding the relationship between the existence of a violent criminal history and neural activity in offenders. Resting-state power spectra activity did not differ as a function of violent offending in Study 2. Conversely, the meta-analytical results from Study 3 indicated that offenders with a violent criminal past presented aberrant neural activity in the left MOG and the left PCC. Thus, a pattern seems to emerge, such that task-based neural disruptions, but not disrupted baseline neural activity, are related to a history of violent crimes. While this dichotomy could reflect differences between the samples (e.g., types of crimes committed), it suggests that there may be a specific relationship to evaluate between violent offending and offender's neural reactivity in the left MOG and left PCC in some, not yet established, contexts. However, this possibility should be formally investigated in future work.

5.1. Nature vs. Nurture

The distinction between the contributions of trait-based and lifestyle factors of offending may make one question whether the identified neural disruptions are mostly related to influences of the environment rather than biologically-mediated. Indeed, genetics explains a large proportion of variance in psychopathic traits (estimated between 63% (Larsson et al., 2006) and 69% (Tuvblad et al., 2014)). On the other hand, the genetic influence of antisocial behaviour is more moderate (meta-estimated at 50%, with higher heritability levels associated with the severity of the behaviour; Mason & Frick, 1994). Conversely, the heritability of illicit substance use and abuse is relatively low (estimated at ~25%; McGue et al., 2000). However, a review of twin studies on the heritability of substance use has shown that while substance use has a low to moderate

heritability, this heritability rate significantly increases when genetic predispositions are paired with social influences and antisocial behaviour (Hopfer et al., 2003). Indeed, antisocial behaviour and substance use share common genetic influences, and it is theorized that in those with this genetic background, antisocial behaviour tends to present itself first and act as a precursor to substance use (Hopfer et al., 2003). Psychopathic traits, which are highly heritable, did not relate to baseline neural disruptions in my dissertation, while criminal behaviour and cocaine use, which have a low to moderate heritability, negatively influenced the integrity of power spectra activity in offenders. Other environmental factors (e.g., childhood trauma, incarceration) not tested within this dissertation have also been demonstrated to relate to neurocognitive disruptions in offenders. Indeed, maltreatment during childhood is associated with various maladaptive outcomes, such as increased psychopathic traits and antisocial behaviour (Moreira et al., 2021). Each instance of trauma that a child experiences increases their chances of becoming a juvenile offender by 35 (Fox et al., 2015). Moreover, the carceral environment itself has been shown to have a negative impact on cognitive functioning in offenders (Meijers et al., 2015, 2018; Umbach et al., 2018). For instance, the general adult incarcerated population tends to present disruptions in attention and set-shifting processes (Meijers et al., 2015), and after three months of incarceration can also present an increase in risk-taking and a decline in attention (Meijers et al., 2018). Similarly, youth offenders have been shown to experience a decline in cognitive control and emotion recognition after four months of incarceration (Umbach et al., 2018). This could suggest a delicate balance between the influence of genetics (i.e., nature) and the environment (i.e., nurture) on baseline neural disruptions in offenders. Indeed, given that

antisocial behaviour is moderately influenced by genetics and that the lifestyle associated with this antisocial behaviour tends to lead to substance use, it could be that baseline neural disruptions in offenders are related to both the innate and lifestyle influences of antisocial behaviour and further nurtured by substance use, and possibly environmental factors such as trauma and incarceration. However, the genetic and environmental influences of the neural disruptions that I observed in offenders should be further studied in future work.

Some variance remains unexplained, and the neural disruptions that I observed could also be explained by other, yet untested, characteristics of offenders. Some of these could be environmental in nature. For instance, the presence of childhood trauma, combined with decreased activity in the right hemisphere, has been shown to predispose towards violent behaviour in adulthood (Raine et al., 2001). Moreover, the cognitive decline associated with incarceration (Meijers et al., 2015, 2018; Umbach et al., 2018) could be accompanied by increased disruptions in neural activity. However, the influence of incarceration on neural activity needs to be further investigated. Other environmental factors such as parental style and *socio-economic status* (SES) also have an influence on offenders. Indeed, offenders who have experienced more controlling parenting styles tend to present reduced empathic concern and increased personal distress, while a less caring parenting style is associated with decreased empathic concern, perspective taking and higher personal distress in offenders (Wang et al., 2021). While SES has shown a weak relationship with delinquent behaviour in juvenile offenders, the economic problems caused by a decreased SES are associated with increased delinquent behaviour (Agnew et al., 2008). Others may be more genetic in nature. For instance, genetic variations have

been associated with antisocial behaviour and could potentially relate to neural disruptions. For instance, DRD2 and DRD4 genetic polymorphisms have been associated with increased contacts with the justice system in youths from low-risk family environments (DeLisi et al., 2008), while polymorphisms of 5HTTPLR (a serotonin transporter gene) and MAOA-uVNTR (the monoamine oxidase A gene) have both often been associated with antisocial behaviour (Ficks & Waldman, 2014). Additionally, the Big5 personality traits are known to differ in offenders, who tend to present higher neuroticism levels than non-offenders (Becerra-García et al., 2013). Thus, other environmental (e.g., trauma, incarceration, SES) and individual (e.g., genetics and Big5 personality traits) risk factors could potentially relate to aberrant neural activity in offenders. However, future work should investigate how these characteristics of offenders interact with neural integrity in offenders.

5.2. Implications for treatment

The neural markers that I identified in offenders could potentially serve as treatment targets. Indeed, previous work has demonstrated the efficacy of neural activity modulation in offenders to reduce behaviours associated with offending (Konicar et al., 2015, 2021; Molero-Chamizo et al., 2019). For instance, Konicar et al. (2015) subjected violent offenders with heightened psychopathic traits to neurofeedback training during which they had to consciously increase and decrease their *electroencephalogram* (EEG) measured slow-frequency cortical activity in the fronto-central area of their brain. After this training, offenders were able to self-modulate this neural activity which led to changes in behaviours associated with their violent criminal history, such as reduced aggression, impulsivity and behavioural approach (BAS) and an increase in behavioural-

inhibition skills and increased cortical sensitivity for error-processing (Konicar et al., 2015). Importantly, this training-induced brain plasticity in offenders led to long-term increased alpha and decreased theta and delta activity (indicating changes in central-nervous system-related activity; Konicar et al., 2021). Another study subjected homicidal and non-homicidal violent offenders to a *transcranial direct current stimulation* (tDCS) protocol to increase cortical excitability in the frontal cortex, which resulted in decreased self-reported aggression across offender groups (Molero-Chamizo et al., 2019). Thus, these studies demonstrate that altering rest-related neural activity in offenders can reduce behaviours associated with offending. However, the specific use of fMRI-based activity in the left IFG and MOG for this purpose has not yet been tested and should be explored in future work to assess its potential as a treatment tool.

Additionally, my results indicated that deficits in cognitive processing could be important to offending. Thus, this suggests that improving cognitive processes in offenders with cognitive deficits could potentially decrease offending behaviour. Previous work has investigated the impact of improving cognitive abilities in offenders with promising success. For instance, *cognitive-behavioural therapy* (CBT) programs are almost consistently linked to reductions in recidivism, especially when they include elements of anger control and interpersonal problem solving (Landenberger & Lipsey, 2005). Moreover, a treatment targeting attention to context during which offenders had to train this skill for six weeks resulted in offenders significantly improving their performance in tasks involving attention processes (Baskin-Sommers et al., 2015). Although this study did not assess if the improvement in attention was associated with a decrease in antisocial behaviour, it does demonstrate that cognitive skills can be

improved in this population with targeted training. However, the relationship between improving cognitive skills and antisocial behaviour should be investigated in future studies to assess this possibility formally.

5.3. Limitations

The research conducted within is not without its limitations. While some of these limitations are relatively common in the fMRI and psychology fields, some limitations might point to caution in the interpretability of specific results reported herein.

First, this dissertation was performed solely using male participants. However, previous research has demonstrated that male and female offenders differ in terms of psychopathic traits, criminal behaviour, and drug use. For instance, work has demonstrated that, compared to male offenders, female offenders present fewer psychopathic traits, as expressed by lower PCL-R (de Vogel & Lancel, 2016) and PCL *Screening Version* (PCL-SV; Strand & Belfrage, 2005) scores. Moreover, female offenders with increased psychopathic traits present different criminal behaviour compared to psychopathic male offenders. Indeed, psychopathic female offenders are more likely to commit fraud, exhibit criminal behaviour motivated by relational frustration, a diagnosis of BPD and use less physical violence in their criminal activity (de Vogel & Lancel, 2016). More generally, women are less likely to commit crimes, with female prisoners representing only 8% of the total prison population in the U.S. in 2019 and being less likely to commit violent crimes (38% of female offenders were incarcerated for a violent crime in 2018 vs 58% of incarcerated men; Carson et al., 2020). Other work has focused on gender differences in substance use in offenders and observed that, compared to male offenders, female offenders are less likely to present a substance

use disorder and less likely to use drugs when committing a crime (de Vogel et al., 2021). Thus, given the gender differences described above, my results might not generalize to female offenders. Consequently, these analyses should be replicated within female offenders or a mixed sample of males and females to explore the extent to which results generalize across genders.

A second limitation may pertain to the recruitment of participants in Studies 1 and 2, which were recruited within the city of Albuquerque, New Mexico. Indeed, New Mexico presents a different criminal profile than other states of the U.S. For instance, in 2017-2018, New Mexico had the second-highest rate of violent crimes in the U.S., with 856.6 violent criminal incidents per 100 000 people, and the second-highest poverty rate at 19.5% (Federal Bureau of Investigation, 2019). Moreover, New Mexico is among the top 1% U.S. states with the highest mortality rate associated with drug use disorders (Dwyer-Lindgren et al., 2018). Thus, results from this sample might not generalize to offenders from other regions of the United States and in other countries. Consequently, future studies may want to replicate the analyses performed in Studies 1 and 2 using offender samples recruited from other locations.

An additional limitation is the sample size used in Studies 1 and 2. Indeed, I used the same sample for both studies, including 102 participants in Study 1 and 97 in Study 2, which corresponded to a post-hoc statistical power of 35% (Study 1) and 38% (Study2) for the omnibus ANOVA models. Thus, the results of studies 1 and 2 present moderate power and may not generalize to the entire offender population. However, it is important to note that sample sizes are generally smaller in fMRI studies, given the high cost of data acquisition (Durnez et al., 2016). To attempt the compensate for smaller sample sizes,

stringent thresholding techniques have been developed in the past decade to increase the statistical power and cost-efficiency of fMRI analysis (Durnez et al., 2016). Notably, a recent study has suggested that including 80 participants or more provides reliable and stable estimates when investigating the relationship between neural activity and behavioural measures (Grady et al., 2021). Thus, while my sample may seem low by some standards, it falls within fMRI guidelines and should provide a reliable estimate of the relationship between rest-related power spectra and the trait-based and lifestyle factors of offending. Nevertheless, my analyses should be replicated with a bigger sample to assess their generalizability.

Another limitation may pertain to the fact that I investigated MRI modalities separately in my dissertation. Indeed, Studies 1 and 2 were performed using resting-state fMRI imaging, while Study 3 was performed using coordinates of task-based neural activity. While my results indicated that some aberrant neural activity was present in the same neural regions within both imaging modalities, I did not use conjunction analyses to statistically test if these neural markers significantly overlapped across these modalities. Thus, although my results suggest that some neural markers transcend across rest and task-related activity, this possibility should be interpreted with caution. With this in mind, future studies should formally investigate if these neural markers of offending statistically overlap across brain imaging modalities.

Of potential import, all studies included in this dissertation were secondary analyses of existing data. Thus, although the choice of variables used was guided by past literature, it was also limited by the scope of data acquisitions initially performed and the comprehensiveness of this data. Nonetheless, I attempted to remedy this limitation by

conducting background checks to obtain the criminal history of the participants included in Studies 1 and 2, and I was able to contact the authors of the articles included in Study 3 to inquire about additional measures (albeit with limited success). However, there remained some limitations to the array of variables that could be investigated.

5.4. Conclusions

In this dissertation, I assessed the integrity of rest-related neural oscillations and task-related activity in offenders and explored how specific lifestyle and trait-based risk factors of offending interact with this neural activity. This work furthered understanding of the neural underpinnings of offenders and the factors that contribute to them. This work uncovered several neural regions of interest in two different modalities of neural activity for the study of offenders and provided potential treatment and prevention targets across offenders and within violent offenders.

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Appendices

Appendix A. Supplementary materials Study 1

Section.1.

Spectral analyses controlled for age and intelligence quotient

An omnibus ANOVA was run to evaluate the relationships between frequency bins, RSNs and groups. As Offender and Non-Offender participants differed significantly on age and IQ, these were added as covariates in the model in the following supplementary analyses. When controlling for age and IQ, a main effect of Group, F (2,97) = 7.43, p = 0.001, was identified with pairwise comparisons indicating that Dependent (M = 0.87; SD = 0.05) and Non-Dependent (M = 0.86; SD = 0.05) groups showed similar overall power spectra, p = 1.00, and that both offender groups presented with increased power spectra in comparison to Non-Offenders (M = 0.84; SD = 0.07; both ps < .05). A main effect of Bin, F (1, 97) = 9.77, p < 0.05, was also observed, with the low frequency bin showing the higher (M = 1.15; SD= 0.08), and the high-frequency the lower (M = 0.56; SD = 0.10) overall power. These main effects were influenced by a significant Group x Bin interaction, F (2, 97) = 3.65, p = 0.030, showing that power spectra differed within frequency bins across the groups, as well as significant interactions between bin and age (F (1, 97) = 13.41, p < 0.001), and between bin and IQ (F (1, 97) = 4.50, p = 0.04). Age was significantly negatively correlated with low-frequency activity (r = -0.44, p < 0.001) and positively correlated with high-frequency activity (r = 0.39, p < 0.001). Conversely, IQ was significantly positively correlated with low-frequency activity (r = 0.28, p = 0.004) and negatively correlated with high-frequency activity (r = -0.28, p = 0.005). However, no significant main effect of Network,

F (7, 97) = 0.98, p = 0.45 was identified. Network x Bin, F (7, 97) = 0.78, p = 0.61, Group x Network, F (14, 97) = 1.53, p = 0.09, Group x Bin x Network, F (14, 97) = 0.65, p = 0.63, Network x Age (F (7, 97) = 1.50, p > 0.05) and Network x IQ (F (7, 97) = 0.61, p > 0.05) interactions did not reach significance.

As no effects or interactions with Network were found, we collapsed mean spectral values across all networks and evaluated for group differences in low-/high-frequency bins via Bonferroni-corrected pairwise comparisons. As can be seen in Figure S1.2 (see also Table S1.1), while the two offender groups did not present significant differences in the frequency bin, several differences were observed with Non-Offenders. Specifically, both offender groups showed significantly increased high-frequency power spectra compared to Non-Offenders (Dependent Offenders: t = 4.24, $p(FWE)$ = .007; Non-dependent Offenders: t = 3.24, $p(FWE)$ = 0.071). These bin-specific differences resulted in Dependent Offenders (M = 5.83, SD = 2.94; t = -2.29, $p(FWE)$ = 0.03) and Non-Dependent Offenders (M = 5.64, SD = 3.13; t = -2.38, $p(FWE)$ = 0.03) presenting with significantly decreased LFPR in comparison to Non-Offenders (M = 10.76, SD = 8.91; see Figure 3), which does not differ from results obtained in our original model.

The inclusion of age and IQ in our models led to changes in our results, whereby global differences in power spectra between offenders and non-offenders were mostly identified within the high-frequency bin (see tables S1.1 and Figure S1.2). However, it is interesting to note that most LFPR results remained when controlling for age and IQ (see Figure 1.5). While the results differ a fair amount when controlling for age and IQ, it is important to recognize that age and IQ are necessarily correlated with drug use; thus, it is difficult to know how to interpret the fact that effects disappear with age and IQ

controlled. Indeed, there is a strong but complicated relationship between drug use and IQ, such that it is not clear if a lower premorbid IQ is prevalent in individuals with a cocaine use disorder or if long-term drug use causes disruptions in cognitive functions (Mahoney, Kalechstein, De Marco, Newton & De La Garza, 2017). Moreover, it could be that the effects of aging are heightened by drug use or, rather, it is the cumulative effects of years of drug use, which increase with age, that impact neural integrity in offenders. We have investigated the interaction effects with age/LCU that further speak to this complexity; they are presented in section 13 of the supplementary materials.

Section 1.1

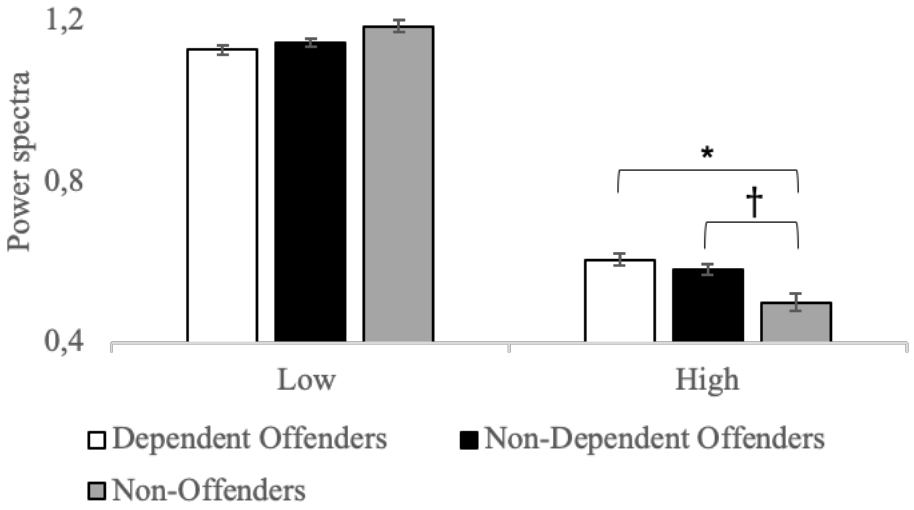
3 (Group) x 2 (Bin) ANOVA controlled for age and intelligence quotient

	F (DF1, DF2)	Partial η^2
Within-Subject Effects:		
Bin	12.72(1, 97) **	0.12
Bin x Group	3.70(2, 97) *	0.07
Bin x Age	14.93(1, 97) **	0.13
Bin x IQ	4.36(1, 97) *	0.04
Between-Subject Effects:		
Group	7.65(2, 97) **	0.14
Pairwise comparisons:		
Low-frequency	2.05	0.04
High-frequency	4.96 *	0.09

*$p < 0.05$ **$p < 0.001$

Section 1.2

Mean power spectra differences between groups during the overall resting-state activity controlled for age and intelligence quotient



Note. Mean power spectra differences in the low- and high-frequency bins between Dependent Offenders (white), Non-Dependent Offenders (black), and Non-Offenders (grey) during overall resting-state activity. Dependent and Non-Dependent Offenders exhibited an overall higher activity amplitude during high-frequency activity in comparison to Non-Offenders. However, no significant differences were observed between the two Offender sub-groups in any frequency bins.

† $p(FWE) < 0.10$ * $p(FWE) < 0.05$, error bars = SEM

Section 1.3

Higher-order effects (F) of the 3 (Group) x 2 (Bin) ANOVAs controlled for age and intelligence quotient for each resting-state network

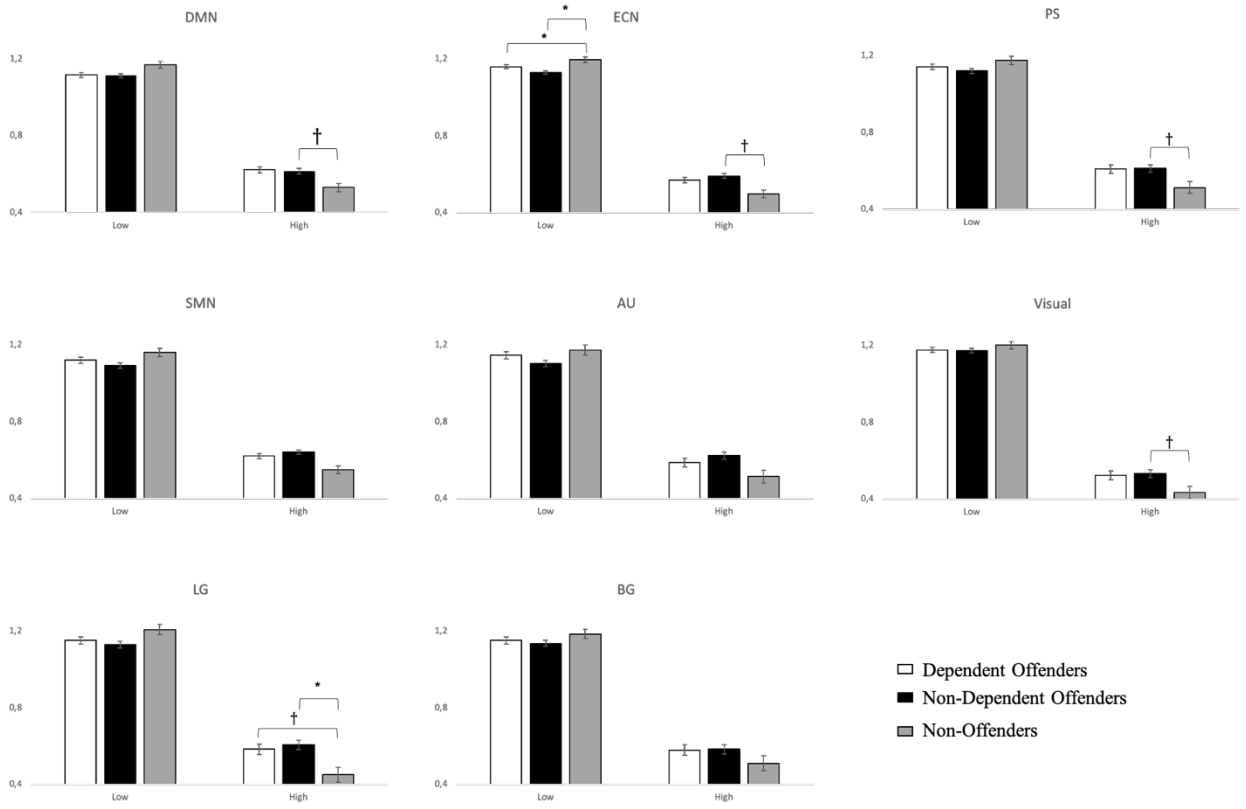
	Within-Subject Effects								Between-Subjects Effects	
	Bin		Bin x Group		Bin x Age		Bin x IQ		Group	
	$F(1, 97)$	partial η^2	$F(2, 97)$	partial η^2	$F(1, 97)$	partial η^2	$F(1, 97)$	partial η^2	$F(2, 97)$	partial η^2
DMN	12.02 **	0.11	2.43	0.05	16.37 **	0.14	2.67	0.03	3.95 *	0.08
ECN	16.31 **	0.14	5.44 *	0.10	13.16 **	0.12	2.68	0.03	3.09 *	0.06
BG	4.17 *	0.04	0.79	0.02	5.48 *	0.05	2.63	0.03	0.76	0.02
SMN	2.66	0.03	2.34	0.05	5.07 *	0.05	3.43	0.03	1.28	0.03
PS	12.08 **	0.11	2.06	0.04	13.29 **	0.12	0.51	0.005	5.43 *	0.10
LG	3.29	0.03	3.53 *	0.07	8.76 *	0.08	3.89	0.04	6.98 **	0.13
Visual	8.37 *	0.08	1.23	0.03	7.92 *	0.08	4.02 *	0.04	7.27 **	0.13
AU	1.99	0.02	2.74	0.05	4.11 *	0.04	3.27	0.03	2.46	0.05

Note. Default-mode network (DMN), Executive control network (ECN), Posterior salience network (PS), Sensorimotor network (SMN), Auditory network (AU), Visual network, Language network (LG) and basal ganglia network (BG).

* $p < 0.05$ ** $p < 0.001$

Section 1.4

Pairwise comparisons in power spectra between groups controlled for age and intelligence quotient in each resting-state network

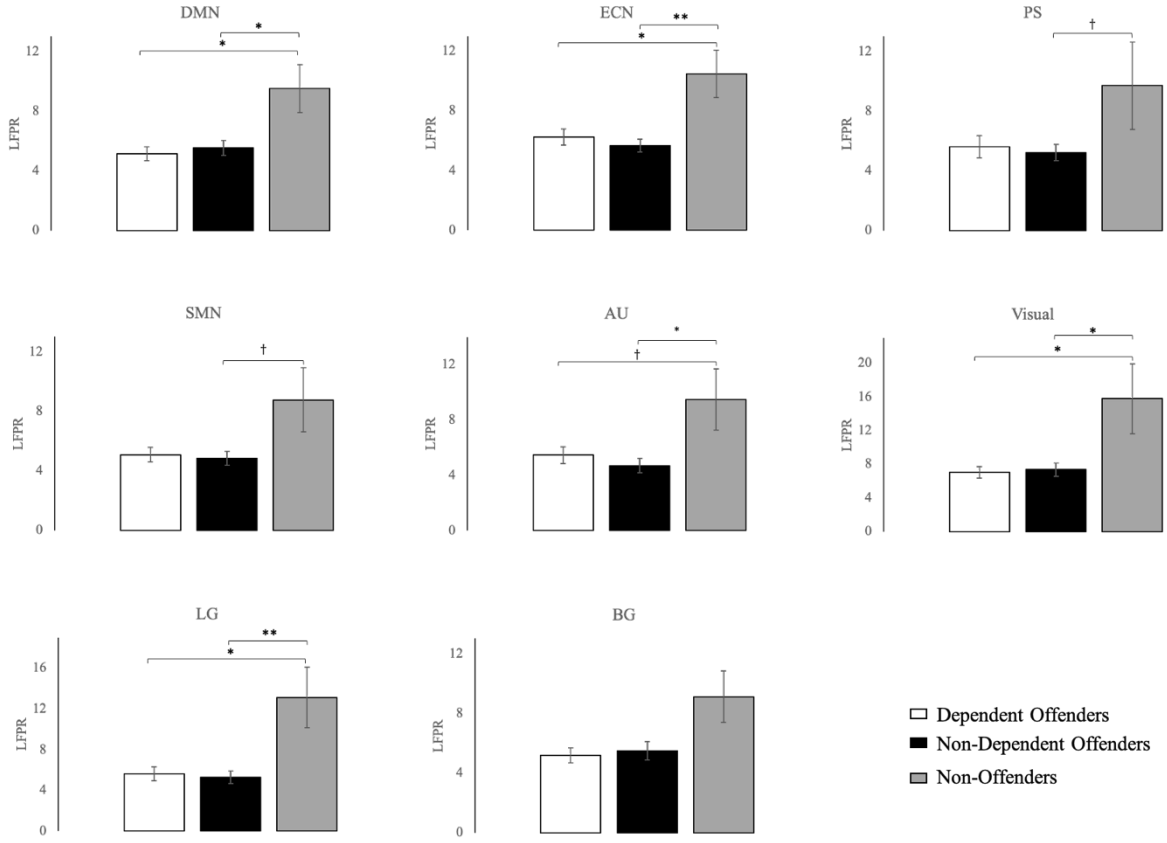


Note. Mean power spectra differences between Dependent Offenders, Non-Dependent Offenders and Non-Offenders in the low- and high-frequency bins in each RSN during rest: Default-mode network (DMN), Executive control network (ECN), Posterior salience network (PS), Sensorimotor network (SMN), Auditory network (AU), Visual network, Language network (LG) and basal ganglia network (BG).

† *p(FWE)* < 0.10 * *p(FWE)* < 0.05, error bars = SEM

Section 1.5

Mean low-frequency power ratio differences between groups controlled for age and intelligence quotient in each resting-state network



Note. Pairwise comparisons of mean differences in low-frequency power ratio (LFPR) between Dependent Offenders, Non-Dependent Offenders and Non-Offenders in each RSN during rest. Default-mode network (DMN), Executive control network (ECN), Posterior salience network (PS), Sensorimotor network (SMN), Auditory network (AU), Visual network, Language network (LG) and basal ganglia network (BG).

† p(FWE) < 0.10 * p(FWE) < 0.05 ** p(FWE) < 0.001, error bars = SEM

Section 2.

Higher-order effects of the 3 (Group) x 3 (Bin) ANOVA

	F(DF1, DF2)	Partial η^2
Within-Subject Effects:		
Bin	1341.47(1, 99) **	0.93
Bin x Group	7.45(2, 99) **	0.13
Between-Subject Effects:		
Group	10.26(2, 99) **	0.17
Pairwise comparisons:		
Low-frequency	5.03 *	0.09
High-frequency	9.02 **	0.15

*$p < 0.05$ **$p < 0.001$

Section 3

Higher-order effects (F) of the 3 (Group) x 2 (Bin) ANOVAs for each resting-state network

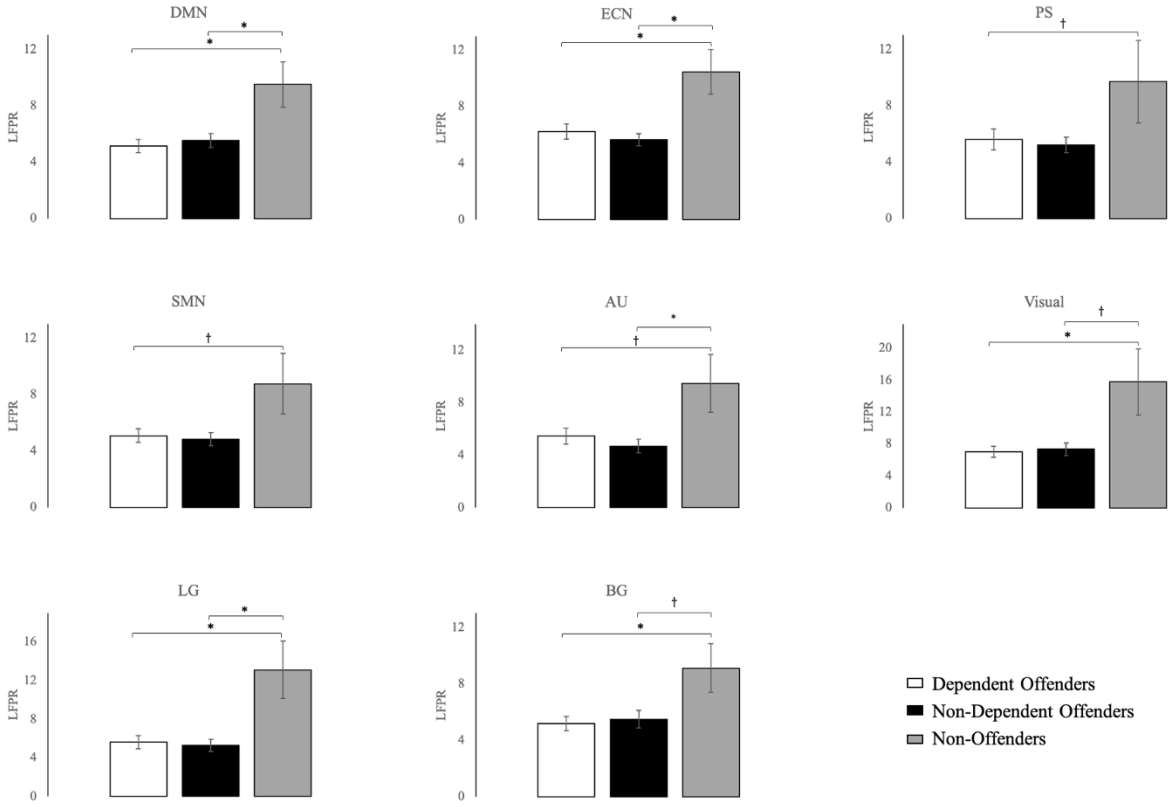
	Within-Subject Effects				Between-Subjects Effects	
	Bin		Bin x Group		Group	
	F (1, 99)	partial η^2	*F* (2, 99)	partial η^2	*F* (2, 99)	partial η^2
DMN	973.70 **	0.91	5.66 *	0.10	6.19 *	0.11
ECN	1457.43 **	0.94	7.58 **	0.13	4.55 *	0.08
BG	627.09 **	0.86	1.97	0.04	1.39	0.03
SMN	546.45 **	0.85	3.84 *	0.07	2.42	0.05
PS	665.41 **	0.87	3.75 *	0.03	6.22 *	0.11
LG	542.08 **	0.85	6.03 *	0.11	7.59 **	0.13
Visual	1157.99 **	0.92	3.08 *	0.06	8.59 **	0.15
AU	476.00 **	0.83	3.83 *	0.07	3.91 *	0.07

Note. Default-mode network (DMN), Executive control network (ECN), Posterior salience network (PS), Sensorimotor network (SMN), Auditory network (AU), Visual network, Language network (LG) and basal ganglia network (BG).

* $p < 0.05$ ** $p < 0.001$

Section 4

Mean low-frequency power ratio differences between groups in each resting-state network



Note. Low-frequency power ratio (LFPR), Default-mode network (DMN), Executive control network (ECN), Posterior salience network (PS), Sensorimotor network (SMN), Auditory network (AU), Visual network, Language network (LG) and basal ganglia network (BG).

† *p(FWE)* < 0.10 * *p(FWE)* < 0.05 ** *p (FWE)* < 0.001, error bars = SEM

Section 5

Hierarchical regression models in all Offenders, Dependent Offenders and Non-Dependent Offenders

	All offenders								
	Model 1			Model 2			Model 3		
	B	SE B	β	B	SE B	β	B	SE B	β
Dependence	1.12	0.83	0.19	0.44	1.19	0.07	3.04	2.16	0.50
Psychopathy	-0.02	0.05	-0.06	-0.04	0.08	-0.09	0.19	0.18	0.47
LCU	-0.14	0.07	-0.26 †	0.22	0.32	0.41	-0.59	0.65	-1.09
Dependence x Psychopathy				0.01	0.12	0.01	-0.22	0.19	-0.32
LCU x Dependence				-0.43	0.32	-0.68	0.39	0.65	0.61
LCU x Psychopathy				0.01	0.01	0.15	0.09	0.05	0.95
Dependence x Psychopathy x LCU							-0.08	0.05	-0.73
R^2		0.05			0.10			0.13	
F for change in R^2		1.43			1.51			2.07	

	Non-Dependents					
	Model 1			Model 2		
	B	SE B	β	B	SE B	β
Psychopathy	-0.08	0.07	-0.19	0.19	0.19	0.46
LCU	0.37	0.30	0.19	-0.59	0.67	-0.31
LCU X Psychopathy				0.09	0.05	0.69
R^2		0.05			0.09	
F for change in R^2		1.07			2.53	

	Dependents					
	Model 1			Model 2		
	B	SE B	β	B	SE B	β
Psychopathy	-0.002	0.07	-0.005	-0.02	0.08	-0.04
LCU	-0.17	0.07	-0.39 *	-0.19	0.08	-0.45 *
LCU X Psychopathy				0.008	0.01	0.12
R^2		0.16			0.17	
F for change in R^2		3.14 *			0.41	

† $p < 0.10$ * $p < 0.05$

Section 6

Multiple multivariate regression of lifetime cocaine use and psychopathy severity on low-frequency power ratio in all eight ICA-derived resting-state networks in Dependents Offenders

	Dependent Offenders		
	Corrected model (F)	LCU (F)	PCL-R (F)
Multivariate Tests:		0.960	0.87
Between subject effects:			
DMN	2.08	3.97 †	0.09
ECN	2.78 †	5.49 *	0.17
BG	0.29	0.19	0.35
SMN	2.73 †	5.10 *	0.55
PS	1.49	2.97 †	0.04
LG	1.77	3.54 †	0.01
Visual	4.08 *	5.99 *	1.73
AU	1.61	3.20 †	0.06

Note. Lifetime cocaine use (LCU), Default-mode network (DMN), Executive control network (ECN), Posterior salience network (PS), Sensorimotor network (SMN), Auditory network (AU), Visual network, Language network (LG) and basal ganglia network (BG).

† $p < 0.10$ *$p < 0.05$

Section 7

Hierarchical regression models with lifetime overall drug use as a regressor

	All offenders								
	Model 1			Model 2			Model 3		
	B	SE B	β	B	SE B	β	B	SE B	β
Dependence	0.67	0.69	0.11	0.83	0.72	0.14	0.76	0.75	0.12
Psychopathy	0.01	0.05	0.03	0.02	0.07	0.05	0.02	0.07	0.05
Overall Drug use	-0.03	0.01	-0.29 *	-0.04	0.02	-0.35 *	-0.04	0.02	-0.34 *
Dependence x Psychopathy				-0.002	0.11	-0.004	-0.003	0.11	-0.004
Overall Drug Use x Dependence				0.00	0.03	-0.002	-0.01	0.03	-0.03
Overall Drug Use x Psychopathy				0.003	0.002	0.18	0.002	0.002	0.16
Dependence x Psychopathy x Overall Drug Use							0.002	0.005	0.05
R^2		0.07			0.09			0.10	
F for change in R^2		2.00			0.83			0.13	

	Non-Dependents					
	Model 1			Model 2		
	B	SE B	β	B	SE B	β
Psychopathy	0.003	0.07	0.01	0.02	0.07	0.05
Overall drug use	-0.03	0.02	-0.29	-0.04	0.02	-0.35 *
Overall drug Use X Psychopathy				0.002	0.002	0.19
R^2		0.08			0.11	
F for change in R^2		1.92			1.63	

	Dependents					
	Model 1			Model 2		
	B	SE B	β	B	SE B	β
Psychopathy	0.02	0.08	0.05	0.02	0.08	0.04
Overall drug use	-0.03	0.02	-0.24	-0.05	0.03	-0.34
Overall drug Use X Psychopathy				0.004	0.004	0.19
R^2		0.05			0.08	
F for change in R^2		0.96			0.91	

* $p < 0.05$

Section 8

Hierarchical regression models with lifetime major drug use as a regressor

	All offenders								
	Model 1			Model 2			Model 3		
	B	SE B	β	B	SE B	β	B	SE B	β
Dependence	0.40	0.72	0.07	0.36	0.75	0.06	0.33	-0.75	0.05
Psychopathy	-0.03	0.05	-0.07	-0.05	0.07	-0.12	-0.06	0.07	-0.15
Major Drug use	-0.03	0.05	-0.06	0.005	0.08	0.01	0.02	0.08	0.04
Dependence x Psychopathy				0.05	0.11	0.07	0.04	0.11	0.06
Major Drug Use x Dependence				-0.04	0.11	-0.07	-0.09	0.13	-0.15
Major Drug Use x Psychopathy				-0.002	0.01	-0.03	-0.01	0.01	-0.11
Dependence x Psychopathy x Major Drug Use							0.01	0.02	0.15
R^2		0.01			0.02			0.02	
F for change in R^2		0.28			0.12			0.55	

	Non-Dependents					
	Model 1			Model 2		
	B	SE B	β	B	SE B	β
Psychopathy	-0.05	0.07	-0.11	-0.06	0.07	-0.14
Major drug use	0.001	0.08	0.001	0.02	0.08	0.04
Major drug Use X Psychopathy				-0.01	0.01	-0.09
R^2		0.01			0.02	
F for change in R^2		0.29			0.36	

	Dependents					
	Model 1			Model 2		
	B	SE B	β	B	SE B	β
Psychopathy	-0.01	0.08	-0.02	-0.02	0.08	-0.04
Major drug use	-0.05	0.07	-0.12	-0.08	0.09	-0.19
Major drug Use X Psychopathy				0.01	0.01	0.12
R^2		0.01			0.02	
F for change in R^2		0.25			0.23	

Section 9

Hierarchical regression models with lifetime minor drug use as a regressor

	All offenders								
	Model 1			Model 2			Model 3		
	B	SE B	β	B	SE B	β	B	SE B	β
Dependence	0.66	0.69	0.11	0.74	0.71	0.12	0.74	0.77	0.12
Psychopathy	0.01	0.05	0.03	0.01	0.07	0.03	0.01	0.07	0.03
Minor Drug use	-0.05	0.02	-0.28 *	-0.06	0.03	-0.32 †	-0.06	0.03	-0.32 †
Dependence x Psychopathy				0.01	0.11	0.02	0.01	0.11	0.02
Minor Drug Use x Dependence				-0.004	0.05	-0.01	-0.003	0.05	-0.01
Minor Drug Use x Psychopathy				0.004	0.003	0.16	0.004	0.003	0.16
Dependence x Psychopathy x Minor Drug Use							0.00	0.007	-0.004
R^2		0.07			0.09			0.09	
F for change in R^2		1.97			0.69			0.001	

	Non-Dependents					
	Model 1			Model 2		
	B	SE B	β	B	SE B	β
Psychopathy	-0.001	0.07	-0.003	0.01	0.07	0.03
Minor drug use	-0.05	0.03	-0.26	-0.06	0.03	-0.30 †
Minor drug Use X Psychopathy				0.004	0.003	0.19
R^2		0.07			0.10	
F for change in R^2		1.65			1.56	

	Dependents					
	Model 1			Model 2		
	B	SE B	β	B	SE B	β
Psychopathy	0.03	0.08	0.07	0.03	0.08	0.06
Minor drug use	-0.06	0.04	-0.28	-0.06	0.04	-0.31
Minor drug Use X Psychopathy				0.004	0.01	0.11
R^2		0.07			0.08	
F for change in R^2		1.26				0.37

† $p < 0.10$ * $p < 0.05$

Section 10

Hierarchical regression models with lifetime overall drug use included as a covariate

	All offenders								
	Model 1			Model 2			Model 3		
	B	SE B	β	B	SE B	β	B	SE B	β
Dependence	1.15	0.82	0.19	0.41	1.18	0.07	3.17	2.13	0.52
Psychopathy	0.01	0.05	0.02	0.001	0.08	0.001	0.25	0.18	0.60
LCU	-0.09	0.08	-0.16	0.27	0.32	0.49	-0.59	0.64	-1.08
Overall drug use	-0.03	0.02	-0.23 †	-0.03	0.02	-0.23 †	-0.03	0.02	-0.24 †
Dependence x Psychopathy				-0.005	0.12	-0.007	-0.24	0.19	-0.36
LCU x Dependence				-0.43	0.31	-0.67	0.44	0.64	0.69
LCU x Psychopathy				0.01	0.01	0.15	0.09	0.05	1.00 †
Dependence x Psychopathy x LCU							-0.08	0.05	-0.77
R^2		0.08			0.14			0.16	
F for change in R^2		1.82			1.53			2.39	

	Non-Dependents					
	Model 1			Model 2		
	B	SE B	β	B	SE B	β
Psychopathy	-0.03	0.07	-0.07	0.27	0.18	0.64
LCU	0.44	0.29	0.23	-0.59	0.65	-0.31
Overall drug use	-0.04	0.02	-0.32 *	-0.04	0.02	-0.33 *
LCU X Psychopathy				0.09	0.05	0.75 †
R^2		0.13			0.19	
F for change in R^2		2.08			3.14†	

	Dependents					
	Model 1			Model 2		
	B	SE B	β	B	SE B	β
Psychopathy	-0.01	0.08	-0.01	-0.02	0.08	-0.05
LCU	-0.18	0.09	0.41 *	-0.21	0.09	-0.47 *
Overall drug use	0.004	0.03	0.03	0.004	0.03	0.03
LCU X Psychopathy				0.01	0.01	0.12
R^2		0.16			0.17	
F for change in R^2		2.04			0.39	

† $p < 0.10$ * $p < 0.05$

Section 11

Hierarchical regression models with lifetime major drug use included as a covariate

	All offenders								
	Model 1			Model 2			Model 3		
	B	SE B	β	B	SE B	β	B	SE B	β
Dependence	1.28	0.84	0.21	0.54	1.23	0.09	3.13	2.19	0.52
Psychopathy	-0.02	0.05	-0.05	-0.03	0.08	-0.08	0.20	0.18	0.49
LCU	-0.14	0.07	-0.26 †	0.21	0.33	0.38	-0.59	0.65	-1.11
Major drug use	-0.03	0.05	-0.06	-0.02	0.05	-0.05	-0.02	0.05	-0.04
Dependence x Psychopathy				0.004	0.12	0.005	-0.22	0.19	-0.32
LCU x Dependence				-0.42	0.32	-0.65	0.40	0.66	0.63
LCU x Psychopathy				0.01	0.01	0.15	0.09	0.05	0.95
Dependence x Psychopathy x LCU							-0.08	0.05	-0.73
R^2		0.05			0.11			0.13	
F for change in R^2		1.14			1.45			2.04	

	Non-Dependents					
	Model 1			Model 2		
	B	SE B	β	B	SE B	β
Psychopathy	-0.08	0.07	-0.19	0.19	0.19	0.46
LCU	0.38	0.31	0.20	-0.58	0.68	-0.31
Major drug use	0.02	0.08	0.03	0.02	0.08	0.03
LCU X Psychopathy				0.09	0.06	0.69
R^2		0.05			0.10	
F for change in R^2		0.71			2.47	

	Dependents					
	Model 1			Model 2		
	B	SE B	β	B	SE B	β
Psychopathy	0.005	0.07	0.01	-0.01	0.08	-0.03
LCU	-0.17	0.07	-0.39 *	-0.19	0.08	-0.45 *
Major drug use	-0.05	0.07	-0.12	-0.05	0.07	-0.13
LCU X Psychopathy				0.01	0.01	0.13
R^2		0.17			0.18	
F for change in R^2		2.25			0.46	

† $p < 0.10$ * $p < 0.05$

Section 12

Hierarchical regression models with lifetime minor drug use included as a covariate

	All offenders								
	Model 1			Model 2			Model 3		
	B	SE B	β	B	SE B	β	B	SE B	β
Dependence	1.16	0.82	0.19	0.26	1.18	0.04	3.07	2.13	0.51
Psychopathy	0.01	0.05	0.02	-0.01	0.08	-0.02	0.24	0.18	0.59
LCU	-0.09	0.08	-0.17	0.32	0.32	0.59	-0.55	0.64	-1.01
Minor drug use	-0.04	0.03	-0.23 †	-0.04	0.02	-0.24 †	-0.05	0.02	-0.25 †
Dependence x Psychopathy				0.02	0.12	0.02	-0.22	0.19	-0.33
LCU x Dependence				-0.48	0.31	-0.75	0.41	0.64	0.64
LCU x Psychopathy				0.01	0.01	0.13	0.09	0.05	1.001 †
Dependence x Psychopathy x LCU							-0.08	0.05	-0.79
R^2		0.09			0.14			0.17	
F for change in R^2		1.84			1.62			2.51	

	Non-Dependents					
	Model 1			Model 2		
	B	SE B	β	B	SE B	β
Psychopathy	-0.03	0.07	-0.08	0.26	0.18	0.62
LCU	0.49	0.29	0.26	-0.53	0.65	-0.28
Minor drug use	-0.06	0.03	-0.31 †	-0.06	0.03	-0.33 *
LCU X Psychopathy				0.09	0.05	0.74 †
R^2		0.12			0.19	
F for change in R^2		2.03			3.10 †	

	Dependents					
	Model 1			Model 2		
	B	SE B	β	B	SE B	β
Psychopathy	0.01	0.08	0.02	-0.01	0.08	-0.02
LCU	-0.16	0.08	-0.36 †	-0.18	0.09	-0.41 †
Minor drug use	-0.02	0.04	-0.08	-0.01	0.04	-0.07
LCU X Psychopathy				0.01	0.01	0.11
R^2		0.16			0.17	
F for change in R^2		2.09			0.34	

† $p < 0.10$ * $p < 0.05$

Section 13

Exploration of the relationship between age, lifetime cocaine use and low-frequency power ratio

We evaluated the potential influence of age in our reported regression models. In the dependent offender group, use was positively related to age ($r = 0.68$, $p < 0.001$), and both use ($r = -0.39$, $p = 0.02$) and age ($r = -0.51$, $p = 0.001$) were significantly negatively correlated with LFPR. Thus, we found that both use and age shared a highly parallel relationship with LFPR. To further investigate this relationship, we separated the group using a median split of age and performed additional regression analyses to assess how cocaine use related to LFPR in younger (<= 32 years old) and older (> 32 years old) cocaine-dependent offenders. The relationship between cocaine use and LFPR ($\beta = -0.39$, $p < 0.05$) was stronger in older cocaine-dependent offenders ($\beta = -0.23$, $p = 0.31$) than in younger cocaine-dependent offenders ($\beta = -0.008$, $p = 0.98$). Despite not reaching significance given the low power associated with a small sample size, these results suggest that the negative effects of cocaine use on LFPR that we identified could result from cumulative years of drug use, which are best identified in older cocaine-dependent offenders. The interaction between age and use was negatively related to LFPR ($r = -0.34$, $p = 0.04$), such that older cocaine-dependent offenders showed significantly lower LFPR than younger cocaine-dependent offenders, regardless of use level. These results suggest that the cumulative effects of years of cocaine use may heighten the negative effect that normal aging has on LFPR, with dependent individuals having used cocaine the longest showing a larger influence of use on LFPR.

In the non-dependent offender group, use was not significantly related to age (r = 0.09, p = 0.54) and both use (r = 0.13, p = 0.40) and age (r = -0.27, p = 0.06) were not significantly correlated with LFPR. While age showed a near significant effect on LFPR, as could be expected, use itself did not impact LFPR in Non-Dependent Offenders; thus, collinearity between age and use was not an issue in this group. The interaction between age and use was positively significantly related to LFPR (r = 0.38, p = 0.009), such that younger non-dependent offenders with a lower level of use showed increased LFPR in comparison to those who were young and showed a high level of use and those who were older and showed a high level of use. These results suggest that use had a negative impact on LFPR in young non-dependent offenders, while it did not seem to impact LFPR in older non-dependent offenders.

Correlations between age and LFPR did not statistically differ between groups (z = -1.24, p = 0.11); however, both groups significantly differed in how the interaction between age and cocaine use related to LFPR (z = 3.25, p = 0.001). Notably, cocaine-dependent offenders with low age/high use presented significantly higher LFPR than non-dependent offenders with low age/high use. This resulted in cocaine-dependent offenders of this category presenting decreased low-frequency and increased high-frequency activity compared to Non-Dependent Offenders. This suggests that younger cocaine-dependent and non-dependent offenders' rest-related spontaneous neural activity are differently impacted by higher levels of cocaine use, with cocaine-dependent offenders showing more neural activity disruptions. Taken together, these results suggest that it is not only cocaine use but rather the impact that cumulative cocaine use has on neural activity at different stages of adulthood that affects LFPR in dependent offenders, while

non-dependent offenders might not have accumulated enough drug use to present aversive effects on their neural integrity. Alternatively, it could be that reaching dependence status is instrumental to this effect, as much is still unknown regarding the neural processes sub-serving substance dependence.

Appendix B. Crime Inventory Questionnaire

CRIME INVENTORY

- The first thing you will do is record the official convictions for the adult section – or juvenile section based on the criminal record you review for the PCL-R or PCL-YV. Dismissed and Nolle Pros. do not count as convictions.
- For adults, if there is no juvenile record in their folder – you will write "No juvenile record available" in the first box of that section on the first page and put an arrow all the way through that section.
- Please complete the rest during the PCL-R (page 21) or PCL-YV (page 17) interview – where it is indicated.
- Start with the 1st crime listed and ask about adult convictions and non-convictions and juvenile convictions and non-convictions for each crime listed. You can say, "Now I am going to ask you some more specific questions about crimes that you have committed (juvenile and adult), including crimes for which you have never been caught."
- Make sure you let the individual know that the juvenile section will include crimes committed before the age of 18 and the adult section will include crimes committed at age 18 and older. For juvenile participants – juvenile crimes should include age 18 and younger and then adult crimes reported will start at age 19.
- Then start with the first item and say, "Have you ever assaulted a police officer as an adult?" If yes, ask, "How many times did you do this and not get convicted?" Then ask, "How many times were you convicted of this crime as an adult?" Then ask, "Did you ever assault a police officer as a juvenile?" If yes, ask, "How many times did you do this and not get convicted?" Follow this pattern for all crimes listed on the inventory and make sure to do the adult and juvenile section for each crime. If the participant is a juvenile – you can omit the adult section.
- For crimes that they committed on a daily basis – like drug possession or drug distribution – you can record years instead of number of times.
- For crimes like weapon possession – if they did not have a permit to carry the weapon (i.e. knife or gun) – that is considered the crime of weapons possession and you should mark it as an offense.
- For crimes like Assault/Battery – if an individual states they got into over 100 fights or over 50 fights then you can write "> 100" or ">50". But do try to get an approximate estimate.
- The "Other" categories are mainly there to note crimes that are found in the criminal file that are not listed on this sheet. This gives you an opportunity to ask the individual if they committed anymore of those types of crimes that they did not get caught for.

URSI: _____ EXAMINER: _____ DATE: _____

CRIME INVENTORY

	Self-Report Non-Convictions *18 and over include non-convicted crimes*	Self-Report Convictions *18 and over include any convictions*	Adult Criminal Record Convictions	Juvenile Self-Report Non-Convictions *Under 18, include non-convicted crimes*	Juvenile Self-Report Convictions *Under 18 include convictions*	Juvenile Criminal Record Convictions
OBSTRUCTION OF JUSTICE						
Assaulting a Police Officer						
Resisting Arrest/Fleeing						
THEFT OFFENSES						
Theft < $250						
Theft = $250 - $1000						
Theft = > $1000						
Burglary						
DRUG OFFENSES						
Drug Possession						
Drug Distribution						
FRAUD						
Fraud/Forgery						

1

URSI: _____ EXAMINER: _____ DATE: _____

	Self-Report Non-Convictions *18 and over include non-convicted crimes*	**Self-Report Convictions** *18 and over include any convictions*	**Adult Criminal Record Convictions**	**Juvenile Self-Report Non-Convictions** *Under 18, include non-convicted crimes*	**Juvenile Self-Report Convictions** *Under 18 include convictions*	**Juvenile Criminal Record Convictions**
ASSAULT						
Assault/Battery						
Domestic Assault/Battery						
Child Abuse/Neglect						
ROBBERY						
Robbery						
WEAPONS OFFENSES						
Weapons Possession						
MAJOR DRIVING OFFENSES						
Driving While Intoxicated						
Other Driving Offenses (specify) _____						

2

URSI: _____ EXAMINER: _____ DATE: _____

	Self-Report Non-Convictions *18 and over include non-convicted crimes*	**Self-Report Convictions** *18 and over include any convictions*	**Adult Criminal Record Convictions**	**Juvenile Self-Report Non-Convictions** *Under 18, include non-convicted crimes*	**Juvenile Self-Report Convictions** *Under 18 include convictions*	**Juvenile Criminal Record Convictions**
SEXUAL OFFENSES						
Rape/Sexual Assault						
Child Sexual Assault/Incest						
Prostitution						
Pimping						
KIDNAPPING						
Unlawful Confinement/Hijacking						
ARSON						
Arson						

3

URSI: _____ EXAMINER: _____ DATE: _____

	Self-Report Non-Convictions *18 and over include non-convicted crimes*	**Self-Report Convictions** *18 and over include any convictions*	**Adult Criminal Record Convictions**	**Juvenile Self-Report Non-Convictions** *Under 18, include non-convicted crimes*	**Juvenile Self-Report Convictions** *Under 18 include convictions*	**Juvenile Criminal Record Convictions**
ESCAPE						
Escape						
Probation/Parole Violation						
Failure to Appear						
MURDER						
Murder						
Manslaughter						
Attempted Murder						

4

URSI: _____ EXAMINER: _____ DATE: _____

	Self-Report Non-Convictions *18 and over include non-convicted crimes*	**Self-Report Convictions** *18 and over include any convictions*	**Adult Criminal Record Convictions**	**Juvenile Self-Report Non-Convictions** *Under 18, include non-convicted crimes*	**Juvenile Self-Report Convictions** *Under 18 include convictions*	**Juvenile Criminal Record Convictions**
MISCELLANEOUS						
Vandalism, Criminal Mischief, Disorderly Conduct, Etc.						
Other (specify) _____						
Other (specify) _____						
Other (specify) _____						
Other (specify) _____						
Other (specify) _____						

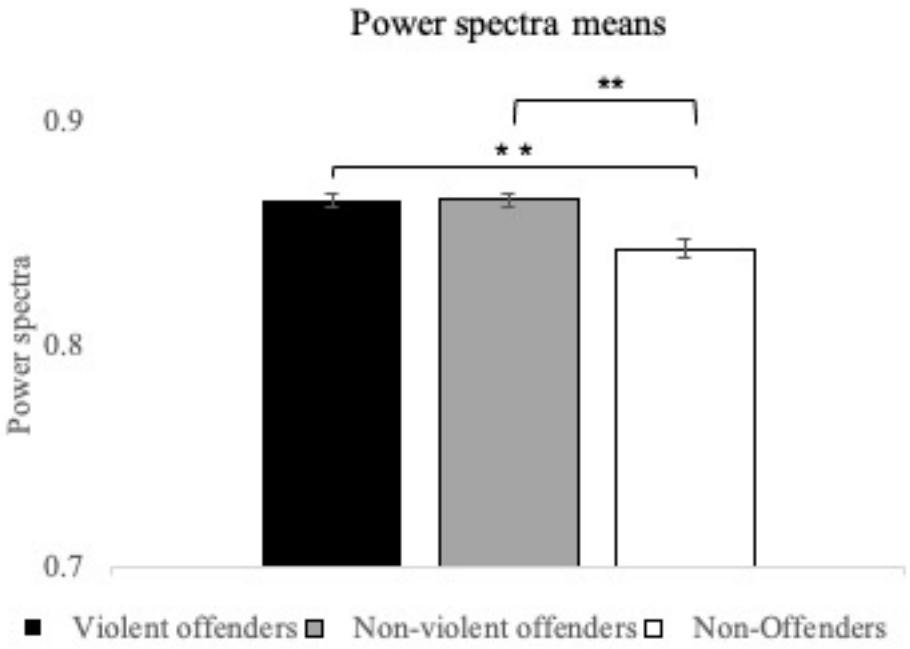
5

Appendix C. Supplementary materials Study 2

C.1. Main effects 3 (Group) x 2 (Bins) x 8 (Network) omnibus ANOVA

The 3 (Group) x 2 (Bins) x 8 (Network) omnibus ANOVA identified a main effect of Group, F (2, 94) = 9.11, $p < 0.001$, with pairwise comparisons indicating that Violent (M = 0.86; SD = 0.03) and Non-Violent (M = 0.87; SD = 0.04) groups showed similar overall power spectra, p = 1.00, that were both higher than Non-Offenders (M = 0.84; SD = 0.05; both ps < .001; see Figure C.1). A main effect of Network, F (7, 94) = 8.14, $p < 0.001$, was observed, with the SMN showing the highest level of power spectra and the Visual network showing the lowest (see Figure C.2 for a representation of mean power spectra in each RSN). A main effect of Bin was also observed, F (1, 94) = 1031.57, $p < 0.001$, with the low frequency bin (M = 1.15; SD= 0.08) showing higher overall power than the high-frequency bin (M = 0.56; SD = 0.10).

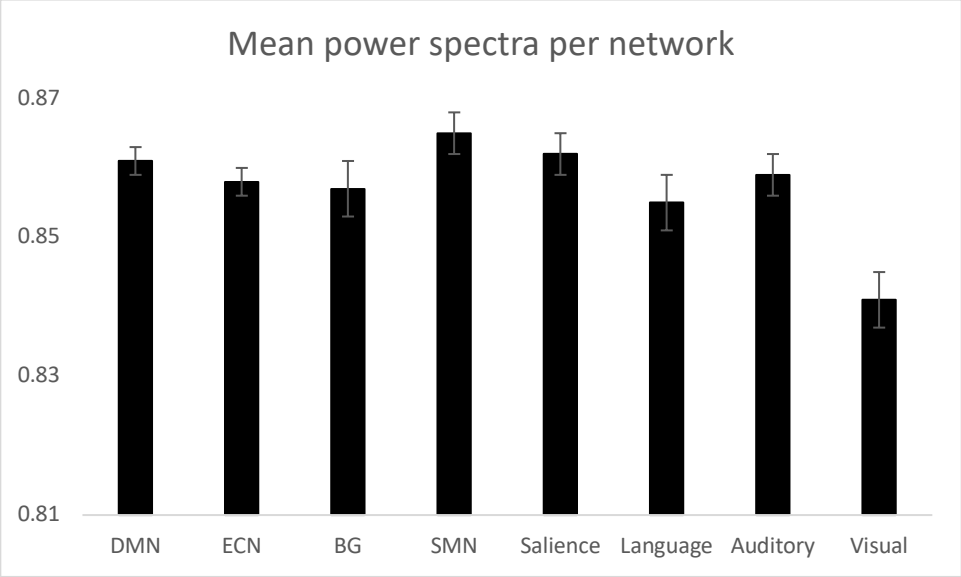
Figure C.1



Note. Violent and Non-Violent offender groups presented with increased power spectra compared to Non-Offenders. No differences were observed between the offender groups.

** *p(FWE)* < 0.001

Figure C.2



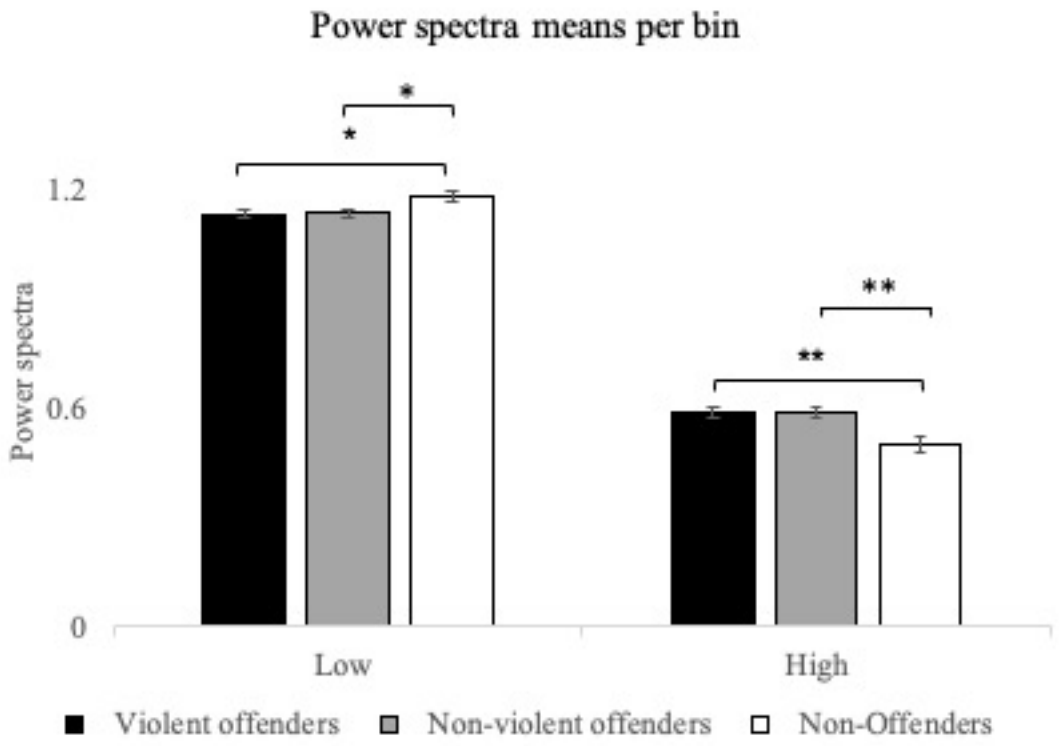
Note. Mean power spectra activity in each resting-state network across groups (i.e., Non-Offenders, Violent-Offenders and Non-Offenders).

C.2. Breakdown of BinxGroup interaction

As shown in Figure C.3, while the two offender groups did not present differences across low or high-frequency bins, several differences were observed between these groups and the Non-Offender group. Specifically, Violent and Non-Violent Offenders showed significantly decreased low-frequency power, *t*s = -2.67, -2.51, *p*s (FWE) < 0.05, and significantly increased high-frequency power, *t*s = 3.45, 3.69, *p*s (FWE) < 0.001, compared to Non-Offenders. As may be expected, given the pattern of low/high frequency activity, both Violent Offenders, $t = -2.21$, $p(FWE) = 0.001$, and Non-Violent Offenders, $t = -3.21$, $p(FWE) = 0.001$, presented with significantly decreased LFPR in comparison to Non-Offenders (see Figure C.4).

Figure C.3

Comparison of mean low- and high-frequency power spectra across groups

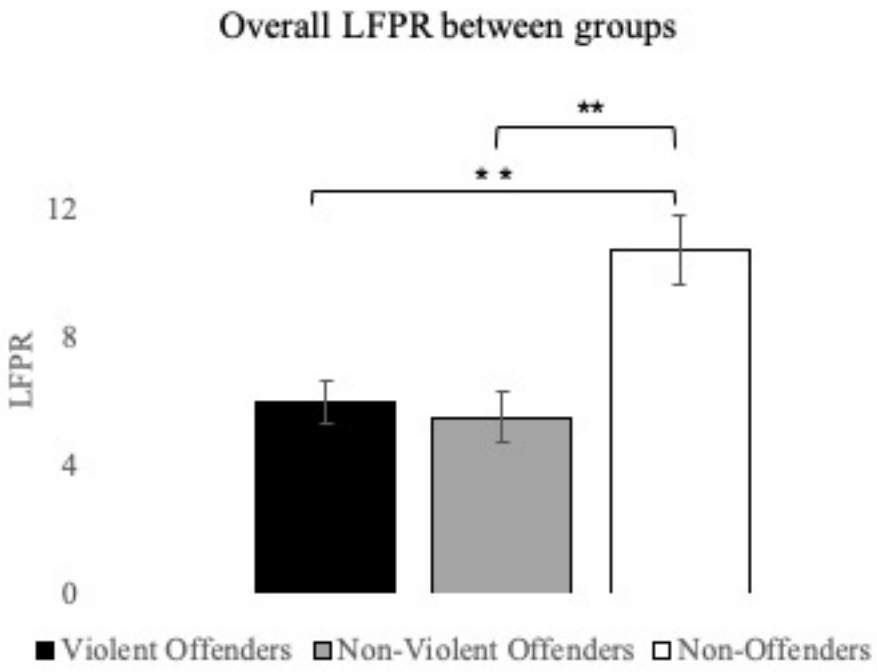


Note. Violent and Non-Violent offenders presented reduced low-frequency activity and increased high-frequency activity compared to Non-Offenders, while no differences were observed between Violent and Non-Violent offenders.

* *p(FWE)* < 0.05 ** *p(FWE)* < 0.001

Figure C.4

Comparison of mean global low-frequency power ratio between groups



Note. Violent and Non-Violent offenders presented reduced global low-frequency power ratio (LFPR) compared to non-offenders, while no differences were observed between Violent and Non-Violent offenders.

** $p(FWE) < 0.001$

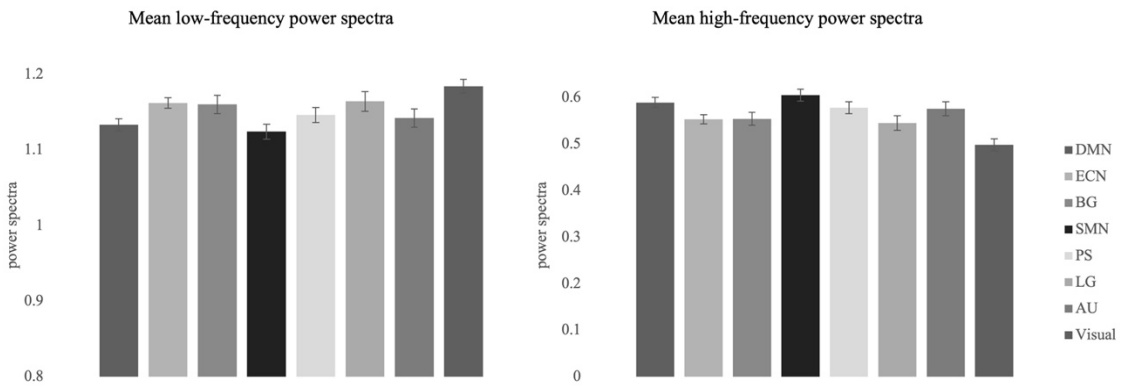
C.3. Bin x Network interaction

Some networks significantly differed from others regarding their mean low and/or high-frequency activity level. The visual network presented a significantly higher level of low-frequency activity in comparison to the DMN, SMN, PS and Au networks (see Figure C.5). Moreover, the DMN presented significantly lower low-frequency activity than the ECN, which presented significantly higher low-frequency activity than the SMN network. The visual network presented a significantly lower high-frequency activity level

than all the other RSNs. Moreover, the DMN presented significantly increased high-frequency activity compared to the ECN and LG networks, and the SMN presented significantly increased high-frequency power compared to the ECN, BG and LG networks.

Figure C.5

Mean low- and high-frequency power spectra in each resting-state network across groups

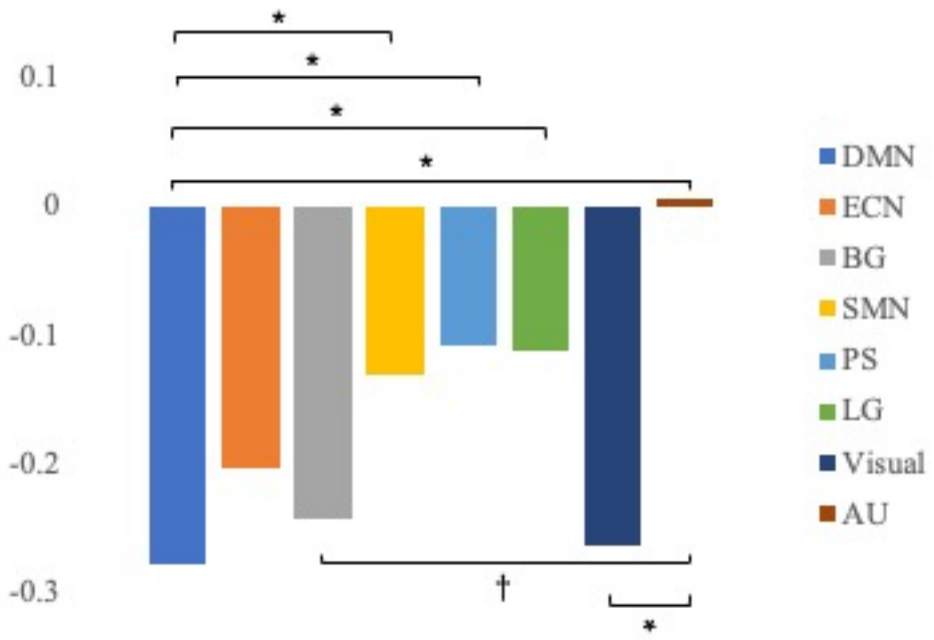


C.4. Fisher's r to z transformation correlations between low-frequency power ratio and crime severity in each resting-state network

Using Fisher's r to z transformation, I compared the magnitude of the correlations between LFPR and crime severity in each RSN (see Figure C.6). The DMN (z = -2.47, p = 0.007) and Visual (z = -1.671, p = 0.047) networks presented significant, and near significant for the BG (z = -1.48, p = 0.07), higher magnitude of correlation in comparison to the AU. Additionally, the DMN presented significantly higher magnitude of correlation in comparison to the SMN (z = -1.66, p = 0.048), PS (z = -1.79, p = 0.037) and Language networks (z = -1.79, p = 0.037).

Figure C.6

Comparison of magnitudes of correlation between global low-frequency power ratio and convictions in each resting-state network across offenders



† *p(FWE)* < 0.10 * *p(FWE)* < 0.05

C.5. Results regression using convictions and cocaine use to predict global low-frequency power ratio

	Influence of number of convictions and cocaine use of LFPR								
	Model 1			Model 2			Model 3		
	B	SE B	β	B	SE B	β	B	SE B	β
Convictions	-0.11	0.06	-0.22*	-0.09	0.06	-0.18	-0.09	0.06	-0.19
Cocaine use				-0.06	0.06	-0.12	-0.08	0.07	-0.15
Convictions x Cocaine use							0.005	0.01	0.07
R^2		0.05			0.06			0.07	
F for change in R^2		3.95*			0.96			0.32	

Note. Low-frequency power ratio (LFPR)

* $p < 0.05$

C.6. Distribution of criminal convictions in Offenders

	N	*Median*	Range
Violent Crimes	45	2	1 - 6
Assault/Battery	34	2	1 - 6
Robbery	6	2	1 - 2
Rape/Sexual assault	6	2	1 - 3
Murder	2	1	0
Kidnapping	3	1	0
Non-Violent Crimes	75	8	1 - 26
Resisting/Escape	47	2	1 - 6
Theft	30	1	1 - 6
Drug-related crimes	36	2	1 - 4
Fraud/Forgery	6	1.5	1 - 3
Weapon possession	3	1	0
DWI	21	1	1 - 4
Other driving offences	51	3	1 - 9
Vandalism	21	1	1 - 6
Miscellaneous	54	2	1 - 6

C.7. Classification metrics of the support vector classifier models

As accuracy only represents the ratio of correct predictions to the total observations, I also tested for the precision, recall and f1-scores of the models to obtain a more detailed representation of the models' classification ability. Precision is the ratio of correctly predicted positive observations to the total predicted positive observations. A high precision score indicates a low false-positive rate. Recall (more commonly referred to as sensitivity) measures the ratio of correctly predicted positive observations to all observations in each class. In other words, it indicates: of those who recidivated, how many did the model correctly predict? The higher the recall score, the more sensitive the model is. Finally, the f1-score is a weighted average of precision and recall, which takes both false positives and false negatives into account. As with the other metrics, higher is

better. As shown in Table C.1, both models showed similarly low precision (indicating that the model included a high number of false positives), moderate recall (indicating that the model correctly identified around half of the participants who had re-offended as having re-offended) and low f1-scores (illustrating the high cost of false-negative identifications on the accuracy of the models) when identifying those who had re-offended. Precision, recall and f1-scores were also very similar when identifying those who did not recidivate in both models. Thus, although Model 2 showed slightly greater accuracy than Model 1, precision, recall, and f1-scores were similar in both models, suggesting that the models did not greatly differ in their capacity to identify offenders who re-offended after a year.

Table C.1

Classification metrics of support vector classifier models

	Model 1			Model 2		
	Precision	Recall	f1-score	Precision	Recall	f1-score
Did not recidivate	0.87	0.48	0.62	0.86	0.52	0.65
Did recidivate	0.12	0.5	0.2	0.11	0.4	0.17