

University of New Mexico

UNM Digital Repository

Psychology ETDs

Electronic Theses and Dissertations

Summer 8-1-2023

IDENTIFYING PSYCHOSOCIAL AND NEURAL CORRELATES ASSOCIATED WITH FUTURE HOMICIDE IN A SAMPLE OF INCARCERATED BOYS

Samantha N. Rodriguez

University of New Mexico - Main Campus

Follow this and additional works at: https://digitalrepository.unm.edu/psy_etds



Part of the [Psychology Commons](#)

Recommended Citation

Rodriguez, Samantha N.. "IDENTIFYING PSYCHOSOCIAL AND NEURAL CORRELATES ASSOCIATED WITH FUTURE HOMICIDE IN A SAMPLE OF INCARCERATED BOYS." (2023). https://digitalrepository.unm.edu/psy_etds/399

This Thesis is brought to you for free and open access by the Electronic Theses and Dissertations at UNM Digital Repository. It has been accepted for inclusion in Psychology ETDs by an authorized administrator of UNM Digital Repository. For more information, please contact disc@unm.edu.

**IDENTIFYING PSYCHOSOCIAL AND NEURAL
CORRELATES ASSOCIATED WITH FUTURE HOMICIDE IN
A SAMPLE OF INCARCERATED BOYS**

by

SAMANTHA N. RODRIGUEZ

**BACHELOR OF SCIENCE IN NEUROSCIENCE
BACHELOR OF ARTS IN SOCIOLOGY
UNIVERSITY OF DELAWARE, 2019**

THESIS

Submitted in Partial Fulfillment of the
Requirements for the Degree of

**Master of Science
Psychology**

The University of New Mexico
Albuquerque, New Mexico

August, 2023

**IDENTIFYING PSYCHOSOCIAL AND NEURAL
CORRELATES ASSOCIATED WITH FUTURE HOMICIDE IN
A SAMPLE OF INCARCERATED BOYS**

by

Samantha N. Rodriguez

B.S., Neuroscience, University of Delaware, 2019

B.A., Sociology, University of Delaware, 2019

M.S., Psychology, University of New Mexico, 2023

ABSTRACT

Previous research has observed significant group differences regarding neuroanatomical and psychosocial variables between incarcerated boys who have and have not previously committed a homicide, resulting in successful postdictive classification (Cope et al., 2014). However, no study to date has investigated whether similar group differences characterize future homicide offenders. Following the methodology of Cope et al. (2014), the current study aimed to identify baseline neural, clinical, and environmental deficits (collected in a sample of $n = 242$ incarcerated juvenile offenders) associated with future homicidal behavior as adults. Results indicated that youth who went on to commit homicide as adults were characterized by higher psychopathic traits and reduced gray matter volume in brain regions related to affective processing compared to youth who did not commit a homicide as adults. The current study provides the foundation for further longitudinal studies investigating the development of traits and neural deficits associated with future homicide, potentially leading to more accurate re-offense risk assessment and early behavioral intervention.

TABLE OF CONTENTS

LIST OF FIGURES	v
LIST OF TABLES	vi
INTRODUCTION.....	1
Juvenile Offending and Recidivism	1
Psychopathic Traits in Youth Offenders	2
Homicide	4
The Current Study	6
METHODS	7
Participants	7
Data Collection.....	9
Data Analysis	14
RESULTS.....	16
Independent Samples T-Tests	16
Fisher’s Exact Test	16
Voxel-Based Morphometry	17
DISCUSSION	17
LIMITATIONS	21
FUTURE DIRECTIONS	22
CONCLUSION.....	23
REFERENCES	24

LIST OF FIGURES

FIGURE 1	33
FIGURE 2	34

LIST OF TABLES

TABLE 1	35
TABLE 2	37
TABLE 3	38

Introduction

Juvenile Offending and Recidivism

There are currently over 60,000 youth offenders housed in correctional and detention facilities across the United States (American Civil Liberties Union, 2022). Up to 80% of these juveniles will re-offend within three years of release (Council of State Governments-Justice Center, 2021). In a study comparing high risk youth who did and did not receive behavioral treatment while housed in a juvenile correctional facility, 49% of boys who did not receive treatment violently re-offended within two years of release (Caldwell et al., 2006). In a separate sample of juvenile offenders, 97% of boys who demonstrated psychopathic traits re-offended within an average of about three years, including 69% who re-offending violently. Conversely, 76% of boys who did not demonstrate psychopathic traits re-offended within about three years, with only 40% re-offending violently (Vincent et al., 2008). Taken together, previous research has demonstrated that while overall recidivism in incarcerated youth offenders is high, it is not uniform across offenders.

According to the life-course-persistent theory of offending, the majority of adolescent offenders stop behaving antisocially when entering adulthood (adolescent-limited offending) while a small subset of adolescent offenders continue to engage in antisocial behavior throughout their life (lifecourse persistent offending) (Moffitt, 1993). While about half of juvenile offenders desist from criminal activity by the age of 25, evidence suggests those who continue to engage in criminal activity in adulthood are characterized by an increased rate of violence and lethality (National Institute of Justice, 2014). Moffitt's 1993 theory has been validated by scientific studies across multiple decades. For example, Wolfgang et al. (1972) found that only 6% of their sample of 10,000 adolescent boys committed the vast majority of all violent crimes (i.e., around 70% of crimes, including more severe crimes such as murder, rape, and aggravated assault).

Similarly, Vaughn et al. (2014) observed that less than 4.7% of adolescents in a nationally representative sample committed over 30% of all severe violent crimes and up to 70.5% of other types of crime (e.g., drug distribution). It follows, then, that the majority of resources allocated for risk assessment of juvenile offenders should be dedicated to identifying these lifecourse-persistent offenders (Vaughn et al., 2014). As demonstrated by the difference in recidivism rate between incarcerated boys who did and did not demonstrate psychopathic traits (Vincent et al., 2008), research should aim to identify if certain variables collected during adolescence can help better characterize the difference between adolescent-limited and life-course persistent offenders.

Psychopathic Traits in Youth Offenders

Psychopathic traits are known to predict risk for future antisocial behavior. Individuals scoring high on psychopathic traits are characterized by a constellation of affective (i.e., superficial charm, pathological lying, impression management, manipulation, callousness, lack of remorse, shallow affect, and failure to accept responsibility) and behavioral (i.e., stimulation-seeking, lack of goals, impulsivity, irresponsibility, parasitic orientation, poor anger control, serious criminal behavior, criminal versatility, early behavioral problems, and serious revocation of release) traits that often manifest as increased antisocial behavior. In adolescents, these traits are commonly assessed via the Hare Psychopathy Checklist: Youth Version (PCL:YV; Forth et al., 2003), which consists of a semi-structured interview and supplemental review of institutional files. Previous research has demonstrated a strong relationship between psychopathic traits in youth offenders and recidivism for both violent and non-violent crimes (Schmidt et al., 2006; Stockdale et al., 2010; Vincent et al., 2008). Additional research indicates a link between higher psychopathic traits, specifically callous-unemotional traits (e.g., callousness, lack of empathy,

guilt, and remorse), and life-course persistent offenders who are at an increased risk for future offending (Frick, 2009).

In adult offenders, a number of neuroanatomical deficits have been associated with individuals scoring high on psychopathic traits. For example, the paralimbic system, consisting of regions including the temporal pole, anterior cingulate cortex (ACC), posterior cingulate cortex (PCC), orbitofrontal cortex (OFC), insula, amygdala, and parahippocampal regions, has been implicated in psychopathy in adults (Ermer et al., 2012; or see Kiehl, 2006; Johanson et al., 2020 for reviews) and youth offenders (Cope et al., 2014; Ermer et al., 2013). Additionally, a systematic literature review found evidence to support the relationship between deficits in the paralimbic system and psychopathic traits in adults, with the addition of the dorsomedial prefrontal cortex, frontal gyrus, temporal gyrus, fusiform gyrus, precuneus, and postcentral gyrus (Johanson et al., 2020). Additionally, a review performed by Deming & Koenigs (2020) observed that psychopathic traits were associated with increased neuroactivity in regions included within the default mode network and reduced activity in regions included in the salience network among adults. Thijssen & Kiehl (2017) also found a significant relationship between psychopathic traits and activation in the default mode network and salience network, as well as executive control network in a sample of youth offenders. Finally, Umbach et al. (2015) also demonstrated that psychopathic traits in youth offenders have been associated with similar neural deficits as adults, including reduced volume and activation in the amygdala and prefrontal cortex in both adolescents and adults. As both youth and adults scoring high on psychopathic traits exhibit similar neural abnormalities, investigating abnormalities associated with psychopathic traits among high-risk youth offenders may prove vital to understanding the underpinnings of various types of violent reoffending in adulthood, including homicide.

Homicide

Homicide is perhaps the ultimate interpersonal violent crime. To date, studies of the psychological and neurological mechanisms behind this behavior are limited. However, studies performed have documented the relationship between psychopathic traits and homicide, and successfully identified a set of neuroanatomical deficits specific to homicide in adult forensic samples. A meta-analysis conducted by Fox and DeLisi (2019) identified a strong relationship between various forms of homicide and psychopathic traits among adult offenders, concluding that higher psychopathy scores put offenders at an increased risk for homicidal behavior. Regarding neural findings, Radeljack et al. (2010) summarized that deficits in the prefrontal cortex (PFC), temporal gyrus, amygdala, hippocampus, and ACC are commonly identified among adult homicide offenders. Specifically, they cite a series of studies conducted by Raine and colleagues in which Positron Emission Tomography revealed asymmetry in activity in the amygdala, thalamus, and medial temporal lobe, as well as reduced metabolism in the PFC, superior parietal gyrus, left angular gyrus, and corpus callosum. More recently, Sajous-Turner et al. (2020) found deficits in gray matter volumes in the orbitofrontal/ventromedial PFC, anterior temporal cortex, insula, medial prefrontal/ACC, and precuneus/PCC in incarcerated men who committed homicide compared to incarcerated men who did not commit homicide. Importantly, the majority of neural deficits previously identified in adult homicide offenders are also regions implicated in psychopathy (i.e., temporal gyrus, amygdala, hippocampus, (Radeljack et al., 2010), ACC (Radeljack et al., 2010; Sajous-Turner et al., 2021), insula, and posterior cingulate cortex (Sajous-Turner et al., 2021)). Taken together, these findings suggest a network of structures associated with psychopathy that may hold value for better elucidating neural deficits associated with homicidal behavior.

Unlike studies in adult samples, the examination of homicide in juvenile samples has focused more on understanding psychosocial factors compared to neuroimaging variables that may contribute to this extreme form of antisocial behavior. For example, Darby et al. (1998) examined the various types of familial abuse that may be contributing factors to acts of homicide committed by adolescents. They found that juveniles who committed a homicide were more likely to have experienced severe childhood abuse and were more likely to engage in substance use and aggressive behavior than juveniles who did not commit a homicide. Additionally, Khachatryan et al. (2016) found that juveniles who committed a homicide as part of a group were more likely to have a criminal record prior to committing homicide and were more likely to reoffend compared to juveniles who committed homicide.

Recently, neuroimaging studies have been performed to better understand neural deficits associated with youth who have previously committed homicide. Importantly, Cope et al. (2014) used a machine learning model that included both neuroanatomical and psychosocial variables to postdictively classify which incarcerated juvenile boys committed a homicide. Specifically, a model including gray matter volumes previously associated with psychopathic traits in adults (i.e., left lateral OFC, medial OFC, anterior cingulate, posterior cingulate, right temporal pole, left temporal pole), and clinical assessment variables, including PCL:YV scores, number of criminal convictions, and socioeconomic status demonstrated 81.2% overall accuracy, 75% specificity (i.e., correctly identifying homicide offenders), and 82.2% sensitivity (i.e., correctly identifying non-homicide offenders). Additionally, Cope et al. (2014) found that juvenile offenders who committed a homicide exhibited reduced gray matter volume in the hippocampus, superior temporal gyrus, middle temporal gyrus, parahippocampal gyrus, fusiform gyrus, and inferior temporal gyrus compared to incarcerated youth who had not previously committed a

homicide. This study was the first to demonstrate the utility of neuroimaging data in classifying juvenile offenders who have previously committed a homicide, and provides evidence for the connection between similar neural deficits associated with psychopathy and homicide. Further, the findings in both incarcerated youth (Cope et al., 2014) and adult offenders (Sajous-Turner et al., 2020) who previously committed homicide suggests the existence of similar neuroanatomical correlates associated with homicide across the lifespan.

Current Study

Cope et al. (2014) were able to successfully classify juvenile offenders who had and had not previously committed homicide based on neuroimaging, clinical, and environmental factors. While this study indicates the presence of group differences between homicide and non-homicide offenders, it does not provide information about the behaviors and neural differences underlying *future* homicidal behavior. Alper et al. (2018) found that 60% of the total arrests in their sample of 401,288 offenders occurred between the fourth and ninth years of follow-up, indicating the importance of extended longitudinal follow-up periods. To fill these gaps, the goal of the current study was to use similar neuroimaging, clinical, and environmental variables as Cope et al. (2014) to identify group differences between juvenile offenders who did and did not commit a future homicide up to 14-years after release from a juvenile correctional facility. Specifically, we sought to identify which of our former participants incarcerated at a maximum-security juvenile correctional facility committed homicide later as adults; once these groups were identified, we investigated whether youth who later committed homicide significantly differed from youth who did not later commit homicide on the same psychosocial and neuroimaging data used in our previous report (Cope et al., 2014). Given that prior work demonstrates a strong relationship between psychopathic traits and homicidal behavior (Cope et al., 2014; Fox & DeLisi, 2019;

Sajous-Turner et al., 2020), we hypothesized that boys who committed a future homicide would score higher on the PCL:YV than boys who did not commit a future homicide. Additionally, we hypothesized similar neural deficits characteristic of youth who *previously* committed homicide would also be associated with youth who committed homicide in the *future*.

Methods

Participants

The total sample includes $n = 242$ incarcerated adolescent boys who were housed at a maximum-security juvenile correctional facility in New Mexico. The sample used in the present study is a subsample of youth included in the Southwest Advanced Neuroimaging Cohort – Youth (SWANC-Y) dataset (R01 MH071896, PI: Kiehl). Participants ranged from 13 to 19 years of age ($M = 17.6$, $SD = 1.1$) at the time of their MRI scan. Participants self-identified their race as American Indian or Alaskan Native ($n = 30$), Black or African American ($n = 13$), Native Hawaiian or Other Pacific Islander ($n = 1$), White ($n = 147$), or more than one race ($n = 2$). An additional 44 participants chose not to disclose their race. Participants also self-identified as either Hispanic or Latino ($n = 183$) or Non-Hispanic or Latino ($n = 52$). An additional seven participants chose not to disclose their ethnicity.

Data for the original study, and that which is used in analyses here, was collected between the years 2007 and 2011. At the time of initial contact, participants under the age of 18 provided written assent and their parents or legal guardians provided written consent, and participants over the age of 18 provided written consent. The inclusion criteria for the current study was a T1-weighted image collected while participants were housed at the juvenile correctional facility. Exclusion criteria of the larger study, and therefore the current study as well, included: traumatic brain injury with loss of consciousness longer than 30 minutes, past or

current history of CNS disease (e.g., stroke, multiple sclerosis, seizures, etc.), current or history of psychotic disorder as defined by the Diagnostic and Statistical Manual of Mental Disorders—Fourth Edition (DSM-IV; American Psychiatric Association, 2000), hypertension or diabetes, mental retardation or fetal alcohol syndrome, MRI contraindication (e.g., ferrous metal in body), and low reading level (i.e., less than fourth-grade reading level). In addition, female sex was an exclusion criterion for the current study. Gender has been found to be the number one predictor of future homicide (Baglivio, 2009), and only $n = 1$ incarcerated girl with a T1-weighted image committed a homicide after release. Because this is an insufficient sample size for an examination of sex differences, female participants were excluded from the current study.

The final $n = 242$ sample was divided into two groups depending on whether or not previous participants committed a homicide in the future compared to control participants who did not commit a homicide following their release from the juvenile correctional facility (see *Assessments* for details on the collection of outcomes data). The Future Homicide group consisted of $n = 23$ former participants who committed a homicide after release from the juvenile correctional facility. Participants were included in the Future Homicide group if they were convicted of Murder in any degree, Manslaughter, or Attempted Murder in any degree after release. However, participants were excluded from the Future Homicide group if they were convicted of Vehicular Manslaughter if review of the crime indicated they did not willfully and intentionally cause of the death of another person (i.e., car accident). $n = 16$ were convicted of murder, $n = 2$ were convicted of manslaughter, and $n = 5$ were convicted of attempted murder after they were released from the juvenile correctional facility. Conversely, participants were included in the No Homicide group if they did not have any of the above convictions after

release from the juvenile detention center *and* did not commit a homicide in the past¹.

Specifically, participants were excluded from the No Homicide group if they had a conviction of Murder, Manslaughter, or Attempted Murder in any degree, or had been charged with Murder, Manslaughter, or Attempted Murder but were convicted of Aggravated Assault resulting in Great Bodily Harm as a juvenile if review of their juvenile institutional file determined they willfully and intentionally caused the death of another person.

Data Collection

Data included in the current study was collected in two phases. In the first phase, data was collected on-location at the juvenile correctional facility from 2007 to 2011, including baseline data to be used in the current analysis (i.e., psychosocial variables and neuroimaging data). In the second phase, outcomes data (i.e., charges and convictions after release from the juvenile correctional facility) was collected from criminal records obtained from the Center for Science and Law's Criminal Record Database (CRD; Ormachea et al., 2015). Re-offense data, including homicide data, was extracted from the CRD of criminal court records for offenders in New Mexico. Data in the CRD were matched to previous participants via four separate identifiers (i.e., first and last name, date of birth, and social security number). Extensive online searches including social media, White Pages, Been Verified, county records, New Mexico Corrections Department offender search, and out of state inmate databases were conducted for the entire sample. This enabled us to compile recidivism data for participants who were not found in the CRD. Homicide offenses after release were operationally defined as a conviction of (a) Murder in any degree, (b) Manslaughter, and (c) Attempted Murder in any degree, but

¹ All n = 20 boys included the Homicide group in Cope et al. (2014) were excluded from the No Homicide group in the current study.

participants with Vehicular Manslaughter convictions were excluded if details from the crime indicated there was no intent to cause death.

High-resolution T1-weighted structural MRI scans were acquired with the Mind Research Network Siemens 1.5 T Avanto mobile scanner, stationed at the correctional facility, using a multi-echo MPRAGE pulse sequence (repetition time = 2530 ms, echo times = 1.64 ms, 3.50 ms, 5.36 ms, 7.22 ms, inversion time = 1100 ms, flip angle = 7°, slice thickness = 1.3 mm, matrix size = 256 × 256) yielding 128 sagittal slices with an in-plane resolution of 1.0 × 1.0 mm. Data were pre-processed and analyzed using Statistical Parametric Mapping software (SPM12; Wellcome Department of Cognitive Neurology, London, UK; <http://www.fil.ion.ucl.ac.uk/spm>). T1 images were automatically oriented to anterior-posterior commissure (AC-PC) alignment using the `auto_acpc_reorient` algorithm (https://github.com/lrq3000/auto_acpc_reorient), and were inspected to ensure proper realignment. Images were then analyzed via the Unified Segmentation approach as implemented in SPM12 (Ashburner & Friston, 2005). Unified Segmentation allows for image registration based on Gaussian mixture modelling, tissue classification with warped prior probability maps, and bias correction to be combined in the same generative model. During spatial normalization data were resampled to 2 × 2 × 2 mm. Subsequent segmentation partitioned the images into gray matter, white matter, and CSF, which were then modulated to preserve total volume. Voxels with a matter value of <.15 were excluded in order to remove possible edge effects between gray and white matter. Finally, segmented images were smoothed with a 10 mm full-width at half-maximum (FWHM) Gaussian kernel.

Assessments

The majority of the variables included in analyses here were derived from Cope et al. (2014), where homicide vs. non-homicide offenders were successfully postdictively classified using machine learning methods. Cope et al. (2014) included age, socioeconomic status, IQ, PCL:YV scores, Inventory of Callous-Unemotional Traits (Youth Self-Report Version; ICU (Essau et al., 2006)) scores, impulsivity scores, substance use history, number of traumatic brain injuries (TBI), number of criminal convictions, brain volume, and clinical diagnoses in analyses. Of these variables, all were included in the current study except for ICU scores. Previous research suggests limited construct overlap between items included in the PCL:YV and those included in self-report measures (Fink et al., 2012; Maurer et al., 2018). Therefore, the PCL:YV was the only assessment of youth psychopathic traits included in the current study. Age ($M = 17.58$, $SD = 1.1$; 13.8 – 19.5) was calculated as participants' age on the day of their MRI scan. Socioeconomic status (SES) ($M = 4.61$, $SD = 0.07$; 4.43 – 5.03) was quantified as the median household income of the county of their last residence prior to incarceration. For participants under the age of 16 at the time of data collection, full-scale IQ ($M = 92.46$, $SD = 10.3$; 63 - 134) was estimated via the Vocabulary and Matrix Reasoning subtests included in the Wechsler Intelligence Scale for Children—Fourth Edition (Wechsler, 2003; Sattler and Dumont, 2004). For participants over the age of 16 at the time of data collection, full-scale IQ was estimated via Vocabulary and Matrix Reasoning subtests of the Wechsler Adult Intelligence Scale (Wechsler, 1997; Ryan et al., 1999). For the majority of participants, psychopathic traits were assessed via the PCL:YV (Forth et al., 2003). However, $n = 3$ participants who were over the age of 18 at the time of data collection were housed at the juvenile correctional facility while awaiting transfer to an adult correctional facility. For these participants, psychopathy scores were assessed via the Psychopathy Checklist—Revised (PCL-R; Hare, 2003). PCL:YV and PCL-R Total ($M = 23.30$,

$SD = 5.5$; 2 – 35), Factor 1 ($M = 6.49$, $SD = 2.8$; 0 - 15), and Factor 2 ($M = 14.52$, $SD = 3.0$; 1 - 20) scores were included in analyses. In both PCL:YV and PCL-R assessments, Factor 1 scores measure interpersonal (e.g., superficial charm, pathological lying, manipulation) and affective (e.g., lack of remorse, callousness, and failure to accept responsibility) traits, and Factor 2 scores measure lifestyle/behavioral (e.g., stimulation seeking, parasitic lifestyle, impulsivity, irresponsibility) and antisocial/developmental (e.g., poor behavioral control, early behavioral problems, probation violation, and criminal versatility) traits. Impulsivity ($M = 66.15$, $SD = 14.6$; 0 - 100) scores were assessed via the Barratt Impulsiveness Scale (BIS-11; Patton et al., 1995). BIS-11 Total scores were included here. Substance use history was quantified via two methods. First, number of substance use dependencies ($M = 2.20$, $SD = 1.5$; 0 - 10) was quantified as the total number of substances for which participants met lifetime dependence criteria for based on the Kiddie Schedule for Affective Disorder and Schizophrenia (Kaufman et al., 1997), a structured interview designed to assess forms of psychopathology and substance use for individuals under the age of 18. Specifically, KSADS assesses substance use for ten substance categories: alcohol, cannabis, stimulants, sedatives/hypnotics/anxiolytics, cocaine, opioids, phencyclidine (PCP), hallucinogens, solvents/inhalants, and other substances. Second, years of substance use ($M = 6.18$, $SD = 2.7$; 0 – 14.5) was assessed via a modified version of the Addiction Severity Index (McLellan et al., 1992). Years of regular substance use were totaled for each substance, divided by age to account for differences in opportunity to use, and square root transformed to adjust for skewness. TBI history ($M = .70$, $SD = .76$; 0 - 4) was assessed via the Rivermead Post-Concussion Symptoms Questionnaire (King et al., 1995). Here, total number of TBIs in which a loss of consciousness was reported were summed for each participant. Number of criminal convictions ($M = .80$, $SD = 0.3$; 0 – 1.6) was totaled per participant upon juvenile

institutional file review, and included misdemeanor, felony, and parole violation convictions.

Total brain volume (BV) ($M = 1231.39$, $SD = 98.7$; $970.0 - 1526.2$) was quantified as the sum of gray matter and white matter volumes for each participant. Finally, clinical diagnoses were assessed via the KSADS (Kaufman et al., 1997). Diagnosis categories were as follows: anxiety disorders, depressive disorders, conduct disorder or oppositional defiant disorder (CD/ODD), attention-deficit/hyperactivity disorder (ADHD), and post-traumatic stress disorder (PTSD).

In addition to the aforementioned variables included here based on the methodology outlined in Cope et al. (2014), history of trauma, parental incarceration, parental separation, gang affiliation, and age at first arrest were investigated in the current study. Multiple studies have implicated childhood trauma in antisocial behavior in general (Baglivio, 2009; Baglivio et al., 2015; Fabian, 2010; Shader, 2003), and in homicide specifically (Darby et al., 1998; Fabian, 2010). Here, trauma was assessed via the Trauma Checklist 2.0 (Shold et al., under review), a modified version of the Trauma Checklist (Dargis et al., 2019). Total, Factor 1, and Factor 2 scores were included in analyses. Factor 1 assesses experience with physical abuse, emotional abuse, sexual abuse, and neglect/poverty, and Factor 2 assesses experience with community violence, traumatic loss, and observed trauma. Additionally, Fabian (2010) and Darby et al. (1998) detailed the importance of the inclusion of parental incarceration and parental separation in the study of homicide, demonstrating a significant positive relationship between parental incarceration and parental separation and homicide offending. Here, parental incarceration was obtained from the TCL 2.0, where it was determined whether or not participants had a parent that had been previously, or is currently, incarcerated. Parental separation was a binary variable defined by responses to both “parents divorced” and “parents never married” on the TCL2.0. (e.g., a “no” response to “parents divorced” and “yes” response to “parents never married” on the

TCL 2.0 would result in a “yes” response to “parental separation”). Shader (2003) identified gang affiliation as a risk factor for future antisocial behavior. Gang affiliation was defined here as a binary variable based on whether participants were involved in a gang, which was obtained from their institutional files. Finally, Baglivio (2009) and Piquero & Chung (2001) identified age of first arrest as a critical variable to consider when assessing recidivism risk, indicating that younger age of onset of offending was related to an increased likelihood of future offending. Age of first arrest ($M = 12.67$, $SD = 2.2$; 6 - 17) was identified from institutional files, and was recorded independent of conviction status for the charge assigned at the time of the arrest. Following the methodology of Cope et al. (2014), mean replacement was conducted for continuous variables that had missing values. Descriptive statistics for all variables are displayed in Table 1.

Regarding a priori regions of interest (ROIs) for VBM analyses, regions previously identified to be associated with psychopathic traits were included in analyses, including the amygdala, insula, parahippocampal gyrus, superior temporal pole, middle temporal pole, and OFC. These regions were identified using images from the Automated Anatomical Labeling atlas 3 (AAL3; Rolls et al, 2010) toolbox available in the Wake Forest University (WFU) Pick Atlas Toolbox (Maldjian et al., 2003; Maldjian et al., 2004) in SPM12. Figure 1 displays these ROIs. Additional details on the selection of ROIs are presented in the *Voxel-Based Morphometry* section.

Data Analysis

Independent Samples T-Test

To identify continuous variables on which the Future Homicide and No Homicide groups significantly differed, independent sample *t*-tests were conducted. Due to a priori hypotheses

regarding group differences on PLC:YV Total, Factor 1, and Factor 2 scores, *t*-tests for these three variables will be 1-tailed, and no corrections for multiple comparisons will be performed. Less evidence exists to support the relationship between the remaining variables (age at scan, SES, IQ, BIS-11 total, number of SUDs, ASI, number of TBIs, number of criminal convictions, total BV, TCL Total, TCL Factor 1, TCL Factor 2, and age of first arrest) and homicidal behavior. Therefore, 2-tailed *t*-tests will be performed to investigate these post hoc relationships. A Bonferroni correction was implemented to control for multiple comparisons (i.e., $.05/13$, or $p < .004$). Prior to analyses, SES and Number of Convictions were transformed to account for skewness (see Table 1).

Fisher's Exact Test

Fisher's Exact Tests were conducted to identify binary variables on which the Future Homicide and No Homicide groups significantly differed. A Bonferroni correction was implemented to control for multiple comparisons (i.e., $.05/8$, or $p = .006$). Fisher's Exact Tests were used here due to small frequencies in these binary variables (See Table 1 for frequencies). Specifically, Parental Incarceration, Parental Separation, Gang Affiliation, and all KSDADS diagnoses were included in these analyses.

Voxel-Based Morphometry

To determine group differences between Future Homicide and No Homicide groups in gray matter volumes, voxel-based morphometry (VBM) was used. VBM is a neuroimaging analysis technique that allows for the examination of volumetric group differences across the whole brain. Here, two-sample *t*-tests were performed on a voxel-by-voxel basis across the whole brain using the general linear model to evaluate differences in regional gray matter

volume between the Future Homicide ($n = 23$) and No Homicide ($n = 219$) groups. BV, number of SUDs, and PCL:YV total scores were included as covariates in these analyses, consistent with Cope et al. (2014).

Binarized masks of each of the a priori ROIs were obtained using AAL3 (Rolls et al., 2010). These binarized masks have a value of 1 in any voxel deemed to be within a specific region and a value of 0 in all other voxels. These binarized masks were used to statistically determine where in the VBM image each ROI was located and extract group differences in gray matter values for each of these ROIs.

Results

Independent Sample T-Tests

The Future Homicide group ($M = 25.7$, $SD = 4.5$) scored significantly higher than the No Homicide group ($M = 23.0$, $SD = 5.6$) on PCL:YV Total scores [$t(240) = 2.27$, $p = .012$]. The Future Homicide group ($M = 15.7$, $SD = 2.3$) also scored significantly higher than the No Homicide group ($M = 14.4$, $SD = 3.1$) on PCL:YV Factor 2 scores [$t(240) = 2.038$, $p = .022$]. Finally, the Future Homicide group ($M = 11.7$, $SD = 2.3$) was a bit younger than the No Homicide group ($M = 12.8$, $SD = 2.2$) at the time of their first arrest [$t(240) = -2.502$, $p = .013$]. However, this relationship between age of first arrest and homicide group did not survive after correcting for multiple comparisons (i.e., $.05/13$, or $p = .004$). There were no significant group differences between the Future Homicide and No Homicide Groups on the remaining psychological variables (see Table 2).

Fisher's Exact Tests

There were no significant group differences on any of the binary variables investigated. Results are displayed in Table 2.

Voxel-Based Morphometry

Whole-brain analyses controlling for BV, PCL:YV Total score, and number of SUDs are displayed in Figure 2, with the Future Homicide group exhibiting reduced GMV compared to the No Homicide group. These deficits are displayed in blue. Analyses in *a priori* ROIs (i.e., amygdala, insula, parahippocampal gyrus, superior temporal pole, middle temporal pole, and OFC) revealed significant group differences (FWE-corrected) in gray matter volume in the bilateral amygdala, left insula, bilateral parahippocampal gyrus, left superior temporal pole, left middle temporal pole, and left OFC. The remaining ROIs (i.e., right insula, right superior temporal pole, right middle temporal pole, and right OFC) did not yield significant group differences in gray matter volume. Coordinates and effect sizes for all ROIs are displayed in Table 3.

Discussion

The current study found that youth offenders who committed a homicide after release from a juvenile correctional facility (Future Homicide group) scored significantly higher on psychosocial variables collected at baseline, including PCL:YV Total and Factor 2, compared to juvenile offenders who did not commit a homicide after release (No Homicide group). Additionally, boys included in the Future Homicide group had significantly less gray matter volume than boys in the No Homicide group in the right and left amygdala, left insula, right and left parahippocampal gyrus, left superior temporal gyrus, left middle temporal pole, and left OFC. Therefore, the current results suggest that psychopathy scores and reduced GMV within paralimbic regions may serve as potential variables to help better delineate which youth may be associated with severe, future antisocial behavior, including homicide.

The current study identified gray matter abnormalities (i.e., reduced volume) in a number of regions previously implicated in individuals scoring high on psychopathic traits. Consistent

with the findings from Cope et al. (2014), we observed that youth who committed a future homicide as adults were associated with reduced GMV collected at baseline MRI scans in several paralimbic regions, including the parahippocampal gyrus, superior temporal pole, and middle temporal pole in youth compared to youth offenders who did not commit homicide. These findings suggest a set of neural abnormalities present in adolescence associated with homicide, regardless of when it has occurred (i.e., before or after their baseline MRI scan). Previous research has demonstrated that regions within the temporal pole are involved in successful affective processing (Jimura et al., 2010; Olson et al., 2011). Regarding offending, research has found temporal pole abnormalities contribute to socioemotional processing, resulting in empathy- and morality-related deficits (Bertsch et al., 2013; Gregory et al., 2012). Additionally, the parahippocampal gyrus has been implicated in behavioral inhibition in homicide offenders (Yang et al., 2010). While these ROIs were selected for the current study because of their association with psychopathic traits, psychopathy scores were controlled for in structural MRI analyses for both the present study and those performed by Cope et al. (2014). Such results indicate that incarcerated youth who committed previous and future homicide may be associated with unique neural abnormalities, even when controlling for important covariates such as psychopathic traits. Taken together, these results indicate that neural deficits in brain regions contributing to successful affective and socioemotional processing may be associated with homicidal behavior, irrespective of psychopathic traits.

A number of gray matter deficits were observed in the current study that were not found by Cope et al. (2014) (i.e., deficits in the bilateral amygdala, left insula, and left OFC), indicating their potential utility in identifying incarcerated youth at an increased risk of committing homicide in the future. Understanding the functions of these regions may help to further

elucidate the specific behavioral deficits potentially associated with future homicidal behavior in adulthood. Previous research has implicated the amygdala in affective processing (Murray et al., 2014; Zald, 2003). The insula has previously been linked with socio-emotional and sensorimotor processing (Kurth et al., 2010; Uddin et al., 2017). Specific to psychopathy and antisocial behavior, abnormalities within the insula have been associated with increased emotional detachment and immorality (Johansen et al., 2020). The OFC has been considered the “apex of the social brain” (Mitchell & Beech, 2011) and is one of the vital neural regions for the evaluation of emotional stimuli and affective signals, regulation of emotion necessary for appropriate empathic responses (Decety, 2011), and reappraisal of negative emotional stimuli (Golkar et al., 2012). Previous studies have found reduced OFC activity during Theory of Mind tasks (Shamay-Tsoory et al., 2010) in offenders, suggesting a diminished ability of offenders with deficits in the OFC to accurately understand the emotional state of others. Taken together, these findings suggest baseline deficits in affective processing among incarcerated boys who later commit a homicide in adulthood. While there were no significant differences between groups on PCL:YV Factor 1 scores, which in part are designed to capture these affective deficits, these findings potentially indicate the incremental utility of neuroimaging data over clinical data, as even interview-based data may still be susceptible to human error in scoring and poor introspection on the part of interviewees.

In addition to significant neural deficits, the present study also found group differences regarding psychopathy scores between participants who committed a future homicide and control participants without homicide convictions after release, such that boys who committed a future homicide scored higher on PCL:YV Total and Factor 2 (antisocial/developmental traits) than the boys who did not commit a homicide. These results align with prior findings (Cope et al., 2014).

In both studies, participants who committed a homicide (both previously and in the future) scored higher on psychopathic traits compared to control participants who did not ever commit a homicide. These findings also support previous literature implicating youth psychopathic traits with an increased propensity towards future antisocial behavior (Lynam et al., 2009; Schmidt et al., 2006; Stockdale et al., 2010). Similar patterns of increased psychopathy scores in homicidal offenders have also been found in adults (Fox & DeLisi, 2019).

The neural and clinical results from the current study are the first to identify brain abnormalities and personality traits in juvenile offenders that are potentially associated with future homicidal behavior as adults, indicating the need for more longitudinal research on the developmental trajectory of antisocial behavior. Moffitt (1993) identified lifecourse-persistent offenders as a subsample of offenders who continue to commit antisocial behavior beyond early adulthood, and studies have found this subtype of offender to commit the majority of major crimes, including homicide (Vaughn et al., 201; Wolfgang et al., 1972). Identifying the early behavioral patterns of these offenders offers a beginning step in understanding what sets this unique group of youth apart from their adolescent-limited offending counterparts. Further, obtaining and analyzing neuroimaging variables early in the cycle of crime may provide an enhanced opportunity to understand the underpinnings of these unique youths' antisocial behavior (Radeljack et al., 2020). Identifying these youth while still in adolescence may provide an opportunity to engage in treatment while their brains, and therefore behavior, are still malleable.

During adolescence, youth typically demonstrate increased reward sensitivity, reduced responsiveness to aversive stimuli, and inhibition control that is often overridden by emotional or stressful events (Spear, 2013). Adolescence is also a period of marked neurodevelopment, during

which experiences (i.e., behavioral interventions) may have a particularly large influence on how these brain changes occur (Spear, 2013). Specifically, the amygdala and hippocampus (Spear, 2013; Walker, 2002), various frontal regions (Spear, 2013; Walker, 2002), and the temporal cortex (Walker, 2002) have been identified as particularly plastic regions during adolescence, and have also been identified here to be related to future homicidal behavior. Thus, longitudinal studies that begin during adolescence and continue well into adulthood are necessary to enhance our ability to identify lifecourse persistent offenders. Once we are readily able to predict which juvenile offenders will fall into this lifecourse-persistent pattern of offending, studies can further be conducted investigating the efficacy of behavioral treatment provided during this period of neurodevelopment on future offending. The efficacy of such treatments has previously been demonstrated by Caldwell et al. (2006). In this study, youth offenders who were deemed to be the most high-risk for future antisocial behavior received a specialized treatment while held in a correctional facility. When compared to another sample of high-risk youth offenders who were incarcerated in a typical facility, this group of boys who received treatment demonstrated longer periods to re-offend. They also committed less serious offenses as compared to the boys who did not receive treatment. This study thus provides evidence to support the efficacy of early behavioral interventions for those youth offenders identified as very high-risk for future antisocial behavior, a process that may be possible using the information gained here.

Limitations

In the current study, neural regions implicated in individuals scoring high on psychopathic traits were chosen as a priori ROIs due to the significant relationship between psychopathy and homicide (Fox & DeLisi, 2019) and the utility of psychopathic traits and neural regions in classifying juvenile offenders who previously committed a homicide (Cope et al., 2014).

However, this selection of ROIs limits our interpretation of the results, as the investigation into the neural deficits specific to future homicide is not exhaustive. Results from Figure 1 demonstrate potential differences in GMV between homicide groups extending beyond our a priori ROIs, such as regions of the cerebellum. Therefore, future studies should explore these additional regions for potential significant differences beyond those studied here. Additionally, the sample included only incarcerated boys due to a limited sample size of incarcerated girls who committed a homicide after release. Gender has previously been identified as the best predictor of future homicide (Baglivio, 2009), indicating sex differences in this type of violent offending. Future studies should include female participants to better elucidate the underlying variables that cause these sex differences in homicide offending. Finally, the analyses performed in the current study only provide insight into significant group differences, and therefore do not provide information on any potential causal relationships between psychosocial and neuroanatomical variables and future homicide offending.

Future Directions

Future studies should investigate the predictive utility of the variables included here. Specifically, entering variables identified in the current study into a machine learning classifier may identify variables capable of predicting which offenders will commit a homicide after release from a correctional facility. This technique has the potential to increase the accuracy of tools currently being used by the criminal justice system to assess risk level for future offending (Rus, 2022; Tortora, 2022). Additionally, evidence suggests offenders who commit different homicide offense types (e.g., general, sexual, sadistic, serial) may have difference psychopathic trait profiles (Fox & DeLisi, 2019). Future studies should therefore aim to differentiate between types of homicide offenders. Greater specificity of offense type will lead to a greater

understanding of a complex form of violent interpersonal antisocial behavior, which may then result in the creation of enhanced intervention or treatment programs tailored to the nuances of human behavior. These more personalized intervention programs should be created with the overall goal of a reduction in recidivism.

Conclusion

This study found that incarcerated boys who would go on to commit a homicide after release from a juvenile correctional facility scored higher on psychopathic traits and had significantly less gray matter within paralimbic brain regions, compared to incarcerated boys who did not commit a homicide after release. This study is the first longitudinal study of its kind to investigate the clinical, environmental, and neural abnormalities in youth offenders who will commit a future homicide up to 14 years after release from a juvenile correctional facility. These results shed light on the underlying traits and neural abnormalities that may put juvenile offenders at heightened risk for future homicidal behavior. Identifying these abnormalities early in development may prove crucial to the reduction of future antisocial behavior, as such high-risk adolescents are amenable to treatment (Caldwell et al., 2006).

References

- Alper, M., Durose, M., Markman, J. (2018). *2018 Update on Prisoner Recidivism: A 9-year Follow-up Period (2005-2014)*. U.S. Department of Justice.
- American Civil Liberties Union. (2022). *Juvenile Justice*. <https://www.aclu.org/issues/juvenile-justice>
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders: DSM-IV-TR*. American Psychiatric Association.
- Ashburner, J., & Friston, K. J. (2005). Unified segmentation. *Neuroimage*, 26(3), 839-851.
- Baglivio, M. T. (2009). The assessment of risk to recidivate among a juvenile offending population. *Journal of Criminal Justice*, 37(6), 596–607. <https://doi.org/10.1016/j.jcrimjus.2009.09.008>
- Baglivio, M. T., Wolff, K. T., Piquero, A. R., & Epps, N. (2015). The Relationship between Adverse Childhood Experiences (ACE) and Juvenile Offending Trajectories in a Juvenile Offender Sample. *Journal of Criminal Justice*, 43(3), 229–241. <https://doi.org/10.1016/j.jcrimjus.2015.04.012>
- Bertsch, K., Grothe, M., Prehn, K., Vohs, K., Berger, C., Hauenstein, K., Keiper, P., Domes, G., Teipel, S., & Herpertz, S. C. (2013). Brain volumes differ between diagnostic groups of violent criminal offenders. *European Archives of Psychiatry and Clinical Neuroscience*, 263(7), 593–606. <https://doi.org/10.1007/s00406-013-0391-6>
- Caldwell, M., Skeem, J., Salekin, R., & Van Rybroek, G. (2006). Treatment Response of Adolescent Offenders With Psychopathy Features: A 2-Year Follow-Up. *Criminal Justice and Behavior*, 33(5), 571–596. <https://doi.org/10.1177/0093854806288176>

- Cope, L. M., Ermer, E., Gaudet, L. M., Steele, V. R., Eckhardt, A. L., Arbabshirani, M. R., Caldwell, M. F., Calhoun, V. D., & Kiehl, K. A. (2014). Abnormal brain structure in youth who commit homicide. *NeuroImage: Clinical*, 4, 800–807. <https://doi.org/10.1016/j.nicl.2014.05.002>
- Darby, P. J., Allan, W. D., Kashani, J. H., Hartke, L., & Reid, J. C. (n.d.). *Analysis of 112 Juveniles Who Committed Homicide: Characteristics and a Closer Look at Family Abuse*.
- Dargis, M., Sitney, M., Caldwell, B., Caldwell, M., Edwards, B. G., Harenski, C., ... & Kiehl, K. A. (2019). Development of an expert-rater assessment of trauma history in a high-risk youth forensic sample. *Psychological trauma: theory, research, practice, and policy*, 11(7), 713.
- Decety, J. (2011). Dissecting the Neural Mechanisms Mediating Empathy. *Emotion Review*, 3(1), 92–108. <https://doi.org/10.1177/1754073910374662>
- Deming, P., & Koenigs, M. (2020). Functional neural correlates of psychopathy: A meta-analysis of MRI data. *Translational Psychiatry*, 10(1), 133. <https://doi.org/10.1038/s41398-020-0816-8>
- Ermer, E., Cope, L.M., Nyalakanti, P.K., Calhoun, V.D., Kiehl, K.A. (2012). Aberrant paralimbic gray matter in criminal psychopathy. *Journal of Abnormal Psychology*, 121, 649–58. <http://dx.doi.org/10.1037/a0026371>, 22149911.
- Essau, C.A., Sasagawa, S., Frick, P.J. (2006). Callous-unemotional traits in a community sample of adolescents. *Assessment*, 13, 454–69. <http://dx.doi.org/10.1177/1073191106287354>, 17050915.
- Fabian, J. M. (2010). Neuropsychological and neurological correlates in violent and homicidal offenders: A legal and neuroscience perspective. *Aggression and Violent Behavior*, 15(3), 209–223. <https://doi.org/10.1016/j.avb.2009.12.004>

- Fink, B. C., Tant, A. S., Tremba, K., & Kiehl, K. A. (2012). Assessment of Psychopathic Traits in an Incarcerated Adolescent Sample: A Methodological Comparison. *Journal of Abnormal Child Psychology*, 40(6), 971–986. <https://doi.org/10.1007/s10802-012-9614-y>
- Forth, A. E., Kosson, D. S., & Hare, R. D. (2003). *The hare psychopathy checklist: youth version*. North Tonawanda, NY: Multi-Health Systems.
- Fox, B., & DeLisi, M. (2019). Psychopathic killers: A meta-analytic review of the psychopathy-homicide nexus. *Aggression and Violent Behavior*, 44, 67–79. <https://doi.org/10.1016/j.avb.2018.11.005>
- Frick, P. J. (2009). Extending the Construct of Psychopathy to Youth: Implications for Understanding, Diagnosing, and Treating Antisocial Children and Adolescents. *The Canadian Journal of Psychiatry*, 54(12), 803–812. <https://doi.org/10.1177/070674370905401203>
- Gregory, S. (2012). The Antisocial Brain: Psychopathy Matters: A Structural MRI Investigation of Antisocial Male Violent Offenders. *Archives of General Psychiatry*, 69(9), 962. <https://doi.org/10.1001/archgenpsychiatry.2012.222>
- Golkar, A., Lonsdorf, T. B., Olsson, A., Lindstrom, K. M., Berrebi, J., Fransson, P., Schalling, M., Ingvar, M., & Öhman, A. (2012). Distinct Contributions of the Dorsolateral Prefrontal and Orbitofrontal Cortex during Emotion Regulation. *PLoS ONE*, 7(11), e48107. <https://doi.org/10.1371/journal.pone.0048107>
- Hare, R. D. (2003). *The hare psychopathy checklist revised* (2nd ed.). Multi-Health Systems

- Jimura, K., Konishi, S., Asari, T., & Miyashita, Y. (2010). Temporal pole activity during understanding other persons' mental states correlates with neuroticism trait. *Brain Research*, 1328, 104–112. <https://doi.org/10.1016/j.brainres.2010.03.016>
- Johanson, M., Vaurio, O., Tiihonen, J., & Lähteenvuo, M. (2020). A Systematic Literature Review of Neuroimaging of Psychopathic Traits. *Frontiers in Psychiatry*, 10, 1027. <https://doi.org/10.3389/fpsyt.2019.01027>
- Justice Center—The Council of State Governments. (2021). *Reducing Juvenile Recidivism*. <https://csgjusticecenter.org/publications/reducing-juvenile-recidivism/>
- Kaufman, J., Birmaher, B., & Brent, D. (1997). Schedule for affective disorders and schizophrenia for school-aged children: Present and lifetime version (K-SADS-PL): Initial reliability and validity data. *Journal of American Academy of Child & Adolescent Psychiatry*, 37(7), 980–988.
- Kiehl, K. A. (2006). A cognitive neuroscience perspective on psychopathy: Evidence for paralimbic system dysfunction. *Psychiatry Research*, 142(2–3), 107–128. <https://doi.org/10.1016/j.psychres.2005.09.013>
- King, N.S., Crawford, S., Wenden, F.J., Moss, N.E., Wade, D.T. (1995). The Rivermead Post Concussion Symptoms Questionnaire: a measure of symptoms commonly experienced after head injury and its reliability. *Journal of Neurology*, 242, 587–92. <http://dx.doi.org/10.1007/BF00868811>, 8551320.
- Khachatryan, N., Heide, K. M., Rad, J., & Hummel, E. V. (2016). Post-incarceration Recidivism of Lone versus Group Juvenile Homicide Offenders: Lone versus group homicide recidivism. *Behavioral Sciences & the Law*, 34(6), 709–725. <https://doi.org/10.1002/bsl.2257>

- Lynam, D. R., Miller, D. J., Vachon, D., Loeber, R., & Stouthamer-Loeber, M. (2009). Psychopathy in Adolescence Predicts Official Reports of Offending in Adulthood. *Youth Violence and Juvenile Justice*, 7(3), 189–207. <https://doi.org/10.1177/1541204009333797>
- Maldjian JA, Laurienti PJ, & Burdette JH (2004). Precentral gyrus discrepancy in electronic versions of the Talairach atlas. *NeuroImage*, 21(1), 450–455.
- Maldjian JA, Laurienti PJ, Kraft RA, Burdette JH. An automated method for neuroanatomic and cytoarchitectonic atlas-based interrogation of fMRI data sets. *Neuroimage*. 2003;19:1233–1239.
- Maurer, J. M., Steele, V. R., Fink, B. C., Vincent, G. M., Calhoun, V. D., & Kiehl, K. A. (2018). Investigating error-related processing in incarcerated adolescents with self-report psychopathy measures. *Biological Psychology*, 132, 96–105. <https://doi.org/10.1016/j.biopsycho.2017.11.009>
- McLellan, A.T., Kushner, H., Metzger, D., Peters, R., Smith, I., Grissom, G., et al. 1992. The fifth edition of the Addiction Severity Index. *Journal of Substance Abuse Treatment* 9, 199–213. [http://dx.doi.org/10.1016/0740-5472\(92\)90062-S](http://dx.doi.org/10.1016/0740-5472(92)90062-S), 1334156.
- Mitchell, I. J., & Beech, A. R. (2011). Towards a neurobiological model of offending. *Clinical Psychology Review*, 31(5), 872–882. <https://doi.org/10.1016/j.cpr.2011.04.001>
- Moffitt, T. E. (1993). Adolescence-Limited and Life-Course-Persistent Antisocial Behavior: A Developmental Taxonomy. *Psychological review*, 100(4), 674.
- Murray, R. J., Brosch, T., & Sander, D. (2014). The functional profile of the human amygdala in affective processing: Insights from intracranial recordings. *Cortex*, 60, 10–33. <https://doi.org/10.1016/j.cortex.2014.06.010>

National Institute of Justice. (2014). *From Youth Justice Involvement to Youth Adult Offending*.

<https://nij.ojp.gov/topics/articles/youth-justice-involvement-young-adult-offending>.

Olson, I. R., Plotzker, A., & Ezzyat, Y. (2007). The Enigmatic temporal pole: A review of findings on social and emotional processing. *Brain*, *130*(7), 1718–1731.

<https://doi.org/10.1093/brain/awm052>

Ormachea, P.A., Haarsma, G., Davenport, S., Eagleman, D.M. (2015). A new criminal records database for large-scale analysis of policy and behavior. *The Journal of Science & Law*, *1*(1), 1-7.

Patton, J.H., Stanford, M.S., Barratt, E.S., (1995). Factor structure of the Barratt Impulsiveness Scale.

Journal of Clinical Psychology, *51*, 768–74. [http://dx.doi.org/10.1002/1097-](http://dx.doi.org/10.1002/1097-4679(199511)51:6<768::AID-JCLP2270510607>3.0.CO;2-1)

[4679\(199511\)51:6<768::AID-JCLP2270510607>3.0.CO;2-1](http://dx.doi.org/10.1002/1097-4679(199511)51:6<768::AID-JCLP2270510607>3.0.CO;2-1), 8778124.

Piquero, A. R., & Chung, H. L. (2001). On the relationships between gender, early onset, and the seriousness of offending. *Journal of Criminal Justice*, *29*(3), 189–206.

[https://doi.org/10.1016/S0047-2352\(01\)00084-8](https://doi.org/10.1016/S0047-2352(01)00084-8)

Radeljak, S., & Dadi, E. (2010). Neuroimaging Techniques in Modern Forensic Psychiatry.

Neuroimaging in Psychiatry.

Rus, A. (2022). Implications of neuroscience on criminal law. *Supremo Amicus*, *30*, 538-544.

Ryan, J.J., Lopez, S.J., Werth, T.R., (1999). Development and preliminary validation of a Satz–Mogel short form of the WAIS-III in a sample of persons with substance abuse disorders. *International Journal of Neuroscience*, *98*, 131–40. <http://dx.doi.org/10.3109/00207459908994796>, 10395365.

- Sajous-Turner, A., Anderson, N. E., Widdows, M., Nyalakanti, P., Harenski, K., Harenski, C., Koenigs, M., Decety, J., & Kiehl, K. A. (2020). Aberrant brain gray matter in murderers. *Brain Imaging and Behavior, 14*(5), 2050–2061. <https://doi.org/10.1007/s11682-019-00155-y>
- Sattler, J. M., & Dumont, R. (2004). *Assessment of children: WISC-IV and WPPSI-III supplement*. Jerome M. Sattler, Publisher.
- Schmidt, F., McKinnon, L., Chattha, H. K., & Brownlee, K. (2006). Concurrent and predictive validity of the Psychopathy Checklist: Youth version across gender and ethnicity. *Psychological Assessment, 18*(4), 393–401. <https://doi.org/10.1037/1040-3590.18.4.393>
- Shader, M. (2003). Risk factors for delinquency: An overview. *U.S. Department of Justice*.
- Shamay-Tsoory, S. G., Harari, H., Aharon-Peretz, J., & Levkovitz, Y. (2010). The role of the orbitofrontal cortex in affective theory of mind deficits in criminal offenders with psychopathic tendencies. *Cortex, 46*(5), 668–677. <https://doi.org/10.1016/j.cortex.2009.04.008>
- Shold JN, Maurer JM, Reynolds BL, Gullapalli AR, Allen CH, Edwards BG, Anderson NE, Harenski CL, Neumann CS, & Kiehl KA (under review). Psychometric properties of the Trauma Checklist 2.0 and its predictive utility of felony re-offending among high-risk juvenile offenders. *Child & Adolescent Psychiatry and Mental Health*.
- Spear, L. P. (2013). Adolescent Neurodevelopment. *Journal of Adolescent Health, 52*(2), S7–S13. <https://doi.org/10.1016/j.jadohealth.2012.05.006>
- Stockdale, K. C., Olver, M. E., & Wong, S. C. P. (2010). The Psychopathy Checklist: Youth Version and adolescent and adult recidivism: Considerations with respect to gender, ethnicity, and age. *Psychological Assessment, 22*(4), 768–781. <https://doi.org/10.1037/a0020044>

- Thijssen, S., & Kiehl, K. A. (2017). Functional connectivity in incarcerated male adolescents with psychopathic traits. *Psychiatry Research: Neuroimaging*, *265*, 35–44.
<https://doi.org/10.1016/j.psychresns.2017.05.005>
- Tortora, L., Meynen, G., Bijlsma, J., Tronci, E., & Ferracuti, S. (2020). Neuroprediction and A.I. in Forensic Psychiatry and Criminal Justice: A Neurolaw Perspective. *Frontiers in Psychology*, *11*.
<https://www.frontiersin.org/articles/10.3389/fpsyg.2020.00220>
- Uddin, L. Q., Nomi, J. S., Hébert-Seropian, B., Ghaziri, J., & Boucher, O. (2017). Structure and Function of the Human Insula: *Journal of Clinical Neurophysiology*, *34*(4), 300–306.
<https://doi.org/10.1097/WNP.0000000000000377>
- Umbach, R., Berryessa, C. M., & Raine, A. (2015). Brain imaging research on psychopathy: Implications for punishment, prediction, and treatment in youth and adults. *Journal of Criminal Justice*, *43*(4), 295–306. <https://doi.org/10.1016/j.jcrimjus.2015.04.003>
- Vaughn, M. G., Salas-Wright, C. P., DeLisi, M., & Maynard, B. R. (2014). Violence and Externalizing Behavior Among Youth in the United States: Is There a Severe 5%? *Youth Violence and Juvenile Justice*, *12*(1), 3–21. <https://doi.org/10.1177/1541204013478973>
- Vincent, G. M., Odgers, C. L., McCormick, A. V., & Corrado, R. R. (2008). The PCL: YV and recidivism in male and female juveniles: A follow-up into young adulthood. *International Journal of Law and Psychiatry*, *31*(3), 287–296. <https://doi.org/10.1016/j.ijlp.2008.04.012>
- Walker, E. F. (2002). Adolescent Neurodevelopment and Psychopathology. *Current Directions in Psychological Science*, *11*(1), 24–28. <https://doi.org/10.1111/1467-8721.00161>

- Wechsler, D. (2003). *Wechsler Intelligence Scale for Children—Fourth Edition: Technical and Interpretive Manual*. Psychological Corporation.
- Wechsler, D. (1997). *WAIS-III: Wechsler Adult Intelligence Scale*. Psychological Corporation.
- Wolfgang, M. E., Figlio, R. M., & Sellin, T. (1987). *Delinquency in a birth cohort*. University of Chicago Press.
- Yang, Y., Raine, A., Han, C.-B., Schug, R. A., Toga, A. W., & Narr, K. L. (2010). Reduced hippocampal and parahippocampal volumes in murderers with schizophrenia. *Psychiatry Research: Neuroimaging*, 182(1), 9–13. <https://doi.org/10.1016/j.psychresns.2009.10.013>
- Zald, D. H. (2003). The human amygdala and the emotional evaluation of sensory stimuli. *Brain Research Reviews*, 41(1), 88–123. [https://doi.org/10.1016/S0165-0173\(02\)00248-5](https://doi.org/10.1016/S0165-0173(02)00248-5)

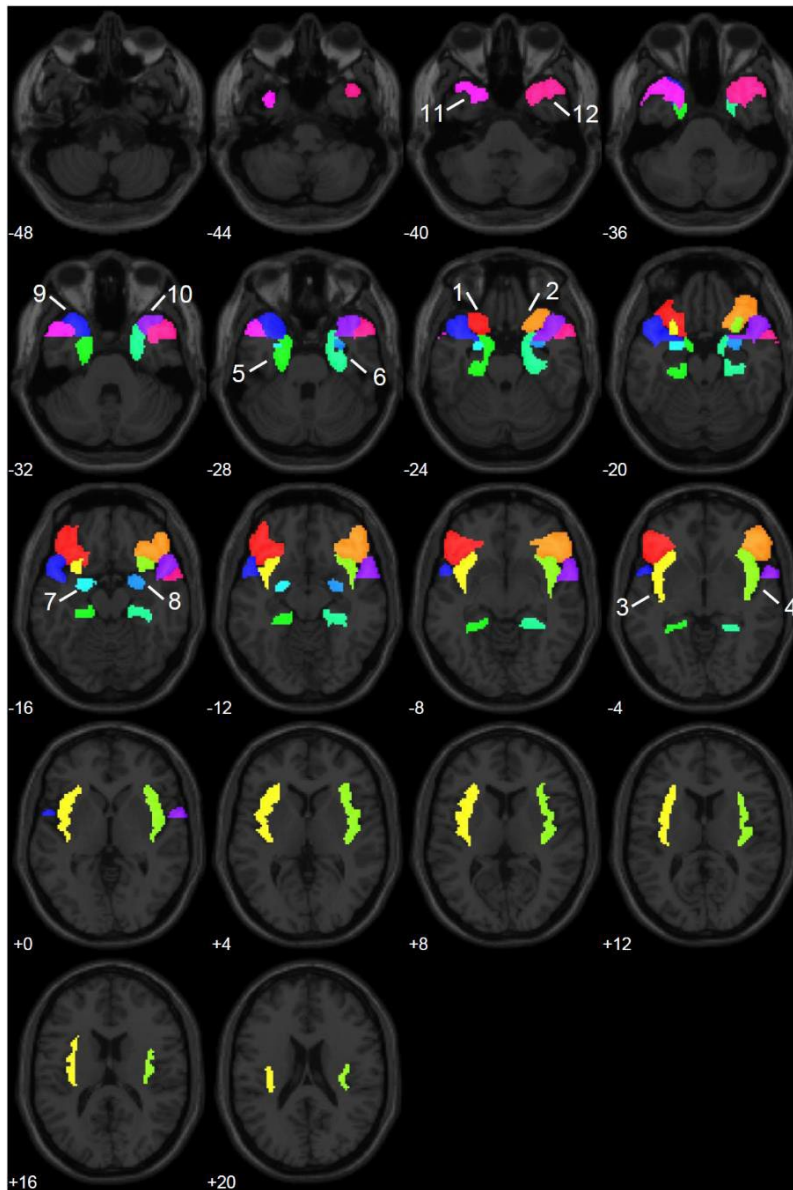


Figure 1. Visual representation of all ROIs included in analyses. 1 = Left OFC. 2 = Right OFC. 3 = Left Insula. 4 = Right Insula. 5 = Left Parahippocampal Pole. 6 = Right Parahippocampal Pole. 7 = Left Amygdala. 8 = Right Amygdala. 9 = Left Superior Temporal Pole. 10 = Right Superior Temporal Pole. 11 = Left Middle Temporal Pole. 12 = Right Middle Temporal Pole.

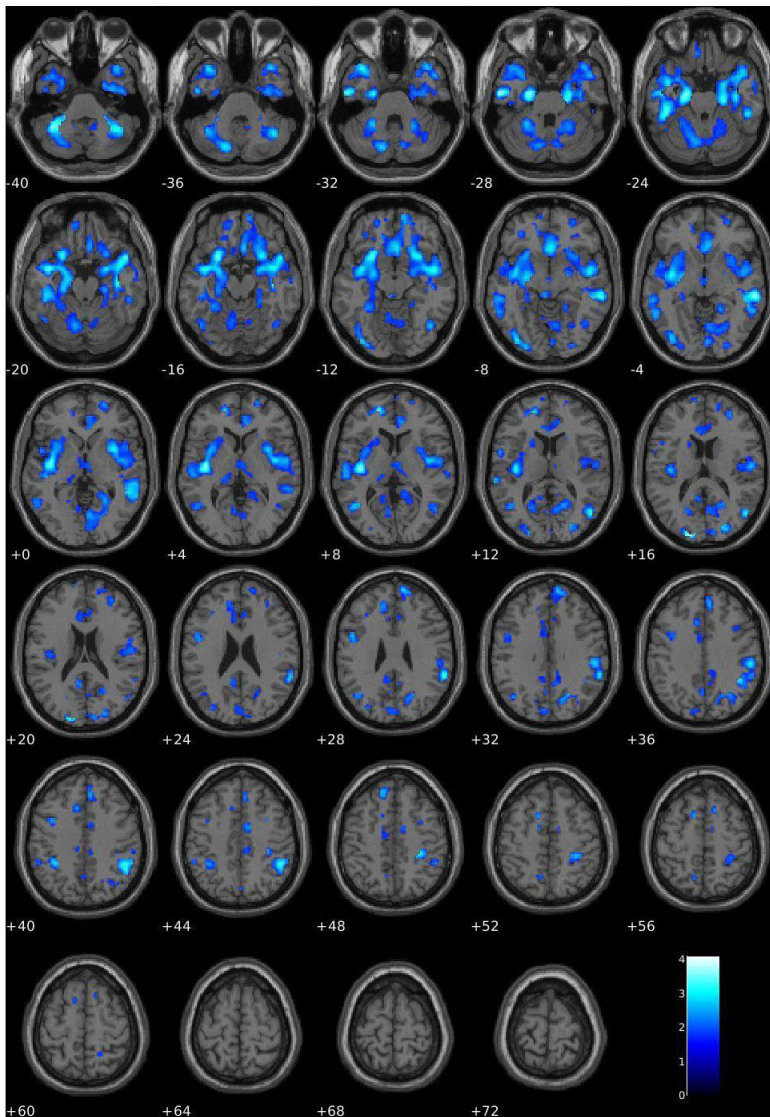


Figure 2. VBM results showing significant group differences in gray matter volume between the Future Homicide ($n = 23$) and No Homicide ($n = 219$) groups. Results here are controlling for number of substance dependencies, PCL:YV scores, and BV (gray matter + white matter). Blue scale indicates t -values for regions in which the Future Homicide group had less gray matter compared to the No Homicide group. Map is thresholded at $t=1.65$ voxel-height level to illustrate all effects. Results from a priori regions-of-interest are presented in Table 3.

Table 1.*Descriptive Statistics and Frequencies.*

Variable	Future Homicide			No Homicide		
	<i>M(SD)</i>	Skewness	Kurtosis	<i>M(SD)</i>	Skewness	Kurtosis
Age	17.18 (1.44)	-.30	-.94	17.63 (1.08)	-.96	.82
SES	4.62 (.07)	-.02	-1.2	4.62 (.07)	.53	3.90
IQ	89.59 (11.29)	-.11	1.24	92.76 (10.13)	.58	.92
PCL:YV						
Total	25.73 (4.49)	-.32	-.31	23.01 (5.56)	-.40	-.02
Factor 1	7.40 (2.16)	-.72	-1.14	6.40 (2.83)	.48	-.21
Factor 2	15.73 (2.32)	-.28	-.31	14.40 (3.06)	-1.20	2.07
BIS-11	62.18 (20.54)	-1.73	3.43	66.56 (13.84)	-1.37	3.28
Number of SUD	1.73 (1.03)	-.25	.14	2.25 (1.48)	.56	.14
ASI	5.84 (3.05)	-.253	1.525	6.22 (2.96)	-.247	.615
Number of TBIs	.77 (.86)	2.54	8.96	.69 (.75)	1.72	3.16
Number of Convictions	.91 (.28)	.04	.37	.78 (.32)	-.24	.51
BV	1217.11 (78.09)	.02	-.19	1232.90 (100.65)	.04	.03
TCL						
Total	8.07 (2.58)	-1.27	3.43	7.68 (2.39)	.04	-.33
Factor 1	2.83 (1.69)	0	-.15	2.66 (1.88)	.55	-.34
Factor 2	5.22 (1.30)	-3.19	12.27	5.02 (1.17)	-1.20	.49
Age of First Arrest	11.59 (2.29)	-.52	-.06	12.78 (2.15)	-.20	-.49
Parental Incarceration	10 (55.6%)			84 (50.9%)		

Parental Separation	11 (68.8%)	136 (80.5%)
Gang Affiliation	13 (59.1%)	128 (64.3%)
KSADS Diagnoses		
Anxiety	0 (0%)	6 (3.3%)
Depression	3 (13%)	20 (9.1%)
PTSD	0 (0%)	11 (6.1%)
ADHD	5 (26.3%)	19 (10.7%)
CD/ODD	19 (100%)	170 (94.9%)

Note. Numbers represent means with standard deviations in parentheses, skewness, and kurtosis values for ordinal variables, or counts and percentages for binary variables. SES = socioeconomic status; IQ = Intelligence Quotient (Wechsler, 1997; Wechsler, 2003); PCL:YV = Psychopathy Checklist: Youth Version (Forth et al., 2003); BIS-11 = total score from Barratt Impulsiveness Score (Patten et al., 1995); Number of SUD = Number of substance use dependencies calculated by summing number of substances for which the participant met a diagnosis of lifetime dependence, via the Kiddie Schedule for Affective Disorders and Schizophrenia (KSADS; Kaufman et al., 1997); ASI = Addiction Severity Index (McLellan et al., 1992); Number of TBIs = Number of traumatic brain injuries in which a loss of consciousness occurred, as assessed via the Rivermead Post-Concussion Symptoms Questionnaire (King et al., 1995); BV = Total brain volume calculated as a sum of gray matter and white matter volumes; TCL = Trauma Checklist (Shold et al., under review); PTSD = Post-Traumatic Stress Disorder; ADHD = Attention-Deficit/Hyperactivity Disorder; CD/ODD = Conduct Disorder/Oppositional Defiant Disorder.

Table 2.*Independent Sample T-Test and Fisher's Exact Test Results*

Variable	<i>t</i>	df	<i>p</i>
PCL:YV			
Total	2.271	240	.012*
Factor 1	1.648	240	.051
Factor 2	2.038	240	.022*
Age	-1.830	240	.069
SES	0.171	240	.865
IQ	-1.410	240	.160
BIS-11	-1.373	240	.171
Number of SUD	-1.621	240	.106
ASI	-.658	240	.511
Number of TBIs	0.454	240	.650
TCL			
Total	0.722	240	.471
Factor 1	0.391	240	.696
Factor 2	0.771	240	.441
Number of Convictions	1.891	240	.060
BV	-0.729	240	.467
Age of First Arrest	-2.502	240	.013
Parental Separation			.328
Parental Incarceration			.806
Gang Affiliation			.645
KSADS Diagnoses			
Anxiety			1.00
Depression			.709
PTSD			.605
ADHD			.062
CD/ODD			1.00

Note. Independent Sample T-Tests were performed on continuous variables, while Fisher's Exact Tests were performed on binary variables; * $p < .05$ for a priori variables of interest. No results survive Bonferroni multiple comparison correction (i.e., $.05/13$, or $p = .004$ for t-tests; $.05/8$, or $p = .006$ for Fisher's Exact tests).

Table 3.

Neuroanatomical differences between Future Homicide (n = 23) and No Homicide (n = 219) groups.

Region	Hemisphere	x	y	z	t	p-FWE	p-uncorrected
Amygdala	R	24	-2	22	2.9	.045*	.002
Amygdala	L	-24	-3	-16	2.88	.046*	.002
Insula	R	28	15	-14	3.39	.064	<.001
Insula	L	-39	-15	6	3.98	.011*	<.001
Parahippocampal Gyrus	R	22	-18	-27	3.61	.024*	<.001
Parahippocampal Gyrus	L	-21	-14	-27	3.88	.009*	<.001
Superior Temporal Pole	R	48	12	-20	3.33	0.58	<.001
Superior Temporal Pole	L	-42	0	-20	3.41	.045*	<.001
Middle Temporal Pole	R	38	16	-39	2.88	.159	.002
Middle Temporal Pole	L	-36	16	-33	3.43	.028*	<.001
Orbitofrontal Cortex	R	24	15	-18	2.68	.294	.004
Orbitofrontal Cortex	L	-24	15	-16	3.47	.043*	<.001

Note. Group differences in gray matter volumes between Future Homicide and No Homicide groups. ROIs were defined by automated anatomical labels (AAL3) in the Wake Forest University PickAtlas toolbox in SPM12; p-FWE = p-value corrected for family-wise error rate; p-uncorrected = raw p-value without any forms of corrections; * $p < .05$.