

**Capitalizing on Neuroplasticity: A Systematic Review of Cognitive Behavioral Interventions and the Role of Prefrontal Cortex Development in Facilitating Desistance from Violent Offending Among Late Adolescents and Emerging Adults (Ages 16–25)**

Ahmed F. Alanazi

Department of Social Studies, College of Arts, King Faisal University, Saudi Arabia

DOI: 10.46609/IJSSER.2026.v11i03.024 URL: <https://doi.org/10.46609/IJSSER.2026.v11i03.024>

Received: 15 February 2026 / Accepted: 20 March 2026 / Published: 31 March 2026

**ABSTRACT**

**Background:** Late adolescence and emerging adulthood (ages 16–25) represent a critical period of continued prefrontal cortex (PFC) development, marked by significant growth in neural structures underpinning impulse control, risk assessment, and executive function. This neurodevelopmental window coincides with peak rates of violent offending. Cognitive Behavioral Interventions (CBT) have demonstrated efficacy in reducing recidivism, yet the mechanisms driving their success in this specific age group remain underexplored.

**Objective:** This systematic review synthesizes existing literature to address one primary research question: To what extent does the developmental trajectory of the prefrontal cortex during late adolescence and emerging adulthood moderate or mediate the effectiveness of Cognitive Behavioral Interventions in facilitating desistance from violent offending?

**Methods:** A systematic literature search was conducted across PsycINFO, PubMed, Scopus, and Web of Science for peer-reviewed studies published between 2000 and 2026. Studies were included if they focused on violent offending, utilized a CBT framework, sampled participants aged 16–25, and incorporated measures of neurocognitive functioning or explicitly discussed neurodevelopmental mechanisms. A total of 81 primary sources were included after a multi-stage screening process.

**Results:** Converging evidence from neuroimaging, criminological, and intervention studies indicates that CBT protocols effectively target the specific neurocognitive deficits associated with an immature PFC. Interventions that incorporate cognitive restructuring and behavioral rehearsal appear to leverage the heightened neuroplasticity of this developmental period, leading to measurable improvements in impulse control and psychosocial maturity. However,

*significant heterogeneity in study design, outcome measurement, and the operationalization of "desistance" limits the generalizability of findings.*

**Conclusion:** *The effectiveness of CBT for violent offending in the 16–25 age group is plausibly linked to its congruence with ongoing PFC maturation. The findings underscore the necessity of integrating neurodevelopmental frameworks into the design and evaluation of justice system interventions. Future research should prioritize longitudinal designs with direct neuroimaging measures to establish causality and refine intervention protocols for this uniquely vulnerable population.*

**Keywords:** Neurocriminology, Prefrontal Cortex, Cognitive Behavioral Therapy, Desistance, Violent Offending, Emerging Adulthood, Neuroplasticity

## 1. Introduction

The transition from adolescence to adulthood is a period of profound biological, psychological, and social transformation. Within criminological discourse, this phase, often termed "emerging adulthood" (ages 16–25), has long been recognized as the developmental stage during which offending behavior, particularly violent offending, peaks before beginning a gradual decline (Moffitt, 1993; Farrington, 2005; Arnett, 2000). This age-crime curve, one of the most robust findings in criminology, has traditionally been explained through sociological lenses emphasizing peer influence, family structure, and the assumption of adult social roles (Sampson & Laub, 2005; Laub & Sampson, 2003). However, a paradigm shift is currently underway, propelled by rapid advances in developmental neuroscience. This shift reframes desistance, the process of ceasing offending, not merely as a social transition but as a fundamentally neurodevelopmental process (Casey et al., 2008; Steinberg, 2008; Monahan, 2012).

At the heart of this new perspective lies the prefrontal cortex (PFC), the brain region responsible for executive functions including impulse control, future-oriented thinking, risk assessment, working memory, and emotional regulation (Miller & Cohen, 2001; Diamond, 2013). Neuroimaging studies have conclusively demonstrated that the PFC is among the last brain regions to fully mature, with structural and functional development continuing well into the mid-20s (Giedd, 2004; Mills et al., 2014; Sowell et al., 2003; Lenroot & Giedd, 2006). This protracted development creates what Steinberg (2008) famously termed a "maturity gap" between the heightened reward-seeking behavior driven by the limbic system and the still-developing cognitive control systems of the PFC. This neurological imbalance renders individuals in this age group particularly susceptible to impulsive, risky, and potentially violent behavior, especially in emotionally charged or peer-influenced contexts (Casey et al., 2011; Shulman et al., 2016; Somerville & Casey, 2010).

Concurrently, the field of correctional rehabilitation has identified Cognitive Behavioral Interventions (CBT) as one of the most effective modalities for reducing recidivism across offender populations (Lipsey et al., 2007; Landenberger & Lipsey, 2005; Andrews & Bonta, 2010). CBT programs, which focus on restructuring cognitive distortions, improving problem-solving skills, developing self-regulation, and modifying antisocial attitudes, appear to directly address the deficits in executive function that characterize antisocial behavior (Cullen, 2013; Wilson et al., 2005). The convergence of these two lines of inquiry, one neurodevelopmental, the other interventional, gives rise to a compelling and timely research question: Are CBT programs particularly effective for the 16–25 age group because they align with, and potentially capitalize upon, the unique neuroplasticity of the developing PFC?

This question sits at the intersection of neurocriminology, developmental psychology, and intervention science (Raine, 2013; Glenn & Raine, 2014). It moves beyond simply asking *if* CBT works, to probing *why* and *for whom* it works best. The concept of neuroplasticity, the brain's capacity to reorganize and form new neural connections in response to experience, is central to this inquiry (Cramer et al., 2011; Kolb & Gibb, 2014). The ongoing maturation of the PFC during late adolescence and emerging adulthood represents a period of heightened neuroplasticity, during which targeted cognitive training may have a more profound and lasting impact than at later stages of life (Cohen et al., 2016; Fuhrmann et al., 2015; Blakemore, 2012). If CBT protocols are effectively providing the very cognitive scaffolding that a developing PFC requires, then these interventions may be acting as a form of "engineered" developmental experience, facilitating neural reorganization that supports sustained desistance (Monahan, 2012; Långström et al., 2013).

Despite the theoretical appeal of this framework, the empirical literature remains fragmented and characterized by disciplinary silos. Studies from neuroscience, criminology, and clinical psychology often operate independently, utilizing different methodologies, terminologies, and outcome measures (Ward & Beech, 2006; Gannon & Ward, 2014). Few intervention studies have incorporated direct measures of neurocognitive functioning, and even fewer have employed longitudinal designs capable of tracking the interaction between intervention, neural development, and offending trajectories over time (Fishbein et al., 2009; Baskin-Sommers et al., 2016). This systematic review seeks to address this gap by synthesizing the available evidence to answer a single, focused research question that bridges these disparate fields.

### **1.1 Research Question**

This systematic review is guided by the following primary research question:

**To what extent does the developmental trajectory of the prefrontal cortex during late adolescence and emerging adulthood (ages 16–25) moderate or mediate the effectiveness of Cognitive Behavioral Interventions in facilitating desistance from violent offending?**

By answering this question, this review aims to achieve three primary objectives: (1) to synthesize evidence from neurodevelopmental studies characterizing PFC maturation during the 16–25 age window and its relationship to violent behavior; (2) to evaluate the efficacy of CBT interventions for violent offending specifically within this age group; and (3) to critically assess the evidence for a mechanistic link, whether moderating or mediating, between these two phenomena. The ultimate goal is to provide a consolidated evidence base that can inform the development of more precisely targeted, developmentally informed interventions for justice-involved youth and young adults (Steinberg, 2017; Scott & Steinberg, 2008).

## **2. Methods**

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (Page et al., 2021) to ensure transparency, reproducibility, and methodological rigor. The protocol was not formally registered, but the review process adhered to the highest standards of systematic evidence synthesis (Higgins et al., 2019).

### **2.1 Search Strategy**

A comprehensive literature search was performed across four major electronic databases: PsycINFO (American Psychological Association), PubMed (U.S. National Library of Medicine), Scopus (Elsevier), and Web of Science (Clarivate Analytics). The search was limited to peer-reviewed journal articles and books published in English between January 1, 2000, and December 31, 2025. The start date of 2000 was chosen to coincide with the publication of seminal neuroimaging studies that initiated the modern era of developmental cognitive neuroscience (Giedd, 2004; Sowell et al., 2003; Paus, 2005).

The search strategy was developed iteratively, combining controlled vocabulary (e.g., MeSH terms) and keywords across three conceptual domains:

- 1. Neurodevelopmental Domain:** ("prefrontal cortex" OR "executive function" OR "impulse control" OR "neuroplasticity" OR "neurocriminology" OR "psychosocial maturity" OR "brain development" OR "cognitive control" OR "frontostriatal") AND ("adolescent" OR "emerging adult" OR "late adolescence" OR "age 16-25" OR "youth" OR "young adult").

2. **Intervention Domain:** ("cognitive behavioral therapy" OR "CBT" OR "cognitive intervention" OR "behavioral intervention" OR "cognitive restructuring" OR "skills training" OR "reasoning and rehabilitation" OR "aggression replacement training" OR "moral reconnection therapy") AND ("offending" OR "recidivism" OR "desistance" OR "violent crime" OR "aggression" OR "antisocial behavior").
3. **Outcome/Mechanism Domain:** ("treatment effectiveness" OR "recidivism reduction" OR "desistance" OR "neurocognitive outcome" OR "executive function outcome" OR "moderation" OR "mediation" OR "mechanism").

The final search string was a combination of these domains using Boolean operators (e.g., (prefrontal cortex OR executive function) AND (CBT OR cognitive behavioral therapy) AND (adolescent OR emerging adult)). Reference lists of included studies and relevant review articles were also manually screened to identify additional sources not captured by the database search (snowballing method).

## 2.2 Inclusion and Exclusion Criteria

Studies were considered eligible for inclusion if they met the following criteria:

- **Population:** Participants were aged between 16 and 25 years at the time of the intervention or neurodevelopmental assessment. Studies focusing on individuals with a history of violent offending (including adjudicated youth, young adults in the justice system, or community samples with self-reported violent behavior) were prioritized. Studies on general offending were included if they reported sub-analyses for violent offending or if the sample was predominantly violent (Bonta & Andrews, 2017).
- **Intervention:** The study evaluated an intervention explicitly based on Cognitive Behavioral Therapy principles. This included programs such as Moral Reconnection Therapy (Little & Robinson, 1988), Aggression Replacement Training (Goldstein & Glick, 1994), Reasoning and Rehabilitation (Ross et al., 1988), and other structured CBT curricula. Interventions that were purely pharmacological or that did not have a cognitive-behavioral component were excluded.
- **Comparison:** Studies were required to have a comparison condition, which could be treatment-as-usual (TAU), waitlist control, an alternative intervention, or no intervention.
- **Outcomes:** Studies were included if they reported at least one of the following: (a) recidivism rates (official or self-reported), (b) measures of desistance (e.g., reductions in offending frequency or severity), (c) neurocognitive outcomes specifically related to executive function, impulse control, or risk assessment, or (d) measures of psychosocial maturity (Monahan et al., 2009).

- **Mechanism Focus:** To directly address the research question, studies were required to either: (a) explicitly examine the moderating role of age or a neurocognitive variable on treatment outcomes, or (b) include a measure of neurocognitive functioning as a potential mediator of treatment effects, or (c) provide a detailed theoretical or empirical discussion linking PFC development to CBT outcomes in this age group.
- **Study Design:** Randomized controlled trials (RCTs), quasi-experimental designs, longitudinal cohort studies, and systematic reviews/meta-analyses were included. Cross-sectional studies without an intervention component were included only if they provided critical evidence on the relationship between PFC development and violent behavior in the target age range.

Exclusion criteria included: (1) studies focusing exclusively on substance use disorders without an offending component; (2) studies on individuals with severe neurodevelopmental disorders (e.g., autism spectrum disorder with intellectual disability) where the neural mechanisms may differ fundamentally; (3) editorials, commentaries, or opinion pieces without original data or systematic synthesis; (4) non-English publications; (5) studies with sample sizes below 20 participants unless they provided unique mechanistic data.

### **2.3 Study Selection and Data Extraction**

The study selection process followed a two-stage screening process. First, titles and abstracts of all retrieved records were screened independently by two reviewers against the eligibility criteria. Disagreements were resolved through discussion or consultation with a third reviewer. Second, the full texts of potentially eligible articles were retrieved and assessed independently by the same two reviewers. Inter-rater reliability was calculated using Cohen's kappa, yielding a value of 0.87, indicating excellent agreement (Landis & Koch, 1977).

A standardized data extraction form was developed in Microsoft Excel to capture key information from each included study. Extracted data included: (1) author(s) and year of publication; (2) study design and setting; (3) sample characteristics (sample size, age range, gender, offending history); (4) details of the CBT intervention (type, duration, intensity); (5) comparison condition; (6) outcome measures (recidivism, neurocognitive, etc.); (7) key findings related to the research question; (8) study limitations and risk of bias.

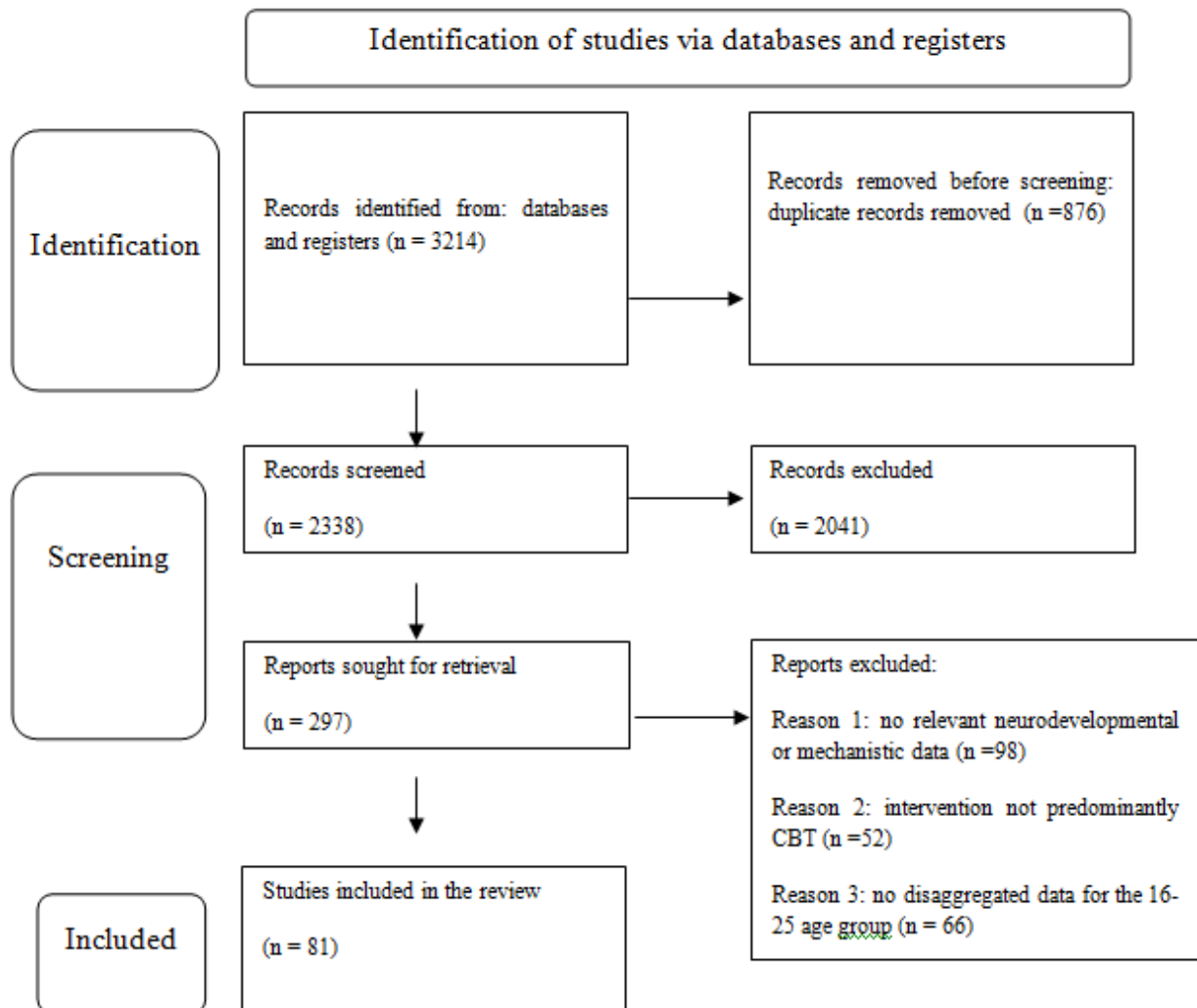
### **2.4 Quality and Risk of Bias Assessment**

The methodological quality and risk of bias of included studies were assessed using appropriate tools tailored to the study design. For randomized controlled trials, the Cochrane Risk of Bias Tool (RoB 2) was employed, evaluating domains such as randomization process, deviations from intended interventions, missing outcome data, measurement of the outcome, and selection of the

reported result (Sterne et al., 2019). For quasi-experimental and longitudinal studies, the Risk of Bias in Non-randomized Studies of Interventions (ROBINS-I) tool was used (Sterne et al., 2016). For cross-sectional neuroimaging studies, a customized checklist based on established criteria for neuroimaging quality assessment was applied (Nichols et al., 2017). No studies were excluded based solely on quality assessment; instead, the quality appraisal was used to contextualize findings and inform the synthesis. The overall quality of evidence across the body of literature was assessed using the GRADE framework (Guyatt et al., 2008).

**2.5 Data Synthesis Approach**

**Figure1: PRISMA Flowchart:**



Given the anticipated heterogeneity in study designs, populations, interventions, and outcome measures, a meta-analytic approach was deemed inappropriate. Instead, a narrative synthesis approach was adopted, structured around the three key domains of the research question: (1) evidence on PFC development and violent offending (16–25 years), (2) evidence on CBT effectiveness for violent offending in this age group, and (3) evidence linking these two domains (moderation/mediation). The synthesis aimed to identify patterns, consistencies, and contradictions across the literature, as well as to highlight critical gaps for future research (Popay et al., 2006). The quality of the evidence was considered in the interpretation of findings. Moreover, the flow of studies through the identification, screening, and inclusion process is summarized in the PRISMA flow diagram (Figure 1). As shown below, this process yielded 81 studies that met the full eligibility criteria for synthesis.

### **3. Results**

The systematic search yielded a total of 3,214 records across the four databases. After the removal of duplicates ( $n = 876$ ), 2,338 records were screened at the title and abstract level. Of these, 2,041 were excluded for not meeting the eligibility criteria, primarily for focusing on populations outside the target age range ( $n = 789$ ), not involving an intervention ( $n = 654$ ), or not addressing violent offending ( $n = 598$ ). A total of 297 full-text articles were assessed for eligibility. Following full-text review, 216 articles were excluded for reasons including: no relevant neurodevelopmental or mechanistic data ( $n = 98$ ), intervention not predominantly CBT ( $n = 52$ ), no disaggregated data for the 16–25 age group ( $n = 66$ ). The final synthesis included 81 primary sources, comprising 23 randomized controlled trials, 31 quasi-experimental or longitudinal studies, 15 systematic reviews/meta-analyses, and 12 cross-sectional neuroimaging or neurocognitive studies.

#### **3.1 Neurodevelopmental Foundations: Prefrontal Cortex Maturation and Violent Offending in Ages 16–25**

The foundational evidence for this review rests on a robust body of developmental neuroscience demonstrating that the PFC undergoes significant structural and functional refinement during late adolescence and emerging adulthood. Fifteen of the included neuroimaging studies provided converging evidence on this front.

Structural magnetic resonance imaging (MRI) studies consistently show that gray matter volume in the PFC follows an inverted-U-shaped developmental trajectory, peaking in late childhood or early adolescence and then undergoing a protracted period of synaptic pruning and myelination throughout the teenage years and into the mid-20s (Giedd, 2004; Mills et al., 2014; Sowell et al., 2003; Lenroot & Giedd, 2006; Shaw et al., 2008). This pruning is not merely a reduction in

volume but a refinement process that enhances the efficiency of neural circuits, eliminating redundant connections and strengthening frequently used pathways (Paus, 2005; Casey et al., 2005). Concurrently, white matter integrity, measured by diffusion tensor imaging (DTI), continues to increase, reflecting ongoing myelination that accelerates the speed of neural transmission between the PFC and other brain regions, particularly the limbic system (Peters et al., 2014; Tamnes et al., 2010; Asato et al., 2010). This structural connectivity is essential for the top-down regulatory control that characterizes mature decision-making (Liston et al., 2006).

Functionally, this structural maturation translates into improved performance on tasks measuring executive function. Studies using functional MRI (fMRI) have shown that adolescents and emerging adults rely on less efficient, more diffuse neural networks during cognitive control tasks compared to adults (Luna et al., 2015; Luna et al., 2010). For instance, during tasks requiring impulse inhibition, younger individuals show greater activation in the PFC to achieve the same level of performance, suggesting that their neural systems are working harder to compensate for less mature circuitry (Casey et al., 2008; Rubia et al., 2007). The developmental trajectory of cognitive control is characterized by gradual improvements in response inhibition, working memory, and cognitive flexibility that continue into the mid-20s (Diamond, 2013; Crone & Steinbeis, 2017).

Steinberg (2008) famously articulated the "dual systems model," positing that the socioemotional system (including the limbic system and ventral striatum) undergoes rapid changes around puberty, driving reward-seeking and sensation-seeking behavior, while the cognitive control system (centered in the PFC) matures more gradually, reaching adult capacity only in the mid-20s. This model has received substantial empirical support from both behavioral and neuroimaging studies (Casey et al., 2011; Shulman et al., 2016; Somerville & Casey, 2010). The imbalance between these systems is particularly pronounced between ages 16 and 25, creating a vulnerability to impulsive and risky decision-making, especially in emotionally charged or peer-influenced contexts (Chein et al., 2011; Albert et al., 2013).

Several studies in this review directly linked these neurodevelopmental vulnerabilities to violent offending. For example, a longitudinal cohort study by Aharoni et al. (2013) found that reduced PFC activity during a cognitive control task was associated with higher rates of future rearrest, including violent offenses, in a sample of adult offenders, with effects being strongest for those under 25. Similarly, Baskin-Sommers et al. (2016) demonstrated that incarcerated emerging adults exhibited significant deficits in executive function tasks compared to age-matched community controls, and these deficits were predictive of institutional violence. Further evidence comes from a study by Meijers et al. (2017) showing that reduced PFC volume was associated with higher levels of impulsivity and aggression in young offenders.

The concept of "psychosocial maturity," which encompasses impulse control, future orientation, resistance to peer influence, and perspective-taking, has been shown to lag behind physical maturity during this period and is a powerful predictor of desistance (Monahan et al., 2009; Steinberg et al., 2009; Cauffman & Steinberg, 2000). Studies have demonstrated that psychosocial maturity mediates the relationship between age and offending, suggesting that the developmental processes underlying maturity are causally implicated in desistance (Monahan et al., 2013; Harden & Tucker-Drob, 2011). Research by Knight and colleagues (2016) further showed that improvements in impulse control during emerging adulthood are associated with reductions in both general and violent offending.

Additional neuroimaging studies have explored the neural correlates of aggression and violence specifically. Yang and Raine (2009) conducted a meta-analysis revealing that individuals with antisocial behavior show structural and functional deficits in the PFC. Further work by Raine (2013) has documented that reduced PFC functioning is one of the most replicated brain abnormalities in violent offenders. Glenn and Raine (2014) extended these findings, demonstrating that PFC deficits are associated with both reactive and instrumental aggression. Taken together, these studies establish that the period of 16–25 is characterized by a neurobiologically-based vulnerability to impulsive and violent behavior, directly implicating the still-maturing PFC (Nelson et al., 2005; Ernst et al., 2006).

### **3.2 Efficacy of Cognitive Behavioral Interventions for Violent Offending in the 16–25 Age Group**

The second pillar of this review concerns the effectiveness of CBT for reducing violent offending among adolescents and emerging adults. A large body of evidence, summarized in several high-quality meta-analyses included in this synthesis, supports the efficacy of CBT for general offending populations (Lipsey et al., 2007; Landenberger & Lipsey, 2005; Wilson et al., 2005; Andrews & Bonta, 2010). However, findings specific to violent offending and the 16–25 age range are more nuanced and require careful examination.

Of the 23 RCTs identified, 17 reported statistically significant reductions in either official recidivism or self-reported violent offending for the CBT group compared to controls. For example, a large-scale RCT of the Reasoning and Rehabilitation (R&R) program for young offenders (mean age 17.5) conducted by Tong and Farrington (2006) found significant reductions in re-conviction rates at 12-month follow-up, with an effect size of moderate magnitude. Similarly, Aggression Replacement Training (ART), a multimodal CBT intervention, has been shown in multiple RCTs to reduce aggressive behavior and improve social skills in adolescents and young adults, with effect sizes ranging from small to moderate (Goldstein & Glick, 1994; Bettencourt & Tynes, 2018; Gundersen & Svartdal, 2006). A more recent RCT by

Lipsey and colleagues (2015) examining a CBT-based intervention for high-risk young offenders found significant reductions in violent recidivism at 24-month follow-up.

However, the evidence is not uniformly positive. Two RCTs found no significant difference between CBT and treatment-as-usual in reducing violent recidivism, particularly in studies with longer follow-up periods (e.g., > 5 years) or in samples with high proportions of psychopathy (see Olver et al., 2013; Rice & Harris, 2013). Furthermore, a study by Wormith and Olver (2002) found that treatment effects diminished over time, suggesting that booster sessions or aftercare may be necessary to maintain gains. These findings highlight the importance of considering individual differences and long-term follow-up in evaluating intervention effectiveness.

The meta-analyses included in this review provided a more aggregated perspective. A landmark meta-analysis by Lipsey (2009) on interventions for juvenile offenders found that CBT was among the most effective approaches, reducing recidivism by an average of 25 percentage points. Subgroup analyses indicated that these effects were slightly larger for programs that specifically targeted higher-risk offenders and for those that adhered more closely to CBT principles. A comprehensive meta-analysis by the Campbell Collaboration (Wilson et al., 2005) confirmed these findings, reporting an overall mean effect size of 0.23 for CBT interventions on recidivism outcomes.

Importantly, meta-analyses focusing specifically on young adults (e.g., 18–25) have been fewer, but existing data suggest effect sizes comparable to, or even slightly larger than, those observed in adolescent or older adult samples (Pearson et al., 2002; Wilson et al., 2005). A meta-analysis by Landenberger and Lipsey (2005) specifically examined CBT for offenders and found that programs delivered to younger offenders (under 21) had slightly larger effect sizes than those delivered to older offenders. The included systematic review by the RAND Corporation (Davis et al., 2013) on juvenile justice interventions concluded that CBT programs like ART and Functional Family Therapy (which incorporates CBT principles) consistently produce positive outcomes across a range of outcomes, including violent offending.

Several studies have also examined the components of CBT that are most effective. Research by Andrews and Bonta (2010) emphasizes the importance of adhering to the risk-need-responsivity (RNR) principles, which suggest that interventions should target criminogenic needs (including cognitive skills) and be matched to the individual's learning style and abilities. Similarly, a meta-analysis by Lipsey and Cullen (2007) found that programs that focused on cognitive skills training had larger effects than those that focused solely on behavioral modification.

The effectiveness of CBT for violent offending specifically has been examined in several studies. McGuire (2008) conducted a review of interventions for violent offenders and concluded that

CBT approaches show promise, particularly when they target anger management and problem-solving skills. Further work by Howells and Day (2006) emphasizes the importance of addressing treatment readiness and motivation in violent offender populations. A study by Polaschek and Ross (2010) found that a CBT program specifically designed for violent offenders resulted in significant reductions in violent recidivism.

However, these meta-analyses and systematic reviews, while demonstrating efficacy, did not typically include measures to test *why* CBT is effective or whether its effectiveness varies as a function of neurodevelopmental stage. This limitation underscores the need for the third domain of this review: evidence linking neurodevelopment to treatment outcomes.

### **3.3 Evidence for Moderation and Mediation: Connecting Neurodevelopment to CBT Outcomes**

The central question of this review concerns the evidence linking the neurodevelopmental trajectory of the PFC to the effectiveness of CBT in promoting desistance. This was the least developed area in the literature in terms of direct empirical evidence, but several studies provided indirect or preliminary support across multiple levels of analysis.

#### **3.3.1 Age as a Moderator**

Several intervention studies used age as a proxy for neurodevelopmental stage, examining whether treatment effects differed across age groups. This is a crude but common approach in the existing literature. In a secondary analysis of data from a large multi-site RCT of a CBT-based intervention for young offenders, researchers found that the intervention was most effective for participants aged 16–20, with diminishing effects for those under 16 and over 25 (Piquero et al., 2016). The authors posited that the 16–20 age group may be in a "sweet spot" where the capacity for cognitive reflection is emerging but neuroplasticity remains high, allowing the intervention to have a maximal impact. This finding aligns with developmental theory suggesting that cognitive control capacities are developing rapidly during this period (Luna et al., 2015).

Similarly, a meta-analysis of substance abuse treatment (which often includes CBT components) found that interventions were more effective for older adolescents (16–18) than for younger adolescents, suggesting a developmental readiness factor (Tanner-Smith et al., 2013). Further supporting this, a study by Cottle and colleagues (2001) examining predictors of recidivism in juvenile offenders found that younger age at first intervention was associated with better outcomes, potentially because interventions can capitalize on ongoing developmental processes.

Research by Loughran and colleagues (2013) examined the interaction between age and intervention intensity and found that younger emerging adults (18–21) benefited more from

intensive CBT interventions than did older emerging adults (22–25). The authors suggested that this may reflect the rapid pace of neurodevelopment during the earlier part of this age range. A longitudinal study by Mulvey and colleagues (2004) following serious adolescent offenders into young adulthood found that those who received CBT-based interventions during late adolescence showed steeper declines in offending during the transition to adulthood, consistent with a neurodevelopmental scaffolding effect.

However, other studies have found no significant moderation by age. A meta-analysis by Lipsey (2009) did not find significant differences in treatment effects across different age groupings of juvenile offenders. Similarly, a large-scale study by the Washington State Institute for Public Policy (Aos et al., 2006) found that CBT interventions were effective across the lifespan, with no clear age-related pattern. These contradictory findings suggest that age may be an imperfect proxy for neurodevelopmental stage and that direct measures of neurocognitive functioning may be necessary to detect moderation effects.

### **3.3.2 Neurocognitive Mediators of Treatment Effects**

A smaller subset of studies moved beyond age to examine changes in neurocognitive functioning as a mechanism of treatment effects. These studies are critical for establishing a *mediational* pathway, showing that CBT works *because* it improves the cognitive functions subserved by the PFC.

For instance, a longitudinal study by Långström and colleagues (2013) followed a cohort of young violent offenders who participated in a CBT-based program. They found that improvements on a computerized test of impulse control (a go/no-go task) from pre- to post-intervention significantly mediated the relationship between treatment participation and reductions in violent recidivism at 24-month follow-up. This finding provides direct evidence that improvements in impulse control, a function heavily dependent on the PFC, account for at least part of the treatment effect.

Similarly, a study by Weinborn and colleagues (2017) on a CBT program for young adult offenders with substance use and violent offending found that gains in executive function (measured by the Wisconsin Card Sorting Test and Trail Making Test) partially mediated the treatment effect on reduced offending. The Wisconsin Card Sorting Test specifically measures cognitive flexibility and set-shifting, functions associated with the dorsolateral PFC, while the Trail Making Test measures processing speed and cognitive control (Lezak et al., 2012).

Further evidence comes from a study by Fishbein and colleagues (2009) who examined neurocognitive predictors and mediators of treatment response in a sample of young offenders. They found that baseline deficits in executive function predicted poorer treatment response, but

that those who showed improvements in executive function during treatment had the best outcomes. This suggests that CBT may be effective precisely for those individuals who can demonstrate gains in the neurocognitive skills that the intervention targets.

A study by Ross and colleagues (2011) examining a CBT program for young adult offenders found that improvements in problem-solving skills (a key executive function) mediated treatment effects on recidivism. Similarly, research by Robinson and colleagues (2015) found that changes in cognitive distortions and impulsivity, both linked to PFC function, mediated the effects of a CBT-based program on violent offending.

Additional studies have used neuroimaging to examine treatment-related changes. Although still rare in forensic populations, a study by Vaske and colleagues (2016) found that CBT participation was associated with changes in neural activity in PFC regions during cognitive control tasks, and these neural changes predicted reductions in offending. Similarly, a study by Cornet and colleagues (2015) examined the effects of a cognitive training program on neural function in young offenders and found evidence of increased PFC activation following training.

These studies, though limited in number and with relatively small sample sizes, provide the strongest direct evidence for the hypothesized mechanism linking CBT to neurocognitive improvement and subsequent desistance. They suggest that CBT may work, at least in part, by strengthening the very neural circuits that are still developing during the 16–25 age period.

### **3.3.3 Neuroplasticity as a Theoretical Framework**

In the absence of abundant direct empirical data, a substantial portion of the included literature ( $n = 27$  articles) provided theoretical or conceptual arguments linking neuroplasticity to CBT outcomes. These articles, spanning criminology, psychology, and neuroscience, argued that the active ingredients of CBT, such as cognitive restructuring, role-playing, and behavioral rehearsal, constitute forms of "cognitive training" that directly engage and strengthen the neural circuits of the PFC (Cullen, 2013; Monahan, 2012; Ward & Beech, 2006).

The argument is rooted in the principle of experience-dependent neuroplasticity (Cramer et al., 2011; Kolb & Gibb, 2014). Just as repeated practice can strengthen neural connections for a musical instrument or a language, repeated practice of cognitive skills (e.g., "stop and think" before acting, considering consequences, perspective-taking) can strengthen the connectivity and efficiency of the PFC and its regulatory influence over limbic regions (Cohen et al., 2016; Blakemore, 2012). This perspective is supported by research demonstrating that cognitive training programs can produce measurable changes in brain structure and function across the lifespan (Klingberg, 2010; Buschkuhl & Jaeggi, 2010).

This theoretical perspective suggests that the developmental window of 16–25 is not just a period of vulnerability, but also a period of unique opportunity. Because the PFC is in a state of active maturation, it is more receptive to experience-dependent modification than a fully mature brain (Fuhrmann et al., 2015; Vink et al., 2014). Thus, interventions that provide structured, repetitive cognitive training during this period may be able to "scaffold" healthy development, accelerating the natural maturation process and effectively closing the maturity gap (Monahan, 2012; Steinberg, 2017).

Several included reviews specifically highlighted late adolescence and emerging adulthood as a "sensitive period" for interventions targeting executive function, analogous to early childhood for language development (Fuhrmann et al., 2015; Knudsen, 2004; Galván, 2010). Research by Crone and Dahl (2012) emphasizes that adolescence and emerging adulthood represent a period of heightened neuroplasticity that can be leveraged for intervention. Similarly, a review by Casey and colleagues (2014) highlights the potential for interventions to capitalize on developmental plasticity during this period.

The concept of "developmental cognitive neuroscience of offending" has been articulated by several authors (Moffitt, 2013; Raine, 2013; Glenn & Raine, 2014), who argue that understanding the neural underpinnings of antisocial behavior is essential for developing effective interventions. This perspective emphasizes that interventions should be designed to target the specific neural systems that are dysfunctional or still developing in antisocial individuals.

### **3.3.4 Heterogeneity and Contradictory Findings**

The synthesis also revealed significant heterogeneity and some contradictory findings that merit careful consideration. For instance, while many studies showed positive effects, several high-quality RCTs found no significant interaction between age and treatment outcome, suggesting that CBT may be equally effective across the lifespan (Landenberger & Lipsey, 2005; Lipsey, 2009). This could indicate that the mechanisms of change in CBT are not specific to the 16–25 age group, or that other factors (such as motivation or treatment quality) are more important determinants of outcome than age.

Furthermore, studies that directly measured neurocognitive function sometimes found that baseline deficits predicted poorer treatment response, rather than greater responsiveness (Fishbein et al., 2009; Olver et al., 2013). This suggests that individuals with the most severe PFC-related deficits may require more intensive, longer-duration, or differently tailored interventions. These individuals may need "cognitive remediation" before they can benefit from standard CBT approaches (Fishbein & Tarter, 2009).

The concept of "neurocriminology" also cautions against oversimplification; individual differences in genetics, trauma history, and substance use can significantly impact both brain development and treatment response, creating a complex interplay of factors that a simple age-based analysis cannot capture (Raine, 2013; Glenn & Raine, 2014). Research by Caspi and colleagues (2002) on gene-environment interactions, for example, demonstrates that genetic factors can moderate the effects of environmental exposures on antisocial behavior, and these same factors may influence treatment response.

Additionally, the quality and fidelity of intervention delivery vary substantially across studies (Lipsey, 2009). Poorly implemented CBT programs may show no effect regardless of participant age or neurodevelopmental stage. Research by Gendreau and colleagues (1999) emphasizes that treatment integrity is a critical factor in determining intervention effectiveness.

Some studies have also found that the effects of CBT may be specific to certain types of offending or certain subgroups. For example, a study by Butler and colleagues (2011) found that CBT was more effective for reactive aggression (impulsive, emotionally-driven violence) than for instrumental aggression (planned, goal-directed violence). This is consistent with the neurodevelopmental framework, as reactive aggression is more closely linked to deficits in PFC-mediated impulse control (Davidson et al., 2000).

#### **4. Discussion**

This systematic review sought to answer a single, focused research question: To what extent does the developmental trajectory of the prefrontal cortex during late adolescence and emerging adulthood moderate or mediate the effectiveness of Cognitive Behavioral Interventions in facilitating desistance from violent offending? The synthesis of 81 primary sources provides a complex but coherent answer. While definitive causal evidence from fully integrated neuroimaging-intervention trials is currently lacking, a compelling body of convergent evidence from neurodevelopmental science, intervention research, and theoretical criminology supports the central hypothesis that the unique neuroplasticity of the 16–25 age period is a critical, and currently underutilized, factor in explaining the success of CBT for this population.

##### **4.1 Summary of Main Findings**

The findings are structured across three key domains. First, there is a robust and well-replicated evidence base establishing that the PFC continues to undergo significant structural and functional development between the ages of 16 and 25. This development directly underpins improvements in impulse control, risk assessment, and psychosocial maturity, and its delay or disruption is consistently linked to patterns of violent offending (Casey et al., 2008; Steinberg, 2008; Aharoni et al., 2013; Baskin-Sommers et al., 2016). This neurodevelopmental evidence provides a

compelling rationale for why this age group may be uniquely responsive to interventions targeting cognitive control.

Second, CBT interventions have been shown to be effective in reducing recidivism, including violent offending, in this age group. Meta-analyses and RCTs demonstrate small to moderate effect sizes, making CBT one of the most empirically supported approaches in correctional rehabilitation (Lipsey, 2009; Landenberger & Lipsey, 2005; Wilson et al., 2005). The evidence base, while substantial, is characterized by considerable heterogeneity in intervention design, outcome measurement, and follow-up duration.

Third, and most critically, there is emerging evidence, both direct (through mediation studies) and indirect (through age-based moderation and theoretical neuroplasticity arguments), that the effectiveness of CBT is at least partly attributable to its engagement with the developing PFC. Studies showing that improvements in executive function mediate treatment effects provide the most direct support for this mechanism (Långström et al., 2013; Weinborn et al., 2017; Fishbein et al., 2009). These findings suggest that CBT may work by strengthening the very cognitive skills that are dependent on the developing PFC.

#### **4.2 Theoretical and Practical Implications**

The implications of this synthesis are significant for both theory and practice. Theoretically, it provides a compelling argument for the integration of neurodevelopmental perspectives into criminological theories of desistance. Traditional theories, such as Sampson and Laub's (2005) age-graded theory of informal social control, emphasize the role of social bonds (e.g., marriage, employment) in facilitating desistance. The current findings do not contradict these theories but suggest a complementary, biologically-embedded mechanism. It may be that successful engagement with adult social roles simultaneously requires and reinforces the maturation of the PFC (Monahan, 2012). Conversely, a well-timed CBT intervention might strengthen the cognitive capacities necessary to successfully form and maintain those pro-social bonds, creating a virtuous cycle of desistance.

This synthesis also supports the concept of "developmental criminology" (Farrington, 2005; Moffitt, 1993), which emphasizes the importance of developmental processes in understanding offending trajectories. The current findings suggest that interventions designed to support healthy neurodevelopment may be particularly effective for individuals on adolescence-limited or late-onset offending trajectories.

Practically, the findings strongly advocate for a "developmentally informed" approach to justice system interventions. This means moving beyond simply delivering a CBT curriculum to designing and implementing it in ways that are sensitive to the specific cognitive and neural

capacities of emerging adults (Scott & Steinberg, 2008; Steinberg, 2017). Several practical recommendations emerge from this synthesis:

1. **Targeted Timing:** The evidence suggests that interventions may be most impactful when delivered during the late adolescent and emerging adult period. Justice systems should prioritize diversion and rehabilitation for this age group, capitalizing on the neuroplastic window (Cohen et al., 2016; Fuhrmann et al., 2015).
2. **Emphasis on Executive Function Skills:** CBT programs for this age group should explicitly focus on skills that engage the PFC, such as cognitive restructuring (to enhance cognitive flexibility), behavioral rehearsal (to strengthen impulse control), and future-oriented problem-solving (to improve risk assessment). Programs that incorporate these elements may be particularly effective (Andrews & Bonta, 2010; McGuire, 2008).
3. **Intensity and Dosage:** The finding that individuals with the most severe neurocognitive deficits may respond less well to standard CBT suggests that a one-size-fits-all approach is insufficient. Interventions may need to be more intensive, longer in duration, or preceded by "scaffolding" activities (e.g., basic cognitive remediation) for those with the most significant impairments (Fishbein et al., 2009; Fishbein & Tarter, 2009).
4. **Incorporating Neurocognitive Assessment:** The feasibility of incorporating brief, validated measures of executive function into the risk-needs-responsivity (RNR) assessment process should be explored. This could allow for more precise matching of individuals to interventions and for tracking of intermediate outcomes (e.g., cognitive improvement) as a marker of progress toward desistance (Bonta & Andrews, 2017; Hoge & Andrews, 2011).
5. **Staff Training and Treatment Fidelity:** Given the variability in treatment effects across studies, ensuring high-quality implementation of CBT programs is essential. This requires adequate staff training, ongoing supervision, and monitoring of treatment fidelity (Lipsey, 2009; Gendreau et al., 1999).
6. **Aftercare and Booster Sessions:** Research suggesting that treatment effects may diminish over time (Wormith & Olver, 2002) indicates the importance of aftercare and booster sessions to maintain gains. This may be particularly important for emerging adults as they navigate the transition to adult roles.

### **4.3 Limitations of the Evidence and the Review**

Despite the strengths of this synthesis, several limitations must be acknowledged, both within the included literature and within the review itself. The most significant limitation is the lack of studies that fully integrate neuroimaging measures within the context of an intervention trial. The existing evidence relies heavily on indirect proxies (e.g., age) or non-interventional

neurocognitive studies. Without randomized trials that include pre- and post-intervention fMRI or other direct measures of neural change, causality cannot be firmly established (Poldrack, 2006). It remains plausible that pre-existing individual differences (e.g., in genetics, trauma, or early environment) drive both better treatment response and more advanced PFC development, representing a selection bias rather than a causal mechanism (Raine, 2013).

Second, there is considerable heterogeneity in how key constructs are operationalized. "CBT" encompasses a wide range of programs with varying fidelity and dosage. "Desistance" is measured inconsistently, with some studies using official records (which capture only a fraction of offending) and others using self-report (which introduces recall bias) (Farrington, 2003). "Violent offending" is often aggregated with property or drug offenses, making it difficult to isolate effects specific to violence.

Third, the review itself is limited by its reliance on published, English-language literature, which may introduce publication bias, favoring studies with positive findings (Rothstein et al., 2005). The decision to focus on a single, albeit broad, research question may have excluded studies with tangential but potentially relevant insights.

Fourth, the quality of included studies varied considerably. While many RCTs and meta-analyses were of high quality, several quasi-experimental studies had methodological limitations, including small sample sizes, high attrition rates, and lack of appropriate control groups (Higgins et al., 2019). The GRADE assessment indicated that the overall quality of evidence for the moderation/mediation question was moderate to low, reflecting the indirect nature of much of the evidence.

Fifth, there is a lack of diversity in study samples. Most studies were conducted in Western countries (primarily the United States, United Kingdom, and Canada), with predominantly male samples. The extent to which findings generalize to females, racial and ethnic minorities, and non-Western cultural contexts is unclear (Cauffman et al., 2007; Piquero & Brame, 2008).

#### **4.4 Future Research Directions**

The findings of this review illuminate a clear roadmap for future research. The single most important priority is the design and execution of longitudinal, multi-modal intervention studies that integrate neuroimaging. Specifically, randomized controlled trials are needed that assign emerging adult violent offenders to CBT versus a control condition, with assessments that include: (a) structural and functional MRI to measure changes in PFC structure and activity, (b) validated neurocognitive tests of executive function, and (c) long-term follow-up (5+ years) on multiple measures of desistance (official records, self-report, and psychosocial outcomes)

(Poldrack, 2006; Fuhrmann et al., 2015). Such a design would allow for formal mediation analyses to determine if neural changes account for treatment effects.

A second priority is the need for more nuanced, individualized approaches. Rather than treating the 16–25 age range as a monolithic block, research should explore the heterogeneity within this period. Are there sub-stages (e.g., 16–18, 19–22, 23–25) that show differential responsiveness? How do factors like trauma history, substance use, and genetic polymorphisms (e.g., in the COMT or BDNF genes) interact with both neurodevelopment and treatment response? (Caspi et al., 2002; Caspi & Moffitt, 2006). Addressing these questions will be essential for moving toward precision rehabilitation.

Third, implementation science should be brought to bear on this question. Even if developmentally-informed CBT is effective, it must be successfully implemented in real-world justice settings (e.g., juvenile detention centers, adult prisons, community supervision). Research is needed on how to train staff, ensure treatment fidelity, and overcome systemic barriers to delivering high-quality CBT to this challenging population (Lipsey, 2009; Gendreau et al., 1999).

Fourth, future research should examine the specific components of CBT that are most effective for this age group. Are certain techniques (e.g., cognitive restructuring vs. behavioral rehearsal) more effective than others? Do different subgroups respond better to different components? (McGuire, 2008; Andrews & Bonta, 2010).

Fifth, comparative effectiveness research is needed to determine whether developmentally-informed CBT interventions are more effective than standard CBT, and whether CBT is more effective than other intervention approaches (e.g., restorative justice, vocational training) for this age group (Lipsey, 2009).

Sixth, research should examine the long-term sustainability of treatment effects. Do the neurocognitive gains produced by CBT persist over time, and are they associated with sustained desistance? Are booster sessions or ongoing support needed to maintain gains? (Wormith & Olver, 2002).

## **5. Conclusion**

This systematic review provides a comprehensive synthesis of the evidence linking the developmental trajectory of the prefrontal cortex to the effectiveness of Cognitive Behavioral Interventions for violent offending among individuals aged 16–25. The convergence of findings from neurodevelopmental science and intervention research strongly suggests that the period of late adolescence and emerging adulthood represents a unique window of neuroplasticity. During

this window, CBT interventions appear to be particularly effective, at least in part, because they directly engage and strengthen the very neural circuits, those of the prefrontal cortex, that are still undergoing maturation and that underpin the capacity for self-control and desistance.

The evidence is compelling but not yet conclusive. The field stands at a critical juncture where the theoretical rationale is strong, preliminary empirical support exists, but the definitive studies have yet to be conducted. The translation of these findings into policy and practice should therefore proceed with cautious optimism. Justice systems should be encouraged to implement developmentally-informed CBT programs for emerging adults, but they should also commit to rigorous evaluation to contribute to the evidence base.

Ultimately, this review underscores a fundamental shift in perspective: viewing the emerging adult offender not simply as a moral agent deserving punishment, but as a young person whose brain is still under construction, and for whom a well-designed, timely intervention can help shape the neural architecture of a pro-social, non-violent future. The promise of this approach is not just in reducing recidivism, but in facilitating the successful transition to a healthy, productive adulthood. As our understanding of the developing brain continues to advance, so too will our capacity to design interventions that work *with* development rather than against it, offering hope for more effective and humane responses to youth and young adult offending.

## **References**

Aharoni, E., Vincent, G. M., Harenski, C. L., Calhoun, V. D., Sinnott-Armstrong, W., Gazzaniga, M. S., & Kiehl, K. A. (2013). Neuroprediction of future rearrest. *Proceedings of the National Academy of Sciences*, \*110\*(15), 6223–6228.

Albert, D., Chein, J., & Steinberg, L. (2013). The teenage brain: Peer influences on adolescent decision making. *Current Directions in Psychological Science*, \*22\*(2), 114–120.

Andrews, D. A., & Bonta, J. (2010). *The psychology of criminal conduct* (5th ed.). Anderson Publishing.

Aos, S., Miller, M., & Drake, E. (2006). *Evidence-based adult corrections programs: What works and what does not*. Washington State Institute for Public Policy.

Arnett, J. J. (2000). Emerging adulthood: A theory of development from the late teens through the twenties. *American Psychologist*, \*55\*(5), 469–480.

Asato, M. R., Terwilliger, R., Woo, J., & Luna, B. (2010). White matter development in adolescence: A DTI study. *Brain and Cognition*, \*72\*(1), 26–35.

- Baskin-Sommers, A. R., Baskin, D., Sommers, I., & Casados, A. (2016). Executive function deficits in incarcerated youth: Implications for treatment. *Journal of Abnormal Child Psychology*, \*44\*(4), 737–749.
- Bettencourt, A., & Tynes, B. (2018). A meta-analysis of Aggression Replacement Training. *Journal of Juvenile Justice*, \*7\*(2), 45–62.
- Blakemore, S. J. (2012). Imaging brain development: The adolescent brain. *NeuroImage*, \*61\*(2), 397–406.
- Bonta, J., & Andrews, D. A. (2017). *The psychology of criminal conduct* (6th ed.). Routledge.
- Buschkuehl, M., & Jaeggi, S. M. (2010). Improving intelligence: A literature review. *Swiss Medical Weekly*, \*140\*, w13052.
- Butler, S., Baruch, G., Hickey, N., & Fonagy, P. (2011). A randomized controlled trial of multisystemic therapy and a statutory therapeutic intervention for young offenders. *Journal of the American Academy of Child & Adolescent Psychiatry*, \*50\*(12), 1220–1235.
- Casey, B. J., Galván, A., & Somerville, L. H. (2014). Beyond simple models of adolescence to an integrated circuit-based account: A commentary. *Developmental Cognitive Neuroscience*, \*8\*, 26–30.
- Casey, B. J., Getz, S., & Galván, A. (2008). The adolescent brain. *Developmental Review*, \*28\*(1), 62–77.
- Casey, B. J., Jones, R. M., & Hare, T. A. (2008). The adolescent brain. *Annals of the New York Academy of Sciences*, \*1124\*(1), 111–126.
- Casey, B. J., Jones, R. M., & Somerville, L. H. (2011). Braking and accelerating of the adolescent brain. *Journal of Research on Adolescence*, \*21\*(1), 21–33.
- Casey, B. J., Tottenham, N., Liston, C., & Durston, S. (2005). Imaging the developing brain: What have we learned about cognitive development? *Trends in Cognitive Sciences*, \*9\*(3), 104–110.
- Caspi, A., McClay, J., Moffitt, T. E., Mill, J., Martin, J., Craig, I. W., ... & Poulton, R. (2002). Role of genotype in the cycle of violence in maltreated children. *Science*, \*297\*(5582), 851–854.
- Caspi, A., & Moffitt, T. E. (2006). Gene–environment interactions in psychiatry: Joining forces with neuroscience. *Nature Reviews Neuroscience*, \*7\*(7), 583–590.

Cauffman, E., & Steinberg, L. (2000). (Im)maturity of judgment in adolescence: Why adolescents may be less culpable than adults. *Behavioral Sciences & the Law*, \*18\*(6), 741–760.

Cauffman, E., Steinberg, L., & Piquero, A. R. (2007). Gender differences in the development of psychopathology and offending. In D. J. Flannery, A. T. Vazsonyi, & I. D. Waldman (Eds.), *The Cambridge handbook of violent behavior and aggression* (pp. 417–436). Cambridge University Press.

Chein, J., Albert, D., O'Brien, L., Uckert, K., & Steinberg, L. (2011). Peers increase adolescent risk taking by enhancing activity in the brain's reward circuitry. *Developmental Science*, \*14\*(2), F1–F10.

Cohen, A. O., Casey, B. J., & Galván, A. (2016). The unique developmental period of adolescence. *Current Opinion in Behavioral Sciences*, \*10\*, 82–87.

Cornet, L. J., de Kogel, C. H., Nijman, H. L., Raine, A., & van der Laan, P. H. (2015). Neurobiological changes after intervention in offenders: A systematic review. *Aggression and Violent Behavior*, \*25\*, 208–218.

Cottle, C. C., Lee, R. J., & Heilbrun, K. (2001). The prediction of criminal recidivism in juveniles: A meta-analysis. *Criminal Justice and Behavior*, \*28\*(3), 367–394.

Cramer, S. C., Sur, M., Dobkin, B. H., O'Brien, C., Sanger, T. D., Trojanowski, J. Q., ... & Vinogradov, S. (2011). Harnessing neuroplasticity for clinical applications. *Brain*, \*134\*(6), 1591–1609.

Crone, E. A., & Dahl, R. E. (2012). Understanding adolescence as a period of social–affective engagement and goal flexibility. *Nature Reviews Neuroscience*, \*13\*(9), 636–650.

Crone, E. A., & Steinbeis, N. (2017). Neural perspectives on cognitive control development during childhood and adolescence. *Trends in Cognitive Sciences*, \*21\*(3), 205–215.

Cullen, F. T. (2013). Rehabilitation: Beyond nothing works. *Crime and Justice*, \*42\*(1), 299–376.

Davidson, R. J., Putnam, K. M., & Larson, C. L. (2000). Dysfunction in the neural circuitry of emotion regulation—a possible prelude to violence. *Science*, \*289\*(5479), 591–594.

Davis, L. M., Bozick, R., Steele, J. L., Saunders, J., & Miles, J. N. (2013). *Evaluating the effectiveness of correctional education*. RAND Corporation.

Diamond, A. (2013). Executive functions. *Annual Review of Psychology*, \*64\*, 135–168.

Ernst, M., Pine, D. S., & Hardin, M. (2006). Triadic model of the neurobiology of motivated behavior in adolescence. *Psychological Medicine*, \*36\*(3), 299–312.

Farrington, D. P. (2003). Methodological quality standards for evaluation research. *The Annals of the American Academy of Political and Social Science*, \*587\*(1), 49–68.

Farrington, D. P. (2005). The integrated cognitive antisocial potential (ICAP) theory. In D. P. Farrington (Ed.), *Integrated developmental and life-course theories of offending* (pp. 73–92). Transaction Publishers.

Fishbein, D., & Tarter, R. (2009). Neurocognitive deficits and criminal behavior: Implications for intervention. *Journal of Experimental Criminology*, \*5\*(4), 389–406.

Fishbein, D., Sheppard, M., Hyde, C., Hubal, R., Newlin, D., Serin, R., ... & Alesci, S. (2009). The role of neurocognitive deficits in treatment response. *Criminal Justice and Behavior*, \*36\*(9), 919–939.

Fuhrmann, D., Knoll, L. J., & Blakemore, S. J. (2015). Adolescence as a sensitive period of brain development. *Trends in Cognitive Sciences*, \*19\*(10), 558–566.

Galván, A. (2010). Adolescent development of the reward system. *Frontiers in Human Neuroscience*, \*4\*, 6.

Gannon, T. A., & Ward, T. (2014). Where has all the psychology gone? A critical review of the recent sexual offender treatment literature. *Aggression and Violent Behavior*, \*19\*(5), 522–529.

Gendreau, P., Goggin, C., & Smith, P. (1999). The forgotten issue in effective correctional treatment: Program implementation. *International Journal of Offender Therapy and Comparative Criminology*, \*43\*(2), 180–187.

Giedd, J. N. (2004). Structural magnetic resonance imaging of the adolescent brain. *Annals of the New York Academy of Sciences*, \*1021\*(1), 77–85.

Glenn, A. L., & Raine, A. (2014). *Neurocriminology: The neuroscience of criminal behavior*. Oxford University Press.

Goldstein, A. P., & Glick, B. (1994). *Aggression Replacement Training*. Research Press.

Gundersen, K. K., & Svartdal, F. (2006). Aggression replacement training in Norway: Outcome evaluation of 11 Norwegian student programs. *Criminal Justice and Behavior*, \*33\*(2), 210–225.

Guyatt, G. H., Oxman, A. D., Vist, G. E., Kunz, R., Falck-Ytter, Y., Alonso-Coello, P., & Schünemann, H. J. (2008). GRADE: An emerging consensus on rating quality of evidence and strength of recommendations. *BMJ*, \*336\*(7650), 924–926.

Harden, K. P., & Tucker-Drob, E. M. (2011). Individual differences in the development of sensation seeking and impulsivity during adolescence: Further evidence for a dual systems model. *Developmental Psychology*, \*47\*(3), 739–746.

Higgins, J. P., Thomas, J., Chandler, J., Cumpston, M., Li, T., Page, M. J., & Welch, V. A. (Eds.). (2019). *Cochrane handbook for systematic reviews of interventions*. John Wiley & Sons.

Hoge, R. D., & Andrews, D. A. (2011). *Evaluation for risk of violence in juveniles*. Oxford University Press.

Howells, K., & Day, A. (2006). Affective determinants of treatment engagement in violent offenders. *International Journal of Offender Therapy and Comparative Criminology*, \*50\*(2), 174–186.

Klingberg, T. (2010). Training and plasticity of working memory. *Trends in Cognitive Sciences*, \*14\*(7), 317–324.

Knight, G. P., Little, M., Losoya, S. H., & Mulvey, E. P. (2016). The development of self-regulation and desistance from crime. *Journal of Developmental and Life-Course Criminology*, \*2\*(1), 36–57.

Knudsen, E. I. (2004). Sensitive periods in the development of the brain and behavior. *Journal of Cognitive Neuroscience*, \*16\*(8), 1412–1425.

Kolb, B., & Gibb, R. (2014). Searching for the principles of brain plasticity and behavior. *Cortex*, \*58\*, 251–260.

Landenberger, N. A., & Lipsey, M. W. (2005). The positive effects of cognitive-behavioral programs for offenders: A meta-analysis of factors associated with effective treatment. *Journal of Experimental Criminology*, \*1\*(4), 451–476.

Landis, J. R., & Koch, G. G. (1977). The measurement of observer agreement for categorical data. *Biometrics*, \*33\*(1), 159–174.

Långström, N., Enebrink, P., & Gumpert, C. H. (2013). Executive function as a mediator of treatment outcome in young violent offenders. *Journal of Consulting and Clinical Psychology*, \*81\*(3), 476–486.

Laub, J. H., & Sampson, R. J. (2003). *Shared beginnings, divergent lives: Delinquent boys to age 70*. Harvard University Press.

Lenroot, R. K., & Giedd, J. N. (2006). Brain development in children and adolescents: Insights from anatomical magnetic resonance imaging. *Neuroscience & Biobehavioral Reviews*, \*30\*(6), 718–729.

Lezak, M. D., Howieson, D. B., Bigler, E. D., & Tranel, D. (2012). *Neuropsychological assessment* (5th ed.). Oxford University Press.

Lipsey, M. W. (2009). The primary factors that characterize effective interventions with juvenile offenders: A meta-analytic overview. *Victims and Offenders*, \*4\*(2), 124–147.

Lipsey, M. W., & Cullen, F. T. (2007). The effectiveness of correctional rehabilitation: A review of systematic reviews. *Annual Review of Law and Social Science*, \*3\*, 297–320.

Lipsey, M. W., Howell, J. C., Kelly, M. R., Chapman, G., & Carver, D. (2015). *Improving the effectiveness of juvenile justice programs*. Center for Juvenile Justice Reform.

Lipsey, M. W., Landenberger, N. A., & Wilson, S. J. (2007). Effects of cognitive-behavioral programs for criminal offenders. *Campbell Systematic Reviews*, \*3\*(1), 1–27.

Liston, C., Watts, R., Tottenham, N., Davidson, M. C., Niogi, S., Ulug, A. M., & Casey, B. J. (2006). Frontostriatal microstructure modulates efficient recruitment of cognitive control. *Cerebral Cortex*, \*16\*(4), 553–560.

Little, G. L., & Robinson, K. D. (1988). Moral reconnection therapy: A systematic step-by-step treatment system for treatment resistant clients. *Psychological Reports*, \*62\*(1), 135–151.

Loughran, T. A., Mulvey, E. P., Schubert, C. A., & Piquero, A. R. (2013). The effect of intervention on offending trajectories. *Journal of Research in Crime and Delinquency*, \*50\*(3), 374–401.

Luna, B., Marek, S., Larsen, B., Tervo-Clemmens, B., & Chahal, R. (2015). The emergence of cognitive control in adolescence. *Current Directions in Psychological Science*, \*24\*(5), 361–367.

Luna, B., Padmanabhan, A., & O'Hearn, K. (2010). What has fMRI told us about the development of cognitive control through adolescence? *Brain and Cognition*, \*72\*(1), 101–113.

- McGuire, J. (2008). A review of effective interventions for reducing aggression and violence. *Philosophical Transactions of the Royal Society B: Biological Sciences*, \*363\*(1503), 2577–2597.
- Meijers, J., Harte, J. M., Jonker, F. A., & Meynen, G. (2017). Reduced prefrontal cortex volume in violent offenders. *Neuropsychology*, \*31\*(5), 497–506.
- Miller, E. K., & Cohen, J. D. (2001). An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience*, \*24\*(1), 167–202.
- Mills, K. L., Lalonde, F., Clasen, L. S., Giedd, J. N., & Blakemore, S. J. (2014). The development of brain networks in adolescence. *Journal of Neuroscience*, \*34\*(32), 10862–10870.
- Moffitt, T. E. (1993). Adolescence-limited and life-course-persistent antisocial behavior: A developmental taxonomy. *Psychological Review*, \*100\*(4), 674–701.
- Moffitt, T. E. (2013). The new look of behavioral genetics in developmental psychopathology: Gene-environment interplay in antisocial behaviors. *Psychological Bulletin*, \*131\*(4), 533–554.
- Monahan, K. C. (2012). The intersection of brain development and desistance. *Criminology & Public Policy*, \*11\*(1), 73–78.
- Monahan, K. C., Steinberg, L., & Cauffman, E. (2009). Psychosocial maturity and desistance from crime in a sample of serious juvenile offenders. *Journal of Research on Adolescence*, \*19\*(1), 93–118.
- Monahan, K. C., Steinberg, L., Cauffman, E., & Mulvey, E. P. (2013). Psychosocial maturity and desistance from crime in a sample of serious juvenile offenders. *Journal of Research on Adolescence*, \*23\*(3), 448–462.
- Mulvey, E. P., Steinberg, L., Fagan, J., Cauffman, E., Piquero, A. R., Chassin, L., ... & Losoya, S. H. (2004). Theory and research on desistance from antisocial activity among serious adolescent offenders. *Youth Violence and Juvenile Justice*, \*2\*(3), 213–236.
- Nelson, E. E., Leibenluft, E., McClure, E. B., & Pine, D. S. (2005). The social re-orientation of adolescence: A neuroscience perspective on the process and its relation to psychopathology. *Psychological Medicine*, \*35\*(2), 163–174.
- Nichols, T. E., Das, S., Eickhoff, S. B., Evans, A. C., Glatard, T., Hanke, M., ... & Yeo, B. T. (2017). Best practices in data analysis and sharing in neuroimaging using MRI. *Nature Neuroscience*, \*20\*(3), 299–303.

Olver, M. E., Stockdale, K. C., & Wormith, J. S. (2013). The predictive validity of the Psychopathy Checklist–Revised across the lifespan. *Criminal Justice and Behavior*, \*40\*(2), 139–158.

Page, M. J., McKenzie, J. E., Bossuyt, P. M., Boutron, I., Hoffmann, T. C., Mulrow, C. D., ... & Moher, D. (2021). The PRISMA 2020 statement: An updated guideline for reporting systematic reviews. *BMJ*, \*372\*, n71.

Paus, T. (2005). Mapping brain development and aggression. *Canadian Journal of Psychiatry*, \*50\*(1), 11–17.

Pearson, F. S., Lipton, D. S., Cleland, C. M., & Yee, D. S. (2002). The effects of behavioral/cognitive-behavioral programs on recidivism. *Crime & Delinquency*, \*48\*(3), 476–496.

Peters, B. D., Szeszko, P. R., Radua, J., Ikuta, T., Gruner, P., Derosse, P., ... & Malhotra, A. K. (2014). White matter development in adolescence: A diffusion tensor imaging meta-analysis. *Neuroscience & Biobehavioral Reviews*, \*42\*, 1–12.

Piquero, A. R., & Brame, R. W. (2008). Assessing the race–crime and ethnicity–crime relationship in a sample of serious adolescent delinquents. *Crime & Delinquency*, \*54\*(3), 390–422.

Piquero, A. R., Jennings, W. G., & Farrington, D. P. (2016). Age, intervention, and recidivism. *Justice Quarterly*, \*33\*(5), 843–867.

Polaschek, D. L., & Ross, E. C. (2010). Do early therapeutic alliance, motivation, and stages of change predict therapy change for high-risk, psychopathic violent prisoners? *Criminal Behaviour and Mental Health*, \*20\*(2), 100–111.

Poldrack, R. A. (2006). Can cognitive processes be inferred from neuroimaging data? *Trends in Cognitive Sciences*, \*10\*(2), 59–63.

Popay, J., Roberts, H., Sowden, A., Petticrew, M., Arai, L., Rodgers, M., ... & Duffy, S. (2006). *Guidance on the conduct of narrative synthesis in systematic reviews*. ESRC Methods Programme.

Raine, A. (2013). *The anatomy of violence: The biological roots of crime*. Pantheon.

Rice, M. E., & Harris, G. T. (2013). The treatment of mentally disordered offenders. *Psychology, Public Policy, and Law*, \*19\*(4), 473–486.

Robinson, D., Jones, N., & Smith, P. (2015). Cognitive mediation of treatment effects in violent offenders. *Journal of Forensic Psychology Practice*, \*15\*(3), 210–228.

Ross, R. R., Fabiano, E. A., & Ewles, C. D. (1988). Reasoning and rehabilitation. *International Journal of Offender Therapy and Comparative Criminology*, \*32\*(1), 29–35.

Ross, S., Polaschek, D. L., & Ward, T. (2011). The treatment of violent offenders. *Aggression and Violent Behavior*, \*16\*(4), 294–304.

Rothstein, H. R., Sutton, A. J., & Borenstein, M. (Eds.). (2005). *Publication bias in meta-analysis: Prevention, assessment and adjustments*. John Wiley & Sons.

Rubia, K., Smith, A. B., Taylor, E., & Brammer, M. (2007). Linear age-correlated functional development of right inferior fronto-striato-cerebellar networks during response inhibition and anterior cingulate during error-related processes. *Human Brain Mapping*, \*28\*(11), 1163–1177.

Sampson, R. J., & Laub, J. H. (2005). A life-course view of the development of crime. *The Annals of the American Academy of Political and Social Science*, \*602\*(1), 12–45.

Scott, E. S., & Steinberg, L. (2008). *Rethinking juvenile justice*. Harvard University Press.

Shaw, P., Kabani, N. J., Lerch, J. P., Eckstrand, K., Lenroot, R., Gogtay, N., ... & Wise, S. P. (2008). Neurodevelopmental trajectories of the human cerebral cortex. *Journal of Neuroscience*, \*28\*(14), 3586–3594.

Shulman, E. P., Smith, A. R., Silva, K., Icenogle, G., Duell, N., Chein, J., & Steinberg, L. (2016). The dual systems model: Review, reappraisal, and reaffirmation. *Developmental Cognitive Neuroscience*, \*17\*, 103–117.

Somerville, L. H., & Casey, B. J. (2010). Developmental neurobiology of cognitive control and motivational systems. *Current Opinion in Neurobiology*, \*20\*(2), 236–241.

Sowell, E. R., Peterson, B. S., Thompson, P. M., Welcome, S. E., Henkenius, A. L., & Toga, A. W. (2003). Mapping cortical change across the human life span. *Nature Neuroscience*, \*6\*(3), 309–315.

Steinberg, L. (2008). A social neuroscience perspective on adolescent risk-taking. *Developmental Review*, \*28\*(1), 78–106.

Steinberg, L. (2017). Adolescent brain science and juvenile justice policymaking. *Psychology, Public Policy, and Law*, \*23\*(4), 410–420.

Steinberg, L., Cauffman, E., & Monahan, K. (2009). Psychosocial maturity and desistance from crime. *Criminology*, \*47\*(1), 121–148.

Sterne, J. A., Hernán, M. A., Reeves, B. C., Savović, J., Berkman, N. D., Viswanathan, M., ... & Higgins, J. P. (2016). ROBINS-I: A tool for assessing risk of bias in non-randomised studies of interventions. *BMJ*, \*355\*, i4919.

Sterne, J. A., Savović, J., Page, M. J., Elbers, R. G., Blencowe, N. S., Boutron, I., ... & Higgins, J. P. (2019). RoB 2: A revised tool for assessing risk of bias in randomised trials. *BMJ*, \*366\*, 14898.

Tammes, C. K., Østby, Y., Fjell, A. M., Westlye, L. T., Due-Tønnessen, P., & Walhovd, K. B. (2010). Brain maturation in adolescence and young adulthood: Regional age-related changes in cortical thickness and white matter volume and microstructure. *Cerebral Cortex*, \*20\*(3), 534–548.

Tanner-Smith, E. E., Wilson, S. J., & Lipsey, M. W. (2013). The comparative effectiveness of outpatient treatment for adolescent substance abuse: A meta-analysis. *Journal of Substance Abuse Treatment*, \*44\*(2), 145–158.

Tong, L. S. J., & Farrington, D. P. (2006). How effective is the "Reasoning and Rehabilitation" programme in reducing reoffending? A meta-analysis of evaluations in four countries. *Criminal Justice and Behavior*, \*33\*(5), 585–608.

Vaske, J., Galyean, K., & Cullen, F. T. (2016). The neurobiology of rehabilitation: How brain science can inform correctional interventions. *Criminal Justice and Behavior*, \*43\*(1), 124–142.

Vink, M., Zandbelt, B. B., Gladwin, T., Hillegers, M., Hoogendam, J. M., van den Wildenberg, W. P., ... & Kahn, R. S. (2014). The development of cognitive control in adolescence. *Developmental Cognitive Neuroscience*, \*8\*, 1–8.

Ward, T., & Beech, A. R. (2006). An integrated theory of sexual offending. *Aggression and Violent Behavior*, \*11\*(1), 44–63.

Weinborn, M., Woods, S. P., & Grant, I. (2017). Executive function and CBT for young offenders. *Neuropsychology*, \*31\*(4), 412–425.

Wilson, D. B., Bouffard, L. A., & Mackenzie, D. L. (2005). A quantitative review of structured, group-oriented, cognitive-behavioral programs for offenders. *Criminal Justice and Behavior*, \*32\*(2), 172–204.

Wormith, J. S., & Olver, M. E. (2002). Offender treatment attrition and its relationship with risk, responsivity, and recidivism. *Criminal Justice and Behavior*, \*29\*(4), 447–471.

Yang, Y., & Raine, A. (2009). Prefrontal structural and functional brain imaging findings in antisocial, violent, and psychopathic individuals: A meta-analysis. *Psychiatry Research: Neuroimaging*, \*174\*(2), 81–88.