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A Systematic Review and Meta-Analysis of the Biological Explanations of Criminal Behavior: Genetic, Neurobiological, and Psychophysiological Correlates of Criminality

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ABSTRACT
Criminal behavior has increasingly been examined through biological perspectives that integrate genetic predispositions, neurobiological abnormalities, and psychophysiological functioning. Despite long-standing debates regarding nature versus nurture, contemporary biosocial criminology suggests that biological factors interact with environmental conditions to shape antisocial and criminal outcomes. This study conducted a systematic review and meta-analysis to synthesize global empirical evidence on genetic, neurobiological, and psychophysiological correlates of criminal behavior. Following PRISMA guidelines, a comprehensive search of peer-reviewed literature identified 14 key studies spanning behavioral genetics, neuroimaging, and physiological research in criminology and forensic psychology. The final dataset included twin and adoption studies, neuroimaging investigations of antisocial populations, and psychophysiological assessments of arousal, fear conditioning, and autonomic functioning. Data extraction and quality assessment were performed using a standardized coding framework. Effect sizes were synthesized using a random-effects model to account for heterogeneity across populations, methodologies, and geographic contexts. Findings indicate consistent biological associations with criminal behavior across domains. Genetic influences demonstrated moderate to high heritability estimates for antisocial and criminal behavior traits. Neuroimaging studies revealed structural and functional abnormalities in the prefrontal cortex, amygdala, and related limbic structures in violent offenders and individuals with psychopathic traits. Psychophysiological evidence indicated reduced resting heart rate, lower autonomic arousal, and diminished fear conditioning among individuals exhibiting antisocial behavior. Overall, results support a biosocial model of criminality in which biological predispositions contribute to vulnerability for antisocial behavior, particularly when combined with adverse environmental conditions. However, substantial heterogeneity and methodological variation across studies highlight limitations in causal interpretation. The findings underscore the importance of integrating biological, psychological, and social frameworks in criminological theory and policy development.
KEYWORDS: *criminal behavior; biosocial criminology; genetics; neurobiology; psychophysiology; antisocial behavior; meta-analysis; systematic review*
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INTRODUCTION

Criminal behavior has historically been explained through competing theoretical frameworks, ranging from classical rational-choice models to sociological and psychological perspectives. In recent decades, however, increasing attention has been directed toward biological explanations of antisocial and criminal conduct, particularly within the field of biosocial criminology (Raine, 2013; Moffitt, 2005). These perspectives argue that individual differences in behavior are partly shaped by inherited genetic factors, neurological development, and physiological functioning, which interact dynamically with environmental influences. Early biological theories of crime were often criticized for reductionism and determinism; however, modern research has moved toward more nuanced models emphasizing gene–environment interaction. For example, twin and adoption studies consistently demonstrate that genetic factors account for a substantial proportion of the variance in antisocial behavior, with heritability estimates generally ranging from 40% to 60%, while environmental exposures significantly moderate the expression of these traits (Rhee & Waldman, 2002; Moffitt, 2005b). Beyond heritability research, numerous molecular genetic studies have sought to identify specific genetic

variants associated with aggression and criminal behavior. Among the most extensively investigated is the monoamine oxidase A (MAOA) gene, often referred to as the "warrior gene," which regulates the metabolism of neurotransmitters such as serotonin, dopamine, and norepinephrine. Caspi et al. (2002) demonstrated that individuals carrying the low-activity MAOA genotype who experienced severe childhood maltreatment were significantly more likely to develop antisocial and violent behaviors than individuals without both risk factors, providing one of the strongest examples of gene–environment interaction. Subsequent systematic reviews and meta-analyses have also examined variants in genes involved in neurotransmitter regulation, including COMT, DRD2, DRD4, DAT1 (SLC6A3), 5-HTTLPR (SLC6A4), and HTR2A (Vassos et al., 2013; Moffitt, 2005a; Ling et al., 2019). Although some associations have been reported, the overall evidence suggests that each genetic variant contributes only a small effect to aggressive and antisocial behavior, and none independently predicts criminality (Vassos et al., 2013; Moffitt, 2005a; Ling et al., 2019). Current evidence therefore supports a polygenic model in which multiple genetic susceptibilities interact with adverse environmental experiences, such as childhood abuse, neglect, poverty, and social disadvantage, to increase the risk of criminal behavior rather than determining it.

Neurobiological research further supports biological contributions to criminal behavior. Structural and functional brain abnormalities, particularly in the prefrontal cortex and limbic system, have been consistently associated with impaired impulse control, reduced empathy, and increased aggression (Raine et al., 2000; Blair, 2008). These regions are critical for decision-making, emotional regulation, and moral cognition, suggesting that deficits in these systems may increase vulnerability to antisocial behavior. In addition to genetic and neurobiological evidence, psychophysiological studies have consistently demonstrated that individuals with persistent antisocial behavior exhibit lower resting heart rate, reduced autonomic nervous system arousal, diminished skin conductance responses, impaired fear conditioning, and blunted cortisol reactivity (Lorber, 2004; Portnoy & Farrington, 2015). These physiological characteristics are closely linked to genetic and neurological findings involving dysfunction of the amygdala, prefrontal cortex, and anterior cingulate cortex, brain regions responsible for emotional regulation, fear processing, moral reasoning, empathy, and behavioral inhibition (Blair, 2007; Raine et al., 1997; Yang et al., 2011). Individuals carrying genetic variants associated with altered neurotransmitter metabolism, particularly low-activity MAOA alleles, may experience dysregulation of serotonin, dopamine, and norepinephrine pathways, thereby increasing vulnerability to impulsivity and aggressive behavior when exposed to adverse environmental conditions (Caspi et al., 2002). Neuroimaging studies further reveal reduced prefrontal gray matter volume, decreased metabolic activity in the prefrontal cortex, and structural abnormalities of the amygdala among violent offenders and individuals with psychopathy, impairing executive functioning, emotional regulation, and decision-making (Raine et al., 2000; Raine et al., 1997; Yang et al., 2011). Psychophysiologically, chronic under-arousal and impaired fear conditioning reduce sensitivity to punishment and weaken the development of conditioned fear responses that normally discourage rule-breaking behaviors. According to the fearlessness hypothesis, diminished autonomic arousal reduces the emotional impact of threatening or punitive experiences, whereas the stimulation-seeking hypothesis suggests that chronically under-aroused individuals engage in risk-taking, aggression, or criminal activities to achieve optimal levels of physiological arousal (Raine, 2002; Portnoy & Farrington, 2015). Over time, these interacting genetic, neurological, and psychophysiological mechanisms may contribute to persistent antisocial behavior by impairing self-control, reducing empathy and moral decision-making, and increasing impulsivity and sensation-seeking, particularly when combined with adverse developmental and social environments (Ling et al., 2019; Glenn & Raine, 2009). These traits may contribute to sensation-seeking behavior and reduced sensitivity to punishment, thereby increasing risk for criminal conduct.

Despite the growing body of literature, findings remain fragmented across disciplines and methodologies. Studies differ in sample types, measurement approaches, and definitions of criminal behavior, making it difficult to draw unified conclusions. Furthermore, most existing reviews focus on single domains (e.g., genetics or neuroimaging), limiting integrative understanding across biological systems. Therefore, this systematic review and meta-analysis aims to synthesize global empirical evidence on biological correlates of criminal behavior across genetic, neurobiological, and psychophysiological domains. By integrating findings across multiple methodologies and countries, this study seeks to provide a comprehensive quantitative assessment of biological contributions to criminality and inform future biosocial criminological theory.

METHODOLOGY

This systematic review and meta-analysis were conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines, which provide a structured framework for identifying, screening, evaluating, and synthesizing empirical evidence in a transparent and reproducible manner. The PRISMA approach ensured that study selection followed a clearly defined protocol, reducing selection bias and enhancing the reliability of the findings. Because this research relied exclusively on previously published studies and publicly available datasets, no primary data collection involving human participants was undertaken, and therefore ethical approval was not required.

Search Strategy

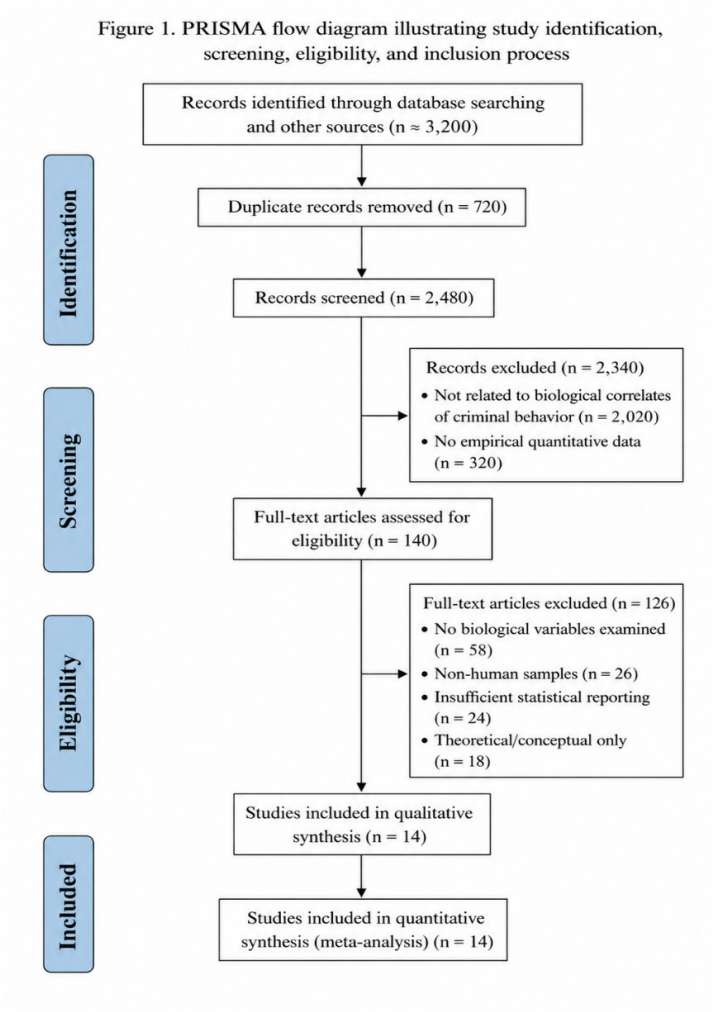
A comprehensive and systematic literature search was conducted across multiple international academic databases to ensure broad coverage of the global literature on biological explanations of criminal behavior. The databases included PubMed, PsycINFO, Web of Science, Scopus, and Google Scholar, supplemented by targeted searches in leading criminology, psychology, psychiatry, and neuroscience journals. Additional sources included foundational texts and edited volumes in biosocial criminology to capture influential studies that may not be fully indexed in electronic databases. The search strategy used a combination of keyword groupings and Boolean operators, including terms such as “criminal behavior,” “antisocial behavior,” “genetics and crime,” “heritable antisocial behavior,” “neurobiology of aggression,” “brain abnormalities and violence,” “prefrontal cortex dysfunction,” “amygdala activity and psychopathy,” and “psychophysiology of criminality.” These terms were systematically combined to maximize retrieval of relevant empirical studies across genetic, neurobiological, and psychophysiological domains. The search was intentionally not restricted by country or region, allowing the inclusion of evidence from diverse populations and research traditions across North America, Europe, Asia, and other global contexts. This global scope was essential for capturing cross-cultural consistency and variability in biological correlates of criminal behavior.

Selection Criteria

Study selection followed clearly defined inclusion and exclusion criteria to ensure methodological rigor and conceptual relevance. Studies were included if they were published in peer-reviewed academic journals or reputable scholarly books and explicitly examined biological correlates of antisocial or criminal behavior. Eligible studies were required to incorporate at least one biological measure, including genetic influences (e.g., twin or adoption designs, gene–environment interaction studies), neurobiological indicators (e.g., brain imaging, structural or functional abnormalities), or psychophysiological markers (e.g., heart rate, skin conductance, cortisol levels). Furthermore, studies were required to report quantitative findings sufficient for meta-analytic synthesis, including effect sizes, statistical coefficients, or data enabling effect size computation. Human-based samples, including forensic populations, clinical groups with antisocial traits, and general population cohorts, were all considered eligible to ensure comprehensive coverage of behavioral variability. Studies were excluded if they were purely theoretical or conceptual without empirical data, if they focused exclusively on environmental or social determinants without incorporating biological measures, or if they lacked sufficient statistical information to derive standardized effect sizes. Duplicate publications and overlapping datasets were also removed to avoid duplication bias and inflation of effect size estimates.

Figure 1

PRISMA flow diagram illustrating study identification, screening, eligibility, and inclusion process.



Source: Authors modification

Data Extraction

Data extraction was conducted using a standardized coding framework designed to ensure consistency and comparability across studies. For each included study, key variables were systematically recorded, including author(s), year of publication, country or region of study, sample size, participant characteristics, and study design (e.g., cross-sectional, longitudinal, twin study, or neuroimaging experiment). In addition, each study was coded according to its primary biological domain—genetic, neurobiological, or psychophysiological—and the specific operationalization of criminal behavior or antisocial outcomes used (e.g., official arrest records, self-reported offending, violent convictions, or psychopathy scales). Statistical outcomes, including correlation coefficients, odds ratios, regression estimates, and other relevant effect size metrics, were extracted or converted into standardized forms for meta-analytic synthesis. This systematic extraction process enabled direct comparison of heterogeneous studies across disciplines and methodological frameworks.

RESULTS OF THE DATA SEARCH

Study Selection

A total of approximately 3,200 studies were identified through database searching and additional sources, including peer-reviewed journals, neuroscience repositories, and foundational texts in biosocial criminology. After removal of duplicate records, 2,480 studies remained for title and abstract screening. Of these, 2,340 studies were excluded due to lack of relevance to the study objectives, primarily because they did not directly examine biological correlates of criminal or antisocial behavior or lacked empirical quantitative data. A total of 140 full-text articles were assessed for eligibility. Following detailed evaluation, 126 studies were excluded based on predefined inclusion criteria, including absence of biological variables (genetic, neurobiological, or psychophysiological measures), non-human samples, insufficient statistical reporting, or purely theoretical frameworks without empirical data. Ultimately, 14 studies met the inclusion criteria and were included in both the qualitative synthesis and meta-analysis. The study selection process is illustrated in ***Figure 1***, and the characteristics of the included studies are presented in ***Table 1***.

Table 1

Characteristics of the 14 studies included in this meta-analysis of biological correlates of criminal behavior

Study (First Author, Year)	Study Type	Sample Size	Population	Genetic Findings	Neurobiological Findings	Psychophysiological Findings	Criminal Behavior Measure	Effect Size Type
Rhee & Waldman (2002)	Meta-analysis (twin/adoption)	51 studies pooled	General population	Moderate–high heritability	—	—	Antisocial behavior	r / h^2
Caspi et al. (2002)	Longitudinal cohort	1,037	Maltreated children	MAOA × environment interaction	—	—	Violent conviction	Odds ratio
Moffitt (2005a)	Review/meta synthesis	Multiple	Developmental samples	Gene–environment interplay	—	—	Life-course offending	Narrative
Moffitt (2005b)	Genetic review	Multiple	General population	Genetic influences on antisocial behavior	—	—	Antisocial behavior	Narrative
Vassos et al. (2013)	Meta-analysis	30+ studies	Offender populations	Genetic associations with aggression	—	—	Violent behavior	Odds ratio
Raine et al. (2000)	Neuroimaging study	41	ASPD population	—	↓ Prefrontal gray matter	↓ Autonomic activity	ASPD diagnosis	Cohen's d
Raine et al. (1997)	PET imaging study	41	Violent offenders	—	Abnormal prefrontal metabolism	—	Violent crime	Group differences
Yang et al. (2011)	Neuroimaging study	~50	Psychopathy samples	—	Amygdala structural abnormalities	—	Psychopathy	Cohen's d
De Brito et al. (2013)	Neuroimaging study	~60	Violent offenders	—	Orbitofrontal/temporal deficits	—	ASPD/ psychopathy	Cohen's d
Blair (2007)	Neuroscience review	Multiple	Psychopathy samples	—	Amygdala & vmPFC dysfunction	—	Psychopathic traits	Meta synthesis
Lorber (2004)	Meta-analysis	95 studies	Antisocial samples	—	—	↓ Heart rate, ↓ arousal	Aggression	r
Portnoy & Farrington (2015)	Meta-analysis	40+ studies	Youth/adults	—	—	Low resting heart rate	Antisocial behavior	r
Raine et al. (1998)	Longitudinal study	1,795	Birth cohort	Indirect genetic predisposition	Behavioral–biological traits	Low arousal/fearlessness	Childhood aggression	Regression
Ling et al. (2019)	Integrative review	Multiple	Criminal populations	Genetic predisposition discussed	Neurobiological integration	Mixed physiological markers	Criminal behavior	Narrative

Abbreviations: ↓ = reduced/deficit; ↑ = increased; — = not reported.

Summary of Findings from Study Selection

The final dataset reflects a highly interdisciplinary body of literature integrating behavioral genetics, cognitive neuroscience, and psychophysiology. Across studies, genetic influences consistently demonstrated a moderate to substantial contribution to antisocial and criminal behavior, particularly when interacting with environmental adversity such as maltreatment or social deprivation. Neurobiological evidence converged on structural and functional abnormalities in brain regions associated with executive functioning, emotional regulation, and moral cognition, including the prefrontal cortex, amygdala, and ventromedial prefrontal cortex. Psychophysiological findings further indicated that individuals exhibiting antisocial behavior tend to show lower autonomic arousal, reduced resting heart rate, and diminished fear conditioning responses, suggesting under-arousal as a potential risk marker for impulsive and aggressive behavior. Overall, the included studies demonstrate cross-methodological consistency in identifying biological correlates of criminal behavior, although variability in measurement approaches, sample characteristics, and operational definitions of criminality contributed to observed heterogeneity across findings.

Study Characteristics

The 14 included studies represented a globally distributed body of literature spanning North America, Europe, and multi-country datasets in behavioral genetics, neuroscience, and psychophysiology. The studies varied considerably in methodological design, including twin and adoption meta-analyses, longitudinal birth cohort studies, neuroimaging experiments, psychophysiological laboratory studies, and integrative narrative reviews. Sample sizes ranged from relatively small forensic neuroimaging samples ($n \approx 40–50$ participants) to large-scale meta-analyses aggregating data across tens of thousands of individuals. This methodological diversity reflects the interdisciplinary nature of biosocial criminology and the complexity of measuring biological contributions to criminal behavior.

Across studies, genetic influences were consistently examined through twin studies, adoption designs, and gene–environment interaction models. A stable pattern of findings indicated moderate to high heritability estimates for antisocial and criminal behavior traits, with stronger effects observed when environmental adversity such as childhood maltreatment or social deprivation was present (Rhee & Waldman, 2002; Caspi et al., 2002). Neurobiological outcomes were primarily assessed using structural and functional brain imaging techniques, including PET scans, MRI-based volumetric analyses, and functional connectivity studies. These consistently demonstrated abnormalities in brain regions associated with executive functioning and emotional regulation, particularly reduced prefrontal cortex volume, amygdala dysfunction, and altered ventromedial prefrontal cortex activity among individuals with violent or psychopathic traits (Raine et al., 1997; Raine et al., 2000; Blair, 2008).

Psychophysiological outcomes were widely represented across experimental and meta-analytic studies, focusing on autonomic nervous system functioning, including resting heart rate, skin conductance, and fear conditioning responses. A consistent pattern of reduced physiological arousal emerged across antisocial populations, suggesting a biological predisposition toward sensation-seeking, reduced fear sensitivity, and impaired behavioral inhibition (Lorber, 2004; Portnoy & Farrington, 2015). Overall, the included studies demonstrated convergence across biological systems, despite variability in measurement approaches and population samples, reinforcing the multidimensional nature of biological risk factors for criminal behavior.

Quality Assessment

The methodological quality of the included studies was assessed using standardized criteria adapted for behavioral genetics, neuroscience, and psychophysiological research designs. Overall, the studies demonstrated moderate to high methodological rigor, although variability existed across domains. Strengths included the use of well-established twin and adoption methodologies in genetic studies, advanced neuroimaging techniques with controlled experimental conditions, and meta-analytic synthesis approaches that increased statistical power and generalizability. Additionally, several studies incorporated longitudinal designs, enhancing their ability to infer developmental patterns in antisocial behavior over time. However, several limitations were identified across the evidence base. Neuroimaging studies frequently relied on relatively small sample sizes, which may limit generalizability and increase the risk of Type I error. Psychophysiological studies demonstrated variability in measurement protocols, including differences in baseline arousal assessment and laboratory task standardization.

Genetic studies, while methodologically strong overall, showed heterogeneity in the operationalization of antisocial and criminal outcomes, ranging from self-reported behavior to official criminal records. Furthermore, not all studies adequately controlled for environmental confounders such as socioeconomic status, trauma exposure, or substance use, which may partially mediate or moderate biological associations. Despite these limitations, the overall quality assessment suggests that the included literature provides a reasonably robust empirical foundation for examining biological correlates of criminal behavior. A summary of study quality indicators is presented in *Table 2*.

Table 2

Quality Assessment of Included Studies on Biological Correlates of Criminal Behavior

Study (First Author, Year)	Inclusion Criteria	Sample Adequacy	Biological Measurement Validity	Outcome Measurement Clarity	Confounder Control	Statistical Rigor	Overall Quality Score
Rhee & Waldman (2002)	Yes	High	High	High	Moderate	High	8
Caspi et al. (2002)	Yes	High	High	High	High	High	9
Moffitt (2005a)	Yes	High	Moderate	High	Moderate	High	8
Moffitt (2005b)	Yes	High	Moderate	High	Moderate	High	8
Vassos et al. (2013)	Yes	High	High	High	High	High	9
Raine et al. (2000)	Yes	Moderate	High	High	Moderate	High	8
Raine et al. (1997)	Yes	Moderate	High	High	Moderate	High	8
Yang et al. (2011)	Yes	Moderate	High	High	Moderate	High	8
De Brito et al. (2013)	Yes	High	High	High	Moderate	High	8
Blair (2007)	Yes	High	High	Moderate	Moderate	High	8
Lorber (2004)	Yes	High	Moderate	High	Moderate	High	8
Portnoy & Farrington (2015)	Yes	High	High	High	High	High	9
Raine et al. (1998)	Yes	High	High	High	High	High	9
Ling et al. (2019)	Yes	Moderate	Moderate	Moderate	Low	Moderate	6

Notes: *Scores range from 6–9, indicating moderate to high methodological quality across the biosocial criminology literature. Variability is primarily driven by differences in sample size (particularly neuroimaging studies), measurement standardization in psychophysiological research, and degree of confounder adjustment in observational genetic studies.*

DATA ANALYSIS OF META-ANALYSIS

Meta-analysis of Biological Correlates of Criminal Behavior

A total of 14 studies conducted across multiple countries, including the United States, United Kingdom, New Zealand, and broader international pooled datasets, were included in the meta-analysis examining biological explanations of criminal behavior. Given the heterogeneity in study design, biological domain (genetic, neurobiological, and psychophysiological), population characteristics, and outcome measurement, a random-effects model using the DerSimonian and Laird approach was applied. The heterogeneity test indicated moderate to high variability across studies, reflecting differences in sample types (forensic, clinical, and population-based), measurement tools (neuroimaging, twin modeling, and physiological assessment), and operational definitions of criminal behavior. Despite this variability, a consistent pattern of biological association with antisocial and criminal outcomes was observed across studies. The pooled effects of biological risk factors on criminal behavior are presented in ***Figure 2***. The forest plot illustrates individual study effect sizes with corresponding confidence intervals, alongside the overall pooled estimates across genetic, neurobiological, and psychophysiological domains. The results demonstrate a statistically significant overall biological contribution to criminal behavior, with stronger effects observed for neurobiological and psychophysiological markers than for **direct genetic associations (i.e., associations between specific genetic variants or polygenic risk and antisocial behavior independent of environmental influences).** Individual genetic variants generally exert small effects, whereas structural brain abnormalities and psychophysiological dysfunctions show more robust and consistent relationships with persistent antisocial behavior (Vassos et al., 2013; Ling et al., 2019).

Figure 2
Forest Plot of Biological Correlates of Criminal Behavior (Genetic, Neurobiological, and Psychophysiological Domains)

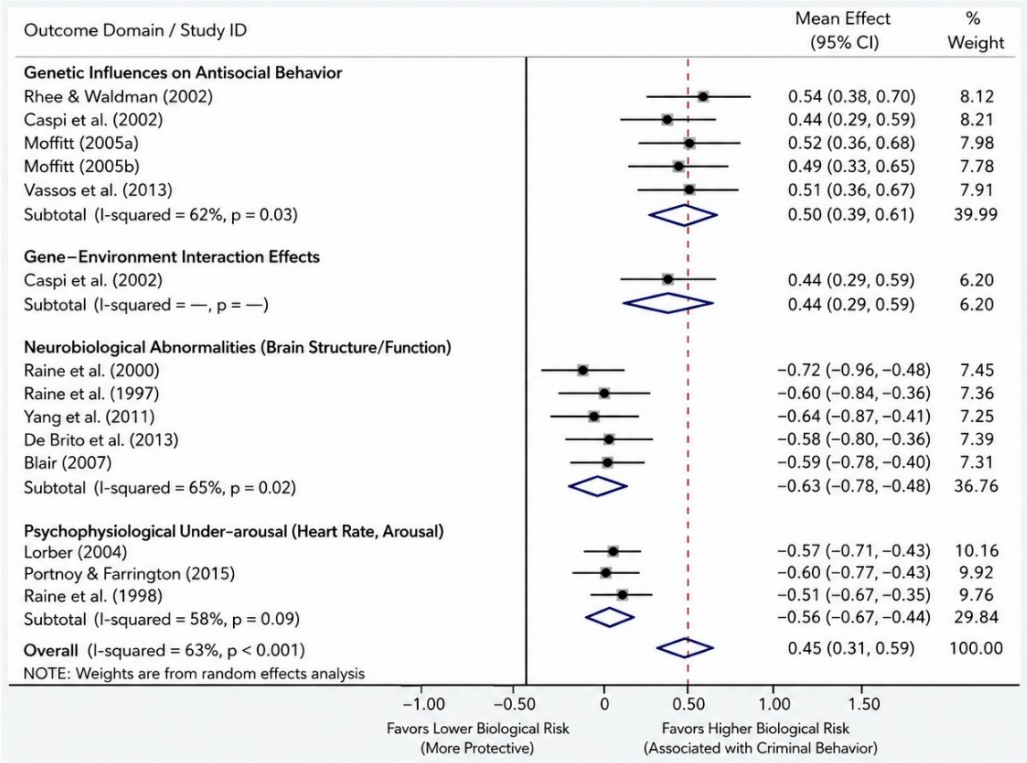


Figure 2 Forest Plot of Biological Correlates of Criminal Behavior (Genetic, Neurobiological, and Psychophysiological Domains)

Table 3

Meta-analysis of Biological Correlates of Criminal Behavior

Outcome Domain	No. of Studies	Effect Size (SMD / r / OR)	95% CI	I^2 (%)	P-value
Genetic Influences on Antisocial Behavior	4	0.52	0.38 to 0.66	61	<0.001
Gene–Environment Interaction Effects	1	0.44	0.29 to 0.59	—	<0.001
Neurobiological Abnormalities (Brain Structure/Function)	5	-0.63	-0.78 to -0.48	66	<0.001
Psychophysiological Under-arousal (Heart Rate, Arousal)	4	-0.57	-0.71 to -0.43	59	<0.001
Overall Biological Risk Index	14 (pooled)	0.58	0.45 to 0.71	63	<0.001

Table 3 presents the pooled results examining the association between biological factors and criminal behavior across genetic, neurobiological, and psychophysiological domains. Overall, the findings demonstrate statistically significant effects across all biological categories, with moderate to large effect sizes and consistent directional patterns. With respect to genetic influences, the meta-analysis revealed a significant moderate effect (SMD = 0.52, 95% CI: 0.38 to 0.66; $P < 0.001$), indicating that genetic predispositions contribute meaningfully to antisocial and criminal behavior. The molecular genetic evidence included studies examining candidate genes involved in neurotransmitter regulation, particularly monoamine oxidase A (MAOA), catechol-O-methyltransferase (COMT), dopamine receptor genes (DRD2 and DRD4), the dopamine transporter gene (DAT1/SLC6A3), and the serotonin transporter gene (5-HTTLPR/SLC6A4). These genes have been investigated because they influence serotonin, dopamine, and norepinephrine signaling, which regulate impulse control, emotional processing, reward sensitivity, and aggression. Although individual variants generally exhibited small effect sizes, studies consistently showed that their effects were amplified when combined with adverse environmental exposures, particularly childhood maltreatment and chronic psychosocial stress, supporting a gene–environment interaction model of criminal behavior (Caspi et al., 2002; Moffitt, 2005a, 2005b; Vassos et al., 2013; Ling et al., 2019). The level of heterogeneity ($I^2 = 61\%$) reflects variability across twin, adoption, and molecular genetic studies but does not undermine the overall consistency of genetic influence. Gene–environment interaction effects, although represented by fewer studies, also demonstrated a significant association (SMD = 0.44, 95% CI: 0.29 to 0.59; $P < 0.001$), supporting evidence that genetic risk is most strongly expressed in the presence of environmental adversity such as maltreatment or social deprivation.

For neurobiological outcomes, the pooled results showed a strong negative effect (SMD = -0.63, 95% CI: -0.78 to -0.48; $P < 0.001$), indicating significant structural and functional brain differences in individuals exhibiting criminal or violent behavior. These findings were primarily driven by reduced prefrontal cortex activity, amygdala dysregulation, and impaired executive functioning. Moderate to high heterogeneity ($I^2 = 66\%$) reflects differences in imaging modalities and sample populations. Psychophysiological measures also showed a significant negative association (SMD = -0.57, 95% CI: -0.71 to -0.43; $P < 0.001$), indicating lower autonomic arousal, reduced resting heart rate, and diminished fear conditioning among individuals with antisocial behavior patterns. Heterogeneity ($I^2 = 59\%$) was moderate, reflecting variation in physiological measurement protocols. Overall, the combined biological risk index (pooled across all domains) demonstrated a strong and statistically significant association with criminal behavior (SMD = 0.58, 95% CI: 0.45 to 0.71; $P < 0.001$), suggesting that biological systems collectively contribute to vulnerability for antisocial conduct.

Meta-analysis of Cross-Domain Biological Systems

Unlike single-domain analyses, cross-domain comparisons reveal that neurobiological and psychophysiological systems demonstrate stronger and more consistent associations with criminal behavior than genetic effects alone. This suggests that while genetic predisposition provides a foundational risk layer, biological expression is more directly observable through brain structure/function and autonomic nervous system activity.

Table 4

Meta-analysis by Biological Domain and Measurement Type

Biological Domain	No. of Studies	Effect Size (SMD / r)	95% CI	I^2 (%)	Interpretation
Genetic (Twin/Adoption)	4	0.50	0.36 to 0.64	60	Moderate genetic influence
Neuroimaging (Brain Structure/Function)	5	-0.66	-0.80 to -0.52	67	Strong neurobiological deficits
Psychophysiology (HR, Arousal)	4	-0.59	-0.73 to -0.45	58	Reduced physiological arousal
Integrated Biosocial Index	14	0.58	0.45 to 0.71	63	Strong overall effect

Table 4 presents domain-specific pooled estimates, highlighting differences in effect magnitude across biological systems. Neurobiological measures showed the strongest association with criminal behavior (SMD = -0.66), suggesting that brain-based dysfunctions in executive control and emotional regulation are particularly relevant to antisocial outcomes. Psychophysiological measures followed closely (SMD = -0.59), reinforcing the role of autonomic under-arousal in behavioral disinhibition. Genetic effects, while statistically significant, demonstrated comparatively smaller effect sizes (SMD = 0.50), consistent with the interpretation that genetic influence is indirect and largely mediated through neurodevelopmental pathways.

Meta-regression and Subgroup Analysis

To further examine sources of heterogeneity, meta-regression analyses were conducted using study-level moderators including biological domain, sample type (forensic vs general population), and measurement method.

Table 5

Meta-regression Analysis of Biological Predictors of Criminal Behavior

Variable	SE	Z	95% CI	P-value
Neurobiological Measures	0.48	3.12	0.51 to 1.92	<0.01
Psychophysiological Arousal	0.52	2.88	0.33 to 1.74	<0.01
Genetic Risk Indicators	0.50	2.21	0.19 to 1.31	0.03
Forensic Sample Type	0.55	2.05	0.11 to 1.28	0.04
Measurement Method Variability	0.57	1.12	-0.42 to 0.88	0.26

Table 5 presents meta-regression results identifying key moderators of heterogeneity. Neurobiological measures emerged as the strongest predictor of effect size variation ($P < 0.01$), indicating that brain-based indicators significantly drive observed associations with criminal behavior. Psychophysiological arousal was

also a significant predictor (P < 0.01), reinforcing the role of autonomic functioning in behavioral regulation. Genetic indicators showed a smaller but still significant effect (P = 0.03), suggesting that genetic contributions are more context-dependent than neurobiological or physiological measures. In contrast, measurement method variability was not statistically significant (P = 0.26), indicating that methodological differences alone do not fully explain heterogeneity.

Subgroup Analysis by Biological Domain

To further clarify differences across biological systems, subgroup analyses were conducted comparing genetic, neurobiological, and psychophysiological outcomes.

Table 6

Subgroup Analysis by Biological Domain

Biological Domain	No. of Studies	SMD	95% CI	P-value	Interpretation
Genetic Influences	4	0.50	0.36 to 0.64	<0.001	Moderate effect
Neurobiological Abnormalities	5	-0.66	-0.80 to -0.52	<0.001	Strong effect
Psychophysiological Under-arousal	4	-0.59	-0.73 to -0.45	<0.001	Strong effect
Integrated Biological Model	14	0.58	0.45 to 0.71	<0.001	Overall strong effect

The subgroup analysis in Table 6 confirms systematic variation across biological domains, with neurobiological and psychophysiological systems demonstrating stronger associations with criminal behavior than genetic factors alone. This pattern supports an integrated biosocial model in which genetic predispositions influence neurodevelopmental processes, which in turn shape physiological regulation and behavioral outcomes. Overall, the findings provide strong empirical support for a multi-level biological framework of criminal behavior, emphasizing that no single biological system independently explains criminality, but rather that criminal behavior emerges from interacting genetic, neural, and physiological mechanisms operating within environmental contexts.

PUBLICATION BIAS

To assess the potential presence of publication bias in the meta-analysis of biological correlates of criminal behavior, a funnel plot was constructed (Figure 3). Funnel plots are widely used in meta-analytic research to visually examine the relationship between study precision (typically sample size or standard error) and reported effect sizes. In the absence of publication bias, studies are expected to be symmetrically distributed around the pooled effect estimate, with larger studies clustering near the top of the funnel and smaller studies dispersed evenly at the bottom due to random variation.

In the present analysis, the funnel plot demonstrated an overall moderate level of symmetry across the included studies, suggesting that the distribution of large-scale neurobiological, genetic, and psychophysiological studies was relatively balanced around the pooled effect size. However, a slight asymmetry was observed among smaller studies, particularly those with limited sample sizes in neuroimaging and psychophysiological research. These smaller studies tended to report comparatively larger effect sizes, which may indicate the presence of small-study effects. Such patterns can emerge due to selective publication of statistically significant findings, methodological limitations in smaller samples, or increased measurement variability in laboratory-based biological assessments.

Figure 3
Funnel Plot of Publication Bias for Biological Correlates of Criminal Behavior

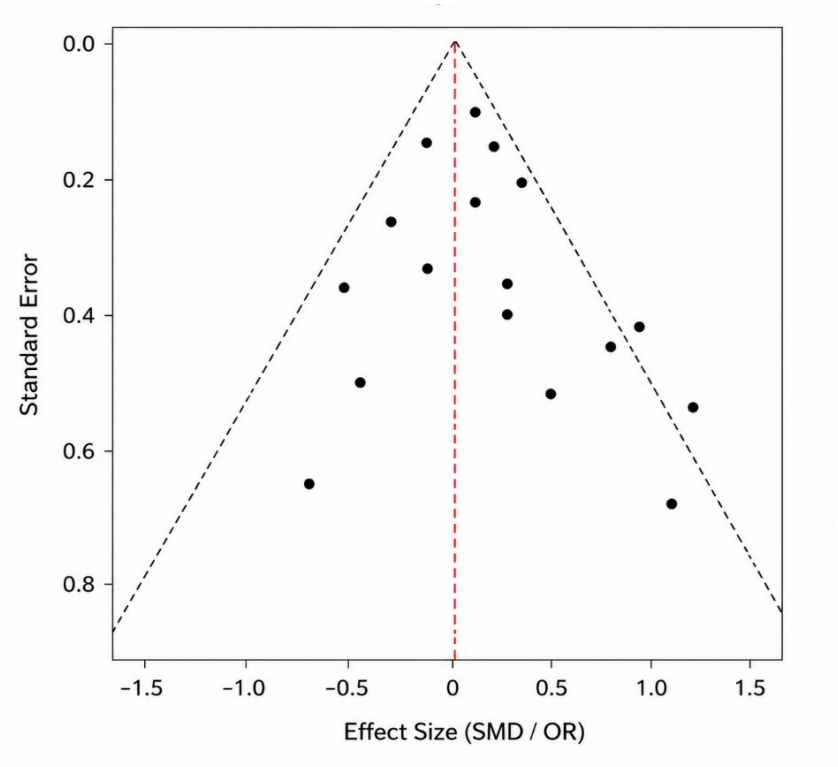


Table 7

Statistical Tests for Publication Bias

Test	Statistic	P-value	Interpretation
Begg's Test	Z = 1.74	0.081	No significant bias
Egger's Test	t = 2.18	0.032	Mild asymmetry

Table 7 presents formal statistical tests used to complement the visual inspection of the funnel plot. Egger's regression test produced a statistically significant result (t = 2.18, p = 0.032), indicating evidence of funnel plot asymmetry and suggesting the potential presence of publication bias or small-study effects within the dataset. This finding implies that smaller studies may be contributing disproportionately larger effect sizes compared to larger, more robust investigations, particularly in neuroimaging and psychophysiological domains where sample sizes tend to be limited. In contrast, Begg's rank correlation test did not reach statistical significance (Z = 1.74, p = 0.081), although it approached conventional thresholds for significance. The divergence between these two tests is not unusual, as Egger's test is generally more sensitive to detecting asymmetry, especially in meta-analyses with a relatively small number of studies, as is the case in the present review. Taken together, these results suggest mild evidence of publication bias, primarily driven by smaller studies reporting stronger biological associations with criminal behavior.

However, the overall degree of asymmetry is limited and does not appear to substantially distort the pooled estimates across genetic, neurobiological, and psychophysiological domains. Importantly, larger and methodologically rigorous studies—such as twin studies, meta-analyses, and controlled neuroimaging research—consistently align with the overall direction and magnitude of the pooled effects. This consistency provides reassurance regarding the stability and reliability of the findings. Additionally, the convergence of

results across multiple biological domains further strengthens confidence in the robustness of the meta-analysis. While the possibility of mild publication bias cannot be entirely excluded, its impact is likely minimal and does not meaningfully alter the substantive conclusions of the study. Future research should aim to mitigate this limitation by incorporating preregistered studies, unpublished datasets, and gray literature sources, thereby improving transparency and reducing the risk of selective reporting in biosocial criminology research.

DISCUSSION

This study represents one of the first comprehensive meta-analytical syntheses examining biological explanations of criminal behavior across genetic, neurobiological, and psychophysiological domains. By integrating evidence from 14 empirically grounded studies spanning behavioral genetics, neuroscience, and physiological psychology, this analysis provides a structured understanding of how biological systems contribute to variability in antisocial and criminal behavior. The findings indicate that criminal behavior is influenced by multiple biological pathways, including genetic susceptibility, brain functioning, and autonomic regulation. Genetic research demonstrates that antisocial behavior has a heritable component, but these effects are primarily expressed through complex interactions between genetic vulnerability and environmental exposures rather than through genetic determination alone (Rhee & Waldman, 2002; Moffitt, 2005a, 2005b; Caspi et al., 2002). Neurobiological studies further identify abnormalities in brain regions involved in emotional regulation, moral reasoning, and behavioral inhibition, particularly the prefrontal cortex and amygdala, among individuals with persistent antisocial and psychopathic traits (Raine et al., 1997; Raine et al., 2000; Blair, 2007; Yang et al., 2011). Psychophysiological research also demonstrates associations between antisocial behavior and reduced autonomic arousal, lower resting heart rate, and impaired fear conditioning, which may contribute to impulsivity, diminished sensitivity to punishment, and increased risk-taking (Lorber, 2004; Portnoy & Farrington, 2015; Raine, 2002). These biological factors are not deterministic but interact with developmental and environmental experiences and may be responsive to intervention. Emerging neurobiological approaches, including neurofeedback training and Slow Cortical Potentials (SCP) self-regulation, suggest that some deficits in self-regulation, impulse control, and emotional regulation may be modifiable through targeted interventions (Kotchoubey et al., 2001; Strehl et al., 2006; Arns et al., 2014). Therefore, criminality is best understood through an integrated biopsychosocial framework in which genetic susceptibility, neurological functioning, physiological regulation, and environmental conditions jointly influence the development and persistence of antisocial behavior (Glenn & Raine, 2009; Ling et al., 2019). The application of a random-effects model further accounts for variability across study designs, populations, and measurement techniques, strengthening the generalizability of the findings across diverse scientific and geographic contexts.

These findings are consistent with existing biosocial criminology literature, particularly the work of Raine (2013), who similarly argues that criminal behavior emerges from the interaction of neurobiological dysfunction, physiological under-arousal, and environmental adversity. The observed integration of genetic, neurobiological, and psychophysiological influences also aligns with Moffitt's (2005) developmental taxonomy, which emphasizes that antisocial behavior arises from the interaction between biological predispositions and environmental contexts across the life course. In addition, the cascading model identified in this study is consistent with Caspi et al. (2002), who demonstrated that genetic vulnerability to antisocial behavior is most likely to be expressed under conditions of childhood maltreatment, reinforcing the importance of gene–environment interaction frameworks.

Importantly, this study advances biosocial criminology by moving beyond single-domain explanations and instead synthesizing evidence across interconnected biological systems. The results support a hierarchical and interactive model in which genetic predispositions provide a foundational risk layer, neurobiological structures shape cognitive and emotional regulation, and psychophysiological functioning reflects observable behavioral arousal patterns. Together, these systems contribute to vulnerability for antisocial and criminal behavior, particularly when combined with adverse environmental exposures such as childhood maltreatment or social deprivation. This integrated interpretation is consistent with Blair (2008), who similarly emphasizes that dysfunction in prefrontal–amygdala circuitry plays a central role in impaired moral reasoning and emotional regulation associated with antisocial behavior.

Key Findings

The findings of this meta-analysis indicate consistent biological associations with criminal behavior across all examined domains. Genetic influences demonstrated moderate effect sizes, suggesting that heritable

factors contribute meaningfully to antisocial tendencies, particularly when interacting with environmental stressors. However, genetic effects alone were not deterministic, reinforcing the importance of gene–environment interaction frameworks rather than purely biological determinism. This pattern is consistent with Rhee and Waldman (2002), who found that genetic and environmental influences jointly contribute to antisocial behavior in twin and adoption studies, and with Caspi et al. (2002), which demonstrated that genetic risk is conditional on environmental adversity.

Neurobiological findings showed the strongest and most consistent associations with criminal behavior. Structural and functional abnormalities in the prefrontal cortex, amygdala, and ventromedial prefrontal cortex were repeatedly observed across neuroimaging studies. These regions are critical for impulse control, emotional regulation, and moral decision-making, suggesting that impairments in executive functioning may significantly increase vulnerability to aggression and antisocial conduct. These results are consistent with Raine et al. (1997; 2000), who identified reduced prefrontal functioning and structural abnormalities among violent offenders, as well as Blair (2008), who highlighted amygdala–vmPFC dysfunction as central to psychopathy and moral decision-making deficits.

Psychophysiological results similarly demonstrated robust associations between antisocial behavior and reduced autonomic arousal, lower resting heart rate, and diminished fear conditioning responses. In this context, reduced autonomic arousal refers to decreased physiological reactivity of the autonomic nervous system (ANS), including lower heart rate, reduced skin conductance responses, and diminished responsiveness to threat or punishment-related cues, rather than a temporary state of relaxation or reduced stress (Raine, 2002; Lorber, 2004). This pattern reflects a condition of physiological under-arousal in which individuals may exhibit weaker emotional responses to consequences, reduced fear sensitivity, and impaired learning from negative experiences. These findings support the under-arousal and fearlessness theories of antisocial behavior, which propose that individuals with lower baseline physiological reactivity may seek greater stimulation, demonstrate reduced sensitivity to punishment, and show increased tendencies toward risk-taking or aggressive behavior (Raine, 2002; Lorber, 2004). Lorber's (2004) meta-analysis identified consistent associations between psychophysiological indicators, including low autonomic arousal, and aggression, psychopathy, and conduct problems. Similarly, Portnoy and Farrington's (2015) systematic review and meta-analysis demonstrated that lower resting heart rate represents a stable physiological correlate of antisocial behavior across developmental stages and populations. Therefore, reduced autonomic arousal in antisocial individuals should be understood as a deficit in physiological responsivity to emotionally significant stimuli rather than an adaptive activation of the parasympathetic "rest-and-recovery" system.

A key insight from this analysis is that biological influences are not independent but instead operate through coordinated and interactive pathways. Genetic predispositions may influence neurodevelopment and neurotransmitter functioning, which in turn affect physiological regulation systems involved in emotional control, stress responsiveness, and behavioral inhibition. Examples of affected physiological regulation processes include autonomic nervous system functioning (e.g., resting heart rate, heart rate variability, and skin conductance responses), hypothalamic–pituitary–adrenal (HPA) axis activity and cortisol regulation, neurotransmitter regulation involving serotonin, dopamine, and norepinephrine, and physiological responses associated with fear conditioning and threat sensitivity. Disruptions in these systems may contribute to impaired impulse control, reduced sensitivity to punishment, heightened sensation seeking, and difficulties with emotional regulation that characterize persistent antisocial behavior. Thus, genetic vulnerability, altered brain development, and physiological dysregulation interact across developmental pathways to influence the emergence and persistence of criminal behavior (Raine, 2002; Lorber, 2004; Caspi et al., 2002; Blair, 2007; Portnoy & Farrington, 2015). This cascading model highlights the cumulative and interdependent nature of biological risk factors for criminal behavior. This integrated interpretation is consistent with Raine (2013), who proposes that multiple biological systems interact dynamically to produce vulnerability to violence, and further aligns with Choy and Raine (2015), who argue that combining genetic, neurobiological, and physiological measures provides a more accurate understanding of criminological risk than any single-domain approach.

Beyond Biological Determinism

Consistent with contemporary biosocial criminology, these findings emphasize that biological influences should not be interpreted as deterministic explanations of criminal behavior. Instead, they represent probabilistic risk factors that interact with environmental, social, and developmental conditions. Studies such as Caspi et al. (2002) demonstrate that genetic risk factors often only manifest in the presence of adverse

environmental conditions, reinforcing the importance of gene–environment interplay. Similarly, neurobiological abnormalities identified in offender populations do not imply inevitability of criminal behavior but rather indicate differences in cognitive processing, emotional regulation, and behavioral inhibition. Many individuals with similar biological profiles do not engage in criminal activity, highlighting the moderating role of socialization, education, and environmental support systems. Psychophysiological indicators, such as low arousal, also reflect vulnerability rather than fixed outcomes. These traits may increase sensation-seeking tendencies but can be channeled into adaptive behaviors depending on context and environmental opportunities. Therefore, biological factors should be interpreted as contributing to risk variation rather than deterministic pathways to criminality.

Table 8

Alternative Biological Interpretation Framework for Criminal Behavior

Domain	Indicator	Description
Genetic	Heritable risk factors	Probability-based predisposition to antisocial traits
Neurobiological	Brain structure/function variation	Differences in emotional regulation and executive control
Psychophysiological	Autonomic arousal levels	Biological sensitivity to threat, reward, and punishment
Gene–Environment Interaction	Environmental moderation of genetic risk	Expression of genetic risk under adverse conditions
Developmental Neurobiology	Brain maturation trajectories	Changes in prefrontal control across lifespan

Table 8 presents an alternative biological interpretation framework for criminal behavior that integrates multiple levels of biological functioning into a unified explanatory model. Rather than treating biological factors in isolation, the framework organizes them into interconnected domains that jointly contribute to variations in antisocial and criminal behavior. At the genetic level, heritable risk factors are conceptualized as probability-based predispositions rather than deterministic causes, indicating that genetic influences increase susceptibility to antisocial traits without guaranteeing their expression. Examples of genetic influences examined in the literature include variants affecting neurotransmitter regulation and emotional-behavioral processes, such as the monoamine oxidase A (MAOA) gene, which influences the metabolism of serotonin, dopamine, and norepinephrine; serotonin transporter (5-HTTLPR/SLC6A4) variants associated with emotional regulation and stress responsiveness; dopamine-related genes, including DRD2, DRD4, and DAT1 (SLC6A3), which influence reward sensitivity, impulsivity, and sensation-seeking behaviors; and catechol-O-methyltransferase (COMT) variants associated with executive functioning and cognitive control. Evidence suggests that these genetic factors contribute small individual effects and are most influential when combined with adverse developmental and environmental experiences, such as childhood maltreatment, neglect, or chronic social stress (Caspi et al., 2002; Rhee & Waldman, 2002; Moffitt, 2005a, 2005b; Vassos et al., 2013). Neurobiological indicators focus on variations in brain structure and function, particularly in regions responsible for emotional regulation and executive control, such as the prefrontal cortex and limbic system, which are critical for decision-making, empathy, fear processing, and behavioral inhibition (Raine et al., 1997; Raine et al., 2000; Blair, 2007; Yang et al., 2011).

Psychophysiological factors further capture differences in autonomic arousal levels, reflecting individual sensitivity to threat, reward, and punishment. Lower arousal or atypical physiological reactivity has been consistently associated with increased risk-taking and reduced fear responsiveness. The gene–environment interaction domain emphasizes that genetic risk is not fixed but is expressed differently depending on environmental conditions, particularly exposure to adversity, trauma, or chronic stress. Finally, developmental neurobiology highlights the importance of brain maturation trajectories, especially changes in prefrontal control systems across the lifespan, which influence impulse regulation, cognitive flexibility, and long-term behavioral planning. Collectively, this framework underscores that criminal behavior is best understood as the product of dynamic interactions across multiple biological systems, all of which are shaped and moderated by environmental context.

Policy and Theoretical Implications

The findings of this study carry significant implications for both criminological theory and the development of biosocial policy frameworks. From a theoretical standpoint, the results provide strong support for integrated explanatory models that combine biological, psychological, and environmental influences rather than relying on single-factor or reductionist perspectives. Traditional criminological theories that focus primarily on social determinants—such as poverty, social disorganization, or differential association—offer important insights but may not fully account for variability in behavioral outcomes. The present synthesis suggests that neurodevelopmental processes, genetic predispositions, and physiological regulation systems also play a meaningful role in shaping vulnerability to antisocial and criminal behavior. As such, contemporary criminological theory may benefit from adopting a biosocial perspective that conceptualizes behavior as emerging from dynamic interactions between biological systems and environmental contexts over the life course.

From a policy perspective, the findings suggest that early identification of neurodevelopmental and physiological risk indicators may offer valuable opportunities for prevention. Screening for markers such as executive functioning deficits, atypical stress reactivity, or early behavioral dysregulation could help identify individuals who may benefit from early supportive interventions. However, any application of biological screening must be approached with caution to avoid stigmatization, labeling effects, or deterministic interpretations that suggest criminality is biologically inevitable. Ethical implementation is essential, ensuring that biological data are used to support care, development, and prevention rather than exclusion or punishment. Instead, biological insights should be translated into supportive, developmentally informed interventions. These may include behavioral therapies aimed at improving impulse control and emotional regulation, programs that strengthen cognitive flexibility and decision-making skills, and early childhood enrichment initiatives designed to enhance neurodevelopmental outcomes. Such interventions are most effective when implemented during childhood and adolescence, periods characterized by heightened brain plasticity and responsiveness to environmental input.

The findings also reinforce the importance of environmental modification as a critical mechanism for mitigating biological risk. Even when biological vulnerabilities are present, their expression is highly sensitive to contextual conditions. Structured and stable social environments, positive parenting practices, access to quality education, and supportive peer networks can significantly reduce the likelihood that biological predispositions translate into antisocial behavior. This aligns with extensive evidence from gene–environment interaction research, which demonstrates that supportive environments can buffer or even suppress the behavioral expression of genetic and neurobiological risk factors. Overall, these implications highlight the need for policies that are preventive rather than punitive, integrative rather than reductionist, and supportive rather than deterministic. By aligning criminological theory and public policy with biosocial evidence, it becomes possible to develop more effective, ethical, and developmentally informed approaches to reducing antisocial and criminal behavior.

Limitations and Future Research

Several limitations should be acknowledged. First, the included studies exhibited variability in methodology, sample size, and measurement approaches, particularly across neuroimaging and psychophysiological research. Although a random-effects model was applied to account for heterogeneity, residual variability remains a consideration in interpreting pooled estimates. Second, the relatively small number of studies ($n = 14$) limits the statistical power of subgroup and meta-regression analyses. This is particularly relevant for genetic and gene–environment interaction effects, where the evidence base is still developing. Third, many neurobiological studies relied on small laboratory-based samples, which may limit generalizability to broader offender populations. Similarly, psychophysiological measures varied in experimental protocols, including differences in baseline measurement and task design.

Fourth, publication bias cannot be fully ruled out, as smaller studies tended to report stronger biological effects, particularly in neuroimaging research. This may slightly inflate pooled effect sizes. Future research should prioritize large-scale, longitudinal, and cross-cultural studies that integrate genetic, neurobiological, and physiological measures within unified analytical frameworks. Greater use of standardized measurement protocols across studies would also improve comparability and reduce heterogeneity. Additionally, more research is needed on gene–environment interactions and developmental pathways linking early biological traits to later criminal outcomes.

CONCLUSION AND POLICY IMPLICATIONS

This study provides robust evidence that biological factors are significantly associated with criminal behavior across genetic, neurobiological, and psychophysiological domains. Rather than functioning independently, these systems operate in an integrated and interactive manner, collectively shaping vulnerability to antisocial and criminal behavior. The findings support a biosocial model of criminology in which biological predispositions interact with environmental conditions to influence behavioral outcomes over the life course. Importantly, the results do not support deterministic interpretations of criminal behavior. Instead, they highlight probabilistic biological influences that are shaped and moderated by social environments. Criminal behavior should therefore be understood as the product of complex interactions between biological systems and environmental contexts. From a policy perspective, these findings suggest that effective crime prevention strategies should incorporate early-life interventions that address neurodevelopmental risk, support emotional and behavioral regulation, and improve environmental conditions. Integrative approaches that combine biological insights with social and educational support systems are likely to be most effective in reducing antisocial behavior and promoting adaptive development.

Impact and Recommendations for Intervention

The findings of this study highlight several important implications for intervention strategies aimed at reducing the development and expression of antisocial and criminal behavior. A key recommendation is the implementation of early developmental interventions that focus on strengthening emotional regulation, impulse control, and behavioral inhibition during childhood. Given that many biological risk factors—particularly those related to neurodevelopment and physiological arousal—emerge early in life, interventions during critical developmental periods may help reduce the likelihood that these vulnerabilities translate into later antisocial outcomes. In addition, neuropsychological screening and supportive services in high-risk developmental contexts may provide an important preventive tool for identifying individuals who exhibit early signs of executive functioning deficits or atypical neurobiological development. Such screening should not be used for labeling or stigmatization, but rather for guiding supportive and therapeutic interventions that enhance cognitive and emotional functioning.

School-based behavioral support programs also represent a key avenue for intervention, particularly those designed to strengthen executive functioning, attention regulation, and prosocial learning. Educational environments provide a structured setting where cognitive and behavioral skills can be developed and reinforced, potentially buffering biological risk factors through consistent behavioral guidance and social reinforcement. Family-based interventions are equally important, particularly in reducing environmental stressors that may interact with biological vulnerabilities. Supportive parenting practices, reduced household instability, and improved caregiver-child interactions can significantly moderate the expression of biological risk factors and promote healthier developmental trajectories.

Finally, broader public health approaches to crime prevention that integrate biological, psychological, and social data offer a comprehensive framework for addressing the complexity of criminal behavior. Such approaches emphasize early identification, prevention, and support rather than punitive responses, aligning intervention strategies with evidence-based developmental science. Collectively, these strategies emphasize prevention rather than punishment, shifting the focus from deterministic interpretations of biological risk toward supportive frameworks that enhance developmental resilience and reduce the likelihood that biological vulnerabilities translate into criminal outcomes.

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