PSYCHOLOGY OF CRIMINAL BEHAVIOR

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ABSTRACT

There is no single psychological cause of criminal behavior.[1] This paper reviews five bodies of research that together explain why some people offend and others do not: biological and neuropsychological factors, personality and psychopathy, learning and social cognition, developmental and environmental adversity, and mental illness and substance use. The evidence shows that genetic risk, prefrontal and autonomic differences, callous-unemotional traits, hostile thinking patterns and early maltreatment all increase the risk of offending, but none is deterministic, and most work through interaction rather than alone. Severe mental illness, often assumed to be a major driver of crime, accounts for only a small share of violence after adjusting for substance misuse and social disadvantage. The paper concludes that integrated, risk-based models, above all the risk need responsivity framework, provide the most defensible basis for assessment, treatment and prevention, if findings are used with caution about stigma, determinism and the limits of prediction.

[1] D.A. Andrews & James Bonta, The Psychology of Criminal Conduct (6th ed. 2016)

INTRODUCTION

Why people commit crime is one of the oldest questions in the behavioral sciences, and no single answer has stood in the test of time. Early theories searched for a criminal type, whether in the body, the mind or the moral character. Modern psychology instead conceptualizes crime as the result of multiple interacting influences and seeks to understand which combinations of traits, experiences and situations increase the likelihood of offending. Crucially, "criminal behavior" is a legal category, not a psychological one: it includes a shoplifting teenager, a fraudster, a domestic abuser and a serial killer, who have little in common psychologically, and so any serious account must be specific about the behavior it is addressing.

This paper argues that criminal behavior should be understood through a biopsychosocial lens. Biological vulnerabilities influence how a person responds to experience, personality and cognition determine how that experience is interpreted, and the social environment provides the opportunities, models and pressures that transform risk into action. Five areas of research are reviewed in turn:[2] biological and neuropsychological factors; personality and psychopathy; learning and social cognition; developmental and environmental adversity; and mental illness and substance use. The paper then considers how this research informs assessment, treatment and prevention, and what restrictions ought to be placed on its application.

BIOLOGICAL AND NEUROPSYCHOLOGICAL PERSPECTIVES

Twin and adoption studies show that antisocial behavior is moderately heritable: in a meta-analysis of 51 studies, Rhee and Waldman (2002) estimated that genetic factors explained roughly 40% of the variance in antisocial behavior, with non-shared environmental influences accounting for most of the remainder.[3] Heritability is not destiny, however: it refers to the variation within a population under certain conditions and says nothing about an individual's future.

The most compelling example of gene environment interplay concerns the MAOA gene, involved in regulating neurotransmitters such as serotonin and dopamine. A New Zealand birth cohort and found that boys carrying the low-activity variant were considerably more likely to

[2] Adrian Raine, The Anatomy of Violence: The Biological Roots of Crime (2013).

[3] Hyun Rhee & Irwin D. Waldman, Genetic and Environmental Influences on Antisocial Behavior: A Meta-Analysis of Twin and Adoption Studies, 128 Psychol. Bull. 490 (2002).

develop antisocial behavior, but only if they had been maltreated in childhood. A follow-up meta-analysis confirmed the interaction in males, although the effect is modest and remains controversial. Genes seem to moderate sensitivity to adversity rather than directly cause crime.

Neuroimaging studies point to the prefrontal cortex, which governs planning, impulse control, and moral judgment reported about 11% less prefrontal gray matter in men with antisocial personality disorder than in controls, together with reduced autonomic arousal. Low resting heart rate, a sign of under-arousal, is one of the most replicated biological correlates of antisocial behavior. One interpretation is that under-aroused individuals crave stimulation and fear punishment less; another that weak amygdala responsiveness reduces the emotional learning that usually restrains harmful acts.

[4]Brain development also helps explain the age of crime curve, in which offending peaks in midto-late adolescence and then declines. Steinberg argues that reward-seeking systems mature earlier than the cognitive-control systems that regulate them, creating a window of heightened risk-taking, particularly in the company of peers. These findings call for caution: brain differences are group averages, can reflect stress, injury or substance use as much as cause offending, and cannot establish guilt or intent in an individual case.

PERSONALITY, PSYCHOPATHY AND ANTISOCIAL PERSONALITY DISORDER

[5]Personality research asks whether stable traits distinguish offenders from non-offenders. In 1977, the proposed that high extraversion, neuroticism and psychoticism, combined with a slowly conditioning nervous system, impedes the acquisition of conscience. Contemporary work uses the five-factor model: a meta-analysis by Miller and Lynam 2001 found that low agreeableness and low conscientiousness were the traits most consistently linked to antisocial behavior: *offenders tend to be hostile, manipulative, impulsive and careless about consequences.*

[6]Psychopathy is the most intensively studied personality pattern in criminology. Assessed using Hare's Psychopathy Checklist–Revised (PCL-R), it combines an interpersonal and affective

[4] Avshalom Caspi et al., Role of Genotype in the Cycle of Violence in Maltreated Children, 297 Science 851 (2002).

[5] Hans J. Eysenck, Crime and Personality (3d ed. 1977).

[6] Joshua D. Miller & Donald Lynam, Structural Models of Personality and Their Relation to Antisocial Behavior: A Meta-Analytic Review, 39 Criminology 765 (2001).

dimension, marked by superficial charm, grandiosity, shallow emotion and lack of remorse, and a lifestyle and antisocial dimension, marked by impulsivity, irresponsibility and early behavioral problems. Psychopathic traits are found in roughly 1% of the general population, but a much larger proportion of prisoners, and psychopathic offenders tend to offend earlier, more violently and with a higher risk of reoffending.

Researchers continue to debate its origins, linking psychopathy to dysfunction in the ventromedial prefrontal cortex, which impairs recognition of others' distress and the formation of moral emotions. Developmental work has identified callous-unemotional traits in children, characterized by lack of guilt, limited empathy and shallow affect, as an early marker of a more severe and persistent pattern of conduct problems. Antisocial personality disorder in the DSM (Diagnostic and Statistical Manual of Mental Disorders) is a broader and more behavioral diagnosis than psychopathy, and many people who meet its criteria fail to display emotional deficits at the psychopathic core.

Two points temper the popular image of the psychopath as an inevitable criminal: many people with elevated psychopathic traits are never convicted, and the construct is dimensional rather than categorical; the label carries heavy legal consequences, so its assessment requires trained clinicians and standardized procedures.

LEARNING, SOCIAL AND COGNITIVE PROCESSES

Learning [7]theories explain crime as behavior acquired and maintained through experience. Sutherland's, in 1947 differential association theory holds that people learn criminal attitudes, motives and techniques through close association with others, and offending results when definitions favorable to law violation outweigh unfavorable ones. Bandura's social learning theory added observation as a mechanism: in the classic Bobo doll experiments, children who watched an adult behave aggressively reproduced the same acts, including novel ones, when given the opportunity. Reinforcement then determines whether the behavior is repeated, which is why aggression that "works" tends to continue.

[8]Cognitive research focuses on how offenders interpret the social world: aggressive children

[7] Edwin H. Sutherland, Principles of Criminology (4th ed. 1947).

[8] Albert Bandura et al., Transmission of Aggression Through Imitation of Aggressive Models, 63 J. Abnormal & Soc. Psychol. 575 (1961).

and adults tend to attribute hostile intent to ambiguous actions (such as being bumped in a corridor) and retaliate. A meta-analysis confirmed a reliable association between hostile attribution bias and aggressive behavior showed that physically abused children later displayed this style of processing, which partly accounted for their higher aggression, suggesting a cognitive route from abuse to violence.

Moral disengagement provides a mechanism for how people switch off self-sanctions: Bandura in 1999 described processes such as euphemistic labeling, displacing responsibility, dehumanizing victims, and blaming them for what happened. These processes explain how otherwise ordinary individuals commit harm without the guilt that would normally restrain them, and they appear in accounts of white-collar crime, gang violence and political violence alike.

In 1993 integrated these ideas in a developmental taxonomy: she distinguished a small group of life-course-persistent offenders whose antisocial behavior began in childhood and stemmed from neuropsychological vulnerability and adverse environments, from a larger group of adolescence limited offenders whose offending reflected peer mimicry during the gap between biological maturity and adult status and usually stopped in early adulthood. The taxonomy explains why most offenders desist while a minority persist for decades.

DEVELOPMENTAL AND ENVIRONMENTAL INFLUENCES

[9]Few findings are as consistent as the link between childhood adversity and later offending. The Adverse Childhood Experiences study demonstrated that abuse, neglect and household dysfunction are common and have cumulative effects on adult health and behavior in 1998 among juvenile offenders, 2015 found that the number of adverse experiences predicted serious, violent and chronic offending. Maltreatment may operate via the hostile attributional and neurobiological pathways described above, and via learned coping responses such as aggression and distrust.

Family process matters too: described a coercive cycle in which a child's aversive behavior is inadvertently rewarded when exhausted parents give in, thus teaching the child that defiance

[9] Vincent J. Felitti et al., Relationship of Childhood Abuse and Household Dysfunction to Many of the Leading Causes of Death in Adults: The Adverse Childhood Experiences (ACE) Study, 14 Am. J. Preventive Med. 245 (1998).

and aggression control others. Harsh or inconsistent discipline, poor supervision and low warmth predict later delinquency, and the quality of attachment influences the development of empathy and emotional regulation. In adolescence, the influence shifts to peers, and association with delinquent friends is among the strongest predictors of offending.

Social context shapes these processes: found that violence in Chicago neighborhoods was lower where residents shared trust and were willing to intervene for the common good, even after adjusting for neighborhood disadvantage. Poverty does not cause crime in any simple way, since most poor people never offend, but it concentrates on risk factors, reduces supervision and limits legitimate opportunities, increasing exposure to the stressors and models that make offending more likely.

Protective factors deserve equal attention: stable relationships, school engagement, employment and a warm bond with at least one caregiver all reduce the likelihood of offending, even among high-risk children.

MENTAL ILLNESS, SUBSTANCE USE AND CRIME

Popular culture often depicts offenders as mentally ill, but the evidence supports a more nuanced position: [10]Analysis found that people with schizophrenia and other psychoses were at elevated risk of violence relative to the general population, but the risk was considerably larger among those who also misused substances and was similar to that of substance misuse without psychosis. At the population level, estimated that severe mental illness accounted for only about5% of violent crime in Sweden. Most people with mental illness are never violent and are more likely to be victims than perpetrators.

Where risk does exist, it is tied mainly to active symptoms and to co-occurring factors such as substance misuse, a history of victimization, unemployment and exposure to violence, rather than to diagnosis alone. Substance use is a stronger correlate of offending found in a meta-analysis that drug users were roughly three to four times as likely to offend as non-users, with the association strongest for heroin and crack cocaine. Intoxication impairs judgment and lowers inhibition, and dependence creates economic pressure to offend.

[10] Seena Fazel & Martin Grann, The Population Impact of Severe Mental Illness on Violent Crime, 163 Am. J. Psychiatry 1397 (2006).

The practical implication is that treating mental illness and addiction is worthwhile for health reasons and reduces risk, but psychiatric diagnosis is a poor predictor of crime and equating the two fuels stigma that discourages people from seeking treatment.

IMPLICATIONS FOR ASSESSMENT, TREATMENT AND PREVENTION

The most influential applied framework is the risk need responsivity (RNR) model the risk principle holds that the intensity of intervention should match an offender's likelihood of reoffending; [11]the need principle holds that treatment should target dynamic, changeable risk factors such as antisocial attitudes, substance misuse, delinquent associates and poor self-control; the responsivity principle holds that programs should be delivered in a style that suits the person's learning style, motivation and abilities. The model draws directly on the learning and cognitive research reviewed above.

Cognitive-behavioral programs are the best-supported treatment: a meta-analysis of 58 studies found that they reduced recidivism on average, with the largest effects for higher-risk offenders and well-implemented programs. Prevention targets earlier points in the causal chain: programs that support parenting and early education address the family and developmental risks described above, and the High Scope Perry Preschool study found that participants had significantly fewer lifetime arrests by age 40 than controls.

Risk assessment tools combine static factors such as criminal history with dynamic factors, and structured methods outperform unaided clinical judgment, although prediction for individuals remains imperfect. Ethical constraints are equally important: psychological findings are probabilistic (a risk score describes a group of people with similar characteristics, not a certain future), biological explanations can be misread as deterministic or as excusing responsibility, psychological ones can blame individuals for the conditions of their upbringing, and tools based on historical data may perpetuate existing biases in policing and sentencing. Applications should be transparent, regularly validated, and used to guide support rather than justify harsher punishment.

[11] Trevor Bennett, Katy Holloway & David P. Farrington, The Statistical Association Between Drug Misuse and Crime: A Meta-Analysis, 13 Aggression & Violent Behav. 107 (2008).

CONCLUSION

Criminal behavior emerges from the interaction of vulnerabilities and circumstances, not from a single flaw in the individual. Genetic and neurological differences influence sensitivity to stress and capacity for self-control, personality traits such as low agreeableness and callous-unemotional traits shape empathy and responsiveness to punishment, learned beliefs and thinking patterns convert provocation into aggression and justify harm, and early maltreatment, family dysfunction, peers and neighborhood conditions determine whether risk is amplified or buffered. Severe mental illness plays a smaller role than commonly assumed, and substance use a larger one.

[12]The research also gives grounds for optimism: because many of the strongest risk factors are dynamic, they can be changed, and interventions that match intensity to risk and target criminogenic needs reduce reoffending. Future work should integrate biological and social data more closely, test findings beyond Western male samples, and develop assessment methods that are accurate, fair and humane. Understanding why people offend is not the same as excusing offending; it is the basis for preventing it.

[12] William M. Grove et al., Clinical Versus Mechanical Prediction: A Meta-Analysis, 12 Psychol. Assessment 19 (2000).

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