NEUROSCIENCE AND CRIMINAL JUSTICE: TIME FOR A "COPERNICAN REVOLUTION?"

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ABSTRACT

The main purpose of this Article is to argue for a fundamental change in the conceptual orientation of criminal justice: from one based on concepts such as free will, desert, and moral responsibility, to one based on empirical science. The Article describes research in behavioral genetics, acquired brain injuries, and psychological traumatization in relation to criminality. This research has reached a level of development at which the traditional approach to criminality is no longer tenable and should be discarded. I argue that mental health legislation provides a model that could be adapted and applied to offenders.

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INTRODUCTION

> If there are beings in the world whose acts shock all accepted prejudices, we must not preach at them or punish them ... because their bizarre tastes no more depend upon themselves than it depends on you whether you are witty or stupid, well made or hump-backed.... What would become of your laws, your morality, your religion, your gallows, your paradise, your gods, your hell, if it were shown that such and such fluids, such fibers, or a certain acridity in the blood, or in the animal spirts, alone suffice to make a man the object of your punishments or your rewards?[1]

> After all, I would probably never have been able to do anything with my magnanimity—neither to forgive, for my assailant may have slapped me because of the laws of nature, and one cannot forgive the laws of nature; nor to forget, for even if it were the laws of nature, it is insulting all the same.[2]

De Sade and Dostoevsky were both writing before the advent of neuroscience.[3] These quotations illustrate that the application of neuroscience research to criminal behavior is casting a fresh light on, and throwing into relief, much older, philosophical questions about the nature of human actions. Are instances of criminal behavior to be understood in the way that we understand other events in the world, such as changes in the weather, or do they have a special quality that sets them apart? This special quality is usually understood in terms of concepts such as free will and responsibility.[4] The idea that we possess a capacity for free will and

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1. HAVELOCK ELLIS, 3 STUDIES IN THE PSYCHOLOGY OF SEX 107-08 (2d ed. 1920) (quoting the Marquis de Sade).

2. Fyodor Dostoevsky, *Notes from Underground*, *in* SIXTEEN SHORT NOVELS: AN ANTHOLOGY 415, 421 (Wilfrid Sheed ed., 1985).

3. De Sade and Dostoevsky's literary works were written in the 1700s and 1800s. John Attarian, *Dostoevsky vs. The Marquis de Sade*, INTERCOLLEGIATE STUD. INST. (Oct. 8, 2014), https://isi.org/modern-age/dostoevsky-vs-the-marquis-de-sade/ [https://perma.cc/F7Z2-4T2V]. Neuroscience emerged as a distinct scientific discipline in the late twentieth century, although other scientific disciplines began to study the nervous system before then. GORDON M. SHEPHERD, CREATING MODERN NEUROSCIENCE: THE REVOLUTIONARY 1950S 3-4 (2010).

4. *See Free Will*, ENCYCLOPEDIA BRITANNICA (Nov. 23, 2017), https://www.britannica.com/topic/free-will [https://perma.cc/5LST-N4GT].

responsibility is an important element of our self-concept that imbues our lives with meaning.[5] The proposal that we are not creatures of God, with a God-given capacity for free will, but instead just puppets dancing on the strings of causal necessity, is a threat to this sense of exceptionalism.[6]

This threat extends beyond individual identity. There are major institutions in our societies that rest, at least implicitly, on foundations that are being challenged by neuroscience research.[7] These include most of the major religions and our systems of justice.[8]

There are also our intuitive, emotional responses to wrongdoing, especially when we, or those who are close to us, are the victim.[9] Nietzsche pointed out that "punishment developed as a *retaliation* absolutely independently of any preliminary hypothesis of the freedom or determination of the will."[10] In most societies, the state has taken over the role of punishment from individual victims and their families or clans.[11] In some cases, punishment of perpetrators may be an important element in helping victims recover from the traumatization of crime.[12] As illustrated by the quote from Dostoevsky, a scientific approach to crime may be seen as invalidating current rationales for blame and punishment.[13] The result is that all of the emotions generated by criminal acts could be denied any form of practical expression.

All of these questions lurk behind neuroscience approaches to criminality and create disquiet about where all of this is leading

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5. *See* Stephen Cave, *There's No Such Thing as Free Will*, THE ATLANTIC, June 2016, at 68, 70.

6. *See id.* at 70, 72-73.

7. *See id.* at 70.

8. *See id.*

9. *See Violent Crime Victims*, U.S. DEP'T OF JUST. (July 6, 2021), https://www.justice.gov/usao-wdwa/victim-witness/violent-crime-victims [https://perma.cc/GZ2E-N3GJ]; *Victim of Crime—Family Member or Friend*, GOV'T OF B.C., https://www2.gov.bc.ca/gov/content/justice/criminal-justice/bcs-criminal-justice-system/family-members-or-friends/victim-of-crime-family-member-or-friend [https://perma.cc/FW7H-QEJ9].

10. FRIEDRICH NIETZSCHE, THE GENEALOGY OF MORALS: A POLEMIC 69 (Horace B. Samuel trans., 1913).

11. U.S. BUREAU OF PRISONS, THE JAIL: ITS OPERATION AND MANAGEMENT 1-2 (Nick Pappas ed., 1970).

12. John S. Callender, *Justice, Reciprocity and the Internalisation of Punishment in Victims of Crime*, 13 NEUROETHICS 43, 44 (2020).

13. *See* Dostoevsky, *supra* note 2, at 421.

us.[14] Norms, practices, and institutions in criminal justice have evolved over centuries, and there are understandable anxieties about the potential for unintended consequences if these are undermined.[15]

The growth of neuroscience research in criminality and moral decision-making has been exponential in the past few decades.[16] It would take several large volumes to summarize this growth. In this Article, I will focus on three areas that seem to be of importance and will describe some research that illustrates how progress is being made. These are behavioral genetics, brain damage or dysfunction, and psychological traumatization. These each have impacts on individuals that increase the risks of criminality.[17] Furthermore, we often see a clustering of risk factors, and it is sometimes the combination of two or more of these factors that results in a criminal act rather than a single risk acting alone.[18]

I will conclude that the growth of research in neuroscience and criminality has taken us to a point at which we need a "Copernican revolution" in criminal justice. This would be a paradigm shift from a system based on prescientific beliefs, such as free will and good and evil, to one based on a scientific worldview.[19]

I. BEHAVIORAL GENETICS

Research into genetic factors in criminality has been carried out for many decades.[20] The two main sources of information have been twin and adoption studies.[21]

In twin studies, the heritability of a characteristic is assessed by comparing concordance rates in monozygotic (MZ) and same-sex

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14. *See* Cave, *supra* note 5, at 70, 73.

15. *See id.* at 70, 72-73.

16. *See, e.g.*, Jesper Ryberg, *Neuroscience and Criminal Justice: Introduction*, 18 J. ETHICS 77, 77 (2014).

17. *See* Andrea L. Glenn & Adrian Raine, *Neurocriminology: Implications for the Punishment, Prediction and Prevention of Criminal Behaviour*, 15 NATURE REVS. NEUROSCIENCE 54, 54, 56, 59 (2014).

18. *See id.* at 59.

19. *See* THOMAS S. KUHN, THE COPERNICAN REVOLUTION: PLANETARY ASTRONOMY IN THE DEVELOPMENT OF WESTERN THOUGHT 1-4 (1957).

20. *See* GAIL S. ANDERSON, BIOLOGICAL INFLUENCES ON CRIMINAL BEHAVIOR 97 (2007).

21. *See id.* at 95.

dizygotic (DZ) twins.[22] The concordance rate is the probability that both twins will possess a characteristic if one member of the twin pair does so.[23] Monozygotic twins are genetically identical.[24] Dizygotic twins have the same genetic similarities as non-twin siblings.[25] Differences in concordance rates can separate the impact of genetic and environmental factors in the causation of crime.[26] Twin studies of adult crime uniformly show higher concordance rates for MZ than DZ twins.[27] MZ concordance was on average around 50 percent, while DZ concordance was less than half of this.[28]

In adoption studies, the prevalence of a characteristic under consideration is ascertained in a cohort of adoptees.[29] The relative influence of genetic and environmental factors is assessed by comparing the prevalence of the characteristic in the adoptive and biological families.[30] A landmark adoption study was carried out in Denmark by Mednick and others in 1984.[31] The researchers ascertained criminal convictions in adopted sons and their biological and adoptive parents.[32] If neither set of parents had a criminal record, the conviction rate in sons was 13.5 percent.[33] If the adoptive parents, but not the biological parents, had a criminal record, the conviction rate in sons was slightly higher at 14.7 percent.[34] If the biological parents alone had a criminal record, the conviction rate in the sons was significantly higher at 20 percent.[35] If both biological and adopted parents had been convicted, the rate in sons was higher still at 24.5 percent.[36] Moreover, there was a correlation

22. *Id.* at 92.
23. *Id.*
24. *Id.* at 88.
25. *Id.*
26. *See id.* at 92.
27. *Id.* at 96.
28. *Id.* (showing that DZ concordance is around 20 percent, on average).
29. *Id.* at 105.
30. *Id.*
31. Sarnoff A. Mednick et al., *Genetic Influences in Criminal Convictions: Evidence from an Adoption Cohort*, 224 SCI. 891 (1984).
32. *Id.* at 891.
33. *Id.* at 892.
34. *Id.*
35. *Id.*
36. *Id.*

between the number of offenses in the biological parents and the number in adoptees.[37]

Both twin and adoption studies therefore point to genetic inheritance as one risk factor for crime.[38] But we need much more detail if this research is to be of practical use. We need a more precise description of what inherited characteristics increase the risk of criminality. We need to know which genes are important and how they produce effects on behavior. We need to know how these genes interact with each other and with environmental causes.

One focus of research has been on callous-unemotional (CU) personality traits in children.[39] CU traits include diminished ability to feel guilt, failure to show emotions, lack of concern about school performance, and lack of concern for the feelings of others,[40] and are found in a group of children who are at high risk of developing persistent antisocial behavior.[41]

Twin studies have shown that such traits are highly heritable.[42] In one study, the heritability of antisocial behavior was compared in children with high levels of CU traits and children with lower levels of CU traits.[43] Antisocial behavior in high-CU children was almost entirely influenced by genes with very little environmental impact.[44] In low-CU children, antisocial behavior was influenced by both genes and environment.[45] The high heritability of CU traits has been confirmed in more recent studies.[46] Takahashi and others

37. *Id.* at 892-93.

38. *See, e.g.*, *id.* at 893; ANDERSON, *supra* note 20, at 96.

39. *See, e.g.*, Yusuke Takahashi et al., *Genetic and Environmental Influences on the Developmental Trajectory of Callous-Unemotional Traits from Childhood to Adolescence*, 62 J. CHILD PSYCH. & PSYCHIATRY 414 (2021); Rebecca Waller et al., *Parenting Is an Environmental Predictor of Callous-Unemotional Traits and Aggression: A Monozygotic Twin Differences Study*, 57 J. AM. ACAD. CHILD & ADOLESCENT PSYCHIATRY 955 (2018); Essi Viding et al., *Evidence for Substantial Genetic Risk for Psychopathy in 7-Year-Olds*, 46 J. CHILD PSYCH. & PSYCHIATRY 592 (2005).

40. Eva R. Kimonis et al., *Assessing Callous-Unemotional Traits in Adolescent Offenders: Validation of the Inventory of Callous-Unemotional Traits*, 31 INT'L J.L. & PSYCHIATRY 241, 243 (2008) (listing the twenty-four items included in the Inventory of Callous-Unemotional Traits).

41. Takahashi et al., *supra* note 39, at 414.

42. *See, e.g.*, Viding et al., *supra* note 39, at 595-96.

43. *Id.* at 593.

44. *Id.* at 595.

45. *Id.* at 595-96.

46. *See* Takahashi et al., *supra* note 39, at 419-20.

assessed CU traits in twin pairs at ages seven, nine, twelve, and sixteen.[47] The heritability of CU traits at baseline was high at 76.5 percent.[48] There was an independent genetic influence on the developmental course of these traits.[49] It was also clear that environmental influences had a major role on whether these traits were maintained, increased over time, or decreased.[50]

One environmental factor that may be important is quality of parenting.[51] Waller and others studied the relationship between parental harshness and warmth with differences in CU traits and aggression in MZ twin pairs.[52] Parental harshness was related to both childhood aggression and CU traits.[53] Low parental warmth was related to CU traits but not aggression.[54] This raises the possibility that intervention in families aimed at improving parenting practices might lower the emergence of aggression and CU traits in children.

A major development over the past twenty years has been research into gene-environment interactions. There are six recognized "nonspecific neurotransmitter projection systems each having its origin in nuclei located in the brainstem."[55] These are specified by the neurotransmitter secreted at the axon terminals (dopamine, serotonin, noradrenaline (norepinephrine), adrenaline (epinephrine), histamine, and acetylcholine).[56] These are said to be nonspecific because they have modulating effects on widely distributed areas of the brain.[57] Abnormalities in these systems have been implicated in the causation of illnesses such as schizophrenia, affective disorders, obsessive-compulsive disorder, and others.[58] The enzyme monoamine oxidase A (MAOA) plays a major role in the

47. *Id.* at 416.
48. *Id.* at 419.
49. *Id.* at 420.
50. *Id.*
51. *See* Waller et al., *supra* note 39, at 955.
52. *Id.* at 957-58.
53. *Id.* at 958.
54. *Id.* at 955, 959.
55. JOHN S. CALLENDER, FREE WILL AND RESPONSIBILITY: A GUIDE FOR PRACTITIONERS 118 (2010).
56. *Id.*
57. *Id.*
58. *Id.*

metabolism of neurotransmitters such as serotonin, noradrenaline, and dopamine, thus rendering them inactive.[59]

The significance of this enzyme in the study of crime first emerged in 1993, when Brunner and others described a family in which several males were affected by a syndrome of borderline mental handicap and abnormal behavior, which took forms such as impulsive aggression, arson, attempted rape, and exhibitionism.[60] The aggression exhibited by these men tended to occur in response to "anger, fear, or frustration."[61] This syndrome was associated with a complete and selective deficiency of MAOA.[62] This was caused by an abnormality of the structural gene for MAOA, which is found on the X chromosome.[63] MAOA activity in carrier females did not differ from noncarrier controls.[64] These women were of normal intelligence and behavior.[65]

This mutation is extremely rare, but there is a range of polymorphisms of this gene which leads to differing levels of enzyme activity.[66] We now know that there are at least seven polymorphisms in the MAOA gene that take the form of variable number tandem repeats (VNTRs).[67] In terms of activity, these can be divided into one group of high-activity variants (MAOA-H) and another of low-activity variants (MAOA-L).[68]

A landmark study was carried out by Caspi and others which examined the impact of these polymorphisms in a large sample of male children.[69] They were divided into one group with MAOA-H (63 percent of the sample) and another with MAOA-L (37 percent).[70]

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59. *Id.*

60. H. G. Brunner et al., *Abnormal Behavior Associated with a Point Mutation in the Structural Gene for Monoamine Oxidase A*, 262 SCI. 578, 578-79 (1993).

61. *Id.* at 579.

62. *Id.*

63. *Id.* at 578.

64. *Id.*

65. *Id.*

66. *See id.* at 579.

67. *See* Nathan J. Kolla & Marco Bortolato, *The Role of Monoamine Oxidase A in the Neurobiology of Aggressive, Antisocial, and Violent Behavior: A Tale of Mice and Men*, 194 PROGRESS NEUROBIOLOGY, no. 101875, 2020, at 1, 5-6.

68. *Id.* at 5.

69. Avshalom Caspi et al., *Role of Genotype in the Cycle of Violence in Maltreated Children*, 297 SCI. 851 (2002).

70. *Id.* at 853 & n.30.

The subjects of this study were assessed on a range of parameters at ages three, five, seven, nine, eleven, thirteen, fifteen, eighteen, twenty-one, and twenty-six.[71] Between the ages of three and eleven, 8 percent of the study sample experienced "severe" maltreatment, 28 percent "probable" maltreatment, and 64 percent no maltreatment.[72]

It is well known that childhood maltreatment is a risk factor for antisocial behavior.[73] Boys who are abused are at risk of developing conduct disorder and antisocial personality, and of committing violent offenses.[74] However, there are large variations in outcome.[75] Despite the increased risk, most maltreated children do not become delinquents or criminals.[76]

In the Caspi study, antisocial behavior was determined in terms of four criteria: diagnosis of conduct disorder in adolescence; convictions for violent crimes; disposition toward violence determined by psychological assessment at age twenty-six; and confirmation of antisocial personality symptoms by collecting information from a third party well known to the study participant.[77]

MAOA-L alone did not increase the risk of overall antisocial behavior, whereas the experience of maltreatment led to a significantly increased risk.[78] The effect of childhood maltreatment on antisocial behavior was significantly weaker among males with MAOA-H.[79] This relationship was also found when the specific domains of antisocial behavior were examined.[80]

Maltreated males with MAOA-L were more likely to develop adolescent conduct disorder than non-maltreated males with this genotype.[81] In contrast, for males with MAOA-H, maltreatment did not confer a significant risk of conduct disorder.[82] For adult violent conviction, maltreated males with MAOA-L were more likely than

71. *Id.* at 852.
72. *Id.*
73. *Id.* at 851.
74. *Id.*
75. *Id.*
76. *Id.*
77. *Id.* at 852.
78. *Id.* at 853.
79. *Id.*
80. *Id.*
81. *Id.*
82. *Id.*

MAOA-L, non-maltreated males to be convicted of a violent crime.[83] In MAOA-H males, maltreatment did not confer a significant risk of violent offending.[84] MAOA-L, maltreated males comprised 12 percent of the study group but committed 44 percent of the violent crimes.[85]

With regard to self- or informant-reported disposition to violence, MAOA-L, maltreated males had higher antisocial scores than their MAOA-L, non-maltreated counterparts.[86] In all, 85 percent of MAOA-L, severely maltreated males developed some form of antisocial behavior.[87] In contrast, maltreatment did not lead to higher antisocial scores in MAOA-H males.[88]

The most interesting overall finding of this study is the demonstration of a link between a gene and the environment.[89] Those who had been maltreated did not differ from others in their MAOA activity, suggesting that genotype does not influence exposure to maltreatment.[90] In the absence of childhood maltreatment, MAOA status had no effect on antisocial behavior.[91] In contrast, high MAOA activity significantly diminished the risk of antisocial behavior in those who had been maltreated.[92]

This research has been extended and replicated over the past twenty years.[93] It was reviewed by Kolla and Bortolato in a paper published in 2020.[94] Several studies have shown that the MAOA-L alleles are inherently associated with antisocial behavior, psychopathy, and, especially, criminal violence.[95] One particular variant—2R—leads to very low enzyme activity and is associated with high levels of violent delinquency.[96]

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83. *Id.*
84. *Id.*
85. *Id.*
86. *Id.*
87. *Id.*
88. *Id.*
89. *Id.* at 851.
90. *See id.* at 853.
91. *Id.*
92. *Id.*
93. Kolla & Bortolato, *supra* note 67, at 8.
94. *Id.*
95. *Id.* at 7.
96. Kevin M. Beaver et al., *The 2-Repeat Allele of the MAOA Gene Confers an Increased Risk for Shooting and Stabbing Behaviors,* 85 PSYCHIATRIC Q. 257, 262-63 (2014).

A study in Finland of a large cohort of incarcerated offenders compared MAOA status in nonviolent offenders, violent offenders implicated in at least one violent crime, and very violent offenders who had committed at least ten violent crimes.[97] The MAOA-L genotype was related to violent offending and even more so to repeated violent offending.[98] There was almost no link between MAOA status and nonviolent offending, suggesting that this effect is specific to violence.[99] The risks were not modified by a history of violent abuse.[100]

This effect may even operate on the unborn child. One study examined the links between adverse life events in pregnancy and negative emotionality in five-week-old infants, and whether this was modified by the MAOA status of the child.[101] It was found that a history of four or more adverse events in pregnancy, compared to none, was associated with a greater than three-times risk of fussing or crying in response to the Neonatal Behavioural Assessment at five weeks in infants with MAOA-L.[102] No such association was found in MAOA-H infants.[103] However, life events were associated with other indices of social stress, such as marital dissatisfaction and single parent status; it may therefore be that these events were a proxy for other environmental risks rather than being directly causal.[104]

Research on MAOA polymorphisms has been focused on males for two main reasons. The first is that violent behavior is more common in men.[105] The second is that the gene for the enzyme is carried on the X chromosome.[106] Because of the single X chromosome in males,

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97. J. Tiihonen et al., *Genetic Background of Extreme Violent Behavior*, 20 MOLECULAR PSYCHIATRY 786, 787 (2015).

98. *Id.* at 788.

99. *Id.* at 791.

100. *Id.* at 787-88.

101. J. Hill et al., *Evidence for Interplay Between Genes and Maternal Stress* in Utero*: Monoamine Oxidase A Polymorphism Moderates Effects of Life Events During Pregnancy on Infant Negative Emotionality at 5 Weeks*, 12 GENES, BRAINS & BEHAV. 388, 389 (2013).

102. *Id.* at 391.

103. *Id.*

104. *Id.* at 393.

105. Angelica Staniloiu & Hans Markowitsch, *Gender Differences in Violence and Aggression—A Neurobiological Perspective*, 33 PROCEDIA—SOC. & BEHAV. SCIS. 1032, 1032 (2012).

106. F. Ducci et al., *Interaction Between a Functional* MAOA *Locus and Childhood Sexual*

a man will possess only one copy of the gene.[107] A woman will either be homozygous (two copies of the gene) or heterozygous (different variants at the allele).[108] This makes it more difficult to interpret gene-environment interactions in women.[109]

The findings of research in women have been less consistent than in men. One study showed that women who had experienced childhood sexual abuse and who were homozygous for MAOA-L were more likely to develop adult antisocial personality disorder and alcohol use disorders than women who were homozygous for the high-activity allele.[110] Women who were heterozygous had an intermediate risk.[111] In contrast, other studies have found that it is MAOA-H that increases the risk of problem behaviors in women who have had adverse childhood experiences.[112]

There is much work still to be done on the complex interplay between MAOA variants with other genes and environmental factors. Nevertheless, Kolla and Bortolato come to a confident conclusion:

> [T]he interplay between low *MAOA* genetic variants and early-life adversity is the best-documented gene × environment (G × E) interaction in the pathophysiology of aggression and [antisocial behavior].... For more than two decades, MAOA has secured its place as one of the most intensely scrutinized genes in psychiatric research and is arguably the most important in the study of human aggression. We fully expect that the synergism of genetic, neuroimaging, and animal research on this gene and its gene product will point to new horizons for understanding the widespread—yet still largely elusive—problem of violence and aggression.[113]

Abuse Predicts Alcoholism and Antisocial Personality Disorder in Adult Women, 13 MOLECULAR PSYCHIATRY 334, 344-45 (2008).

107. *See* Caspi et al., *supra* note 69, at 853 n.30.

108. *Id.*

109. *Id.*

110. Ducci et al., *supra* note 106, at 338.

111. *Id.*

112. *See, e.g.*, Amy L. Byrd & Stephen B. Manuck, MAOA*, Childhood Maltreatment, and Antisocial Behavior: Meta-Analysis of a Gene-Environment Interaction*, 75 BIOLOGICAL PSYCHIATRY 9, 15 (2014); *cf.* I. Hyun Ruisch et al., *Interplay Between Genome-Wide Implicated Genetic Variants and Environmental Factors Related to Childhood Antisocial Behavior in the UK ALSPAC Cohort*, 269 EUR. ARCHIVES PSYCHIATRY & CLINICAL NEUROSCIENCE 741, 746 (2019).

113. Kolla & Bortolato, *supra* note 67, at 1, 16.

Aside from some inconsistencies, the picture presented is one that shows MAOA-L as something that causes nothing but harm. But this is where the story takes an intriguing turn. The model that has been implicitly applied is the *diathesis-stress* one.[114] The diathesis (or disposition) is a characteristic that, if present, renders the individual more vulnerable to environmental stress or adversity.[115]

An alternative model is *differential susceptibility*.[116] This postulates that some individuals will be more disposed to the effects of adversity, for genetic or other reasons, but the same disposition will mean that they do better in more congenial environments.[117] In the diathesis-stress model, the person is worse off in adverse circumstances, but no better if these are absent.[118] The differential susceptibility model predicts that the person will not only be worse off in adverse circumstances, but also will do better in environments that are benign and positive.[119] The differential susceptibility model does not treat absence of abuse as the positive end of the environmental spectrum and the absence of mental disorder, such as depression, as the positive end of the spectrum of mental well-being.[120] Instead, it goes beyond this to examine the impacts of better-than-average environments and the presence of better-than-average self-regulation and social functioning.[121]

It is known that children and adolescents who receive warm, sensitive, and supportive care are better able to "concentrate on tasks, regulate their emotions under challenging circumstances and engage in goal-directed behavior" than those whose parenting is hostile, unresponsive, disengaged, or neglectful.[122] This phenomenon was examined in an interesting study of a large cohort of adolescents by Belsky and Beaver in 2011.[123] It drew on data from

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114. *See, e.g.*, J. Belsky et al., *Vulnerability Genes or Plasticity Genes?* 14 MOLECULAR PSYCHIATRY 746, 746 (2009).

115. *Id.* at 747.

116. *Id.*

117. *Id.*

118. *See id.* at 746-47.

119. *Id.* at 747.

120. *Id.*

121. *See id.* at 747, 749.

122. Jay Belsky & Kevin M. Beaver, *Cumulative-Genetic Plasticity, Parenting and Adolescent Self-Regulation*, 52 J. CHILD PSYCH. & PSYCHIATRY 619, 619 (2011).

123. *Id.*

a long-term, longitudinal study of American adolescents.[124] The environmental factor studied was maternal parenting quality, and the outcome was emotional self-regulation.[125]

The study was designed to test the differential susceptibility model.[126] It examined the impacts not only of MAOA-L, but also of four other enzyme "plasticity alleles": DAT1 10R; DRD2 A1; DRD4 7R; and 5HTTLPR.[127] These enzymes were selected on the basis of prior research that individuals with these genes might be affected "for-better-*and*-for-worse" by a range of environmental factors.[128] All of the enzymes play a role in the functioning of dopamine and/or serotonin in the central nervous system.[129] It was hypothesized that this would enhance sensitivity both to pleasure and rewards, and also to displeasure and punishment.[130]

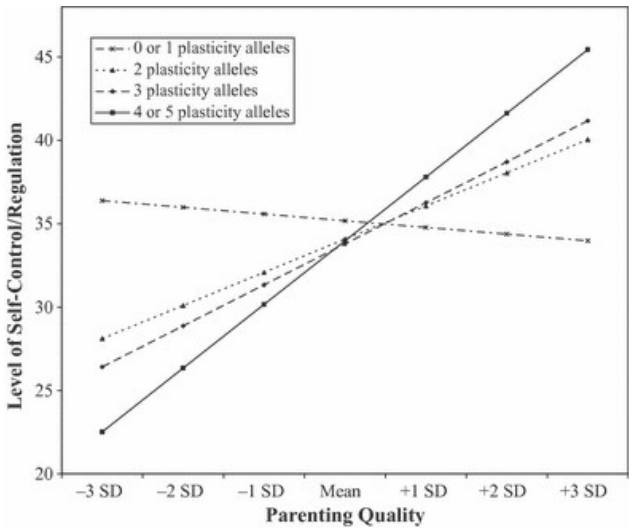
Maternal parenting quality and adolescent self-regulation were assessed by a set of detailed questions at the time of enrollment into the study.[131] At that time, the participants were in eleventh grade and sixteen to seventeen years old.[132] All subjects were tested for the presence of the five plasticity alleles, and these were combined to give a cumulative plasticity index.[133]

The first main finding was a striking confirmation of the differential susceptibility model.[134] Second, it was shown that the more plasticity alleles possessed by males, the more they were harmed by poor parenting, and the more they benefitted from good parenting.[135] Thus, male research subjects with zero or one plasticity allele were little affected one way or the other by parenting, while those with four or five plasticity alleles were most harmed by poor parenting

124. *Id.* at 621.
125. *Id.* at 622.
126. *Id.* at 619-20, 624.
127. *Id.* at 622.
128. *Id.* at 620.
129. *Id.* at 621.
130. *Id.*
131. *Id.* at 622.
132. *See id.* at 621.
133. *Id.* at 622.
134. *Id.* at 624.
135. *Id.*

and derived the greatest benefit from good parenting.[136] This effect was not seen in females.[137]

Figure 1. Interaction Between Cumulative Genetic Plasticity and Parenting Quality in the Prediction of Self-Regulation for Males[138]



This study raises many important scientific questions. We need to know if this is simply an additive effect of genes or whether there are complex interactions between different genes and between genes and the environment. We also need to know why the effect is confined to males.

It may be that this research has major implications for how we respond to adolescent delinquents, especially in relation to incarceration. If there are offenders whose genetic makeup renders them highly susceptible to environmental influences, perhaps the worst thing we can do is place them in environments that are traumatizing, hostile, and punitive; that is, a typical youth detention

136. *Id.*
137. *Id.* at 623-25.
138. *Id.* at 624 (figure reproduced with permission).

facility. Conversely, such individuals may derive exceptional benefit from environments that are caring and supportive.

It is important to note that research in this area has resulted in inconsistent findings.[139] There are many possible reasons for this, such as complex interactions between the genes that have been studied and epigenetic effects in which environmental factors lead to changes in gene expression.[140] It is likely that there are other genes whose impacts have yet to be discovered.[141]

Childhood maltreatment and adverse experiences come in many shapes, forms, combinations, durations, ages of onset, and levels of severity, and will occur in the context of other malign influences, such as poverty, deprivation, and substance misuse.[142] Almost all children who have been physically or sexually abused have also experienced emotional neglect or abuse.[143] Outcomes will be determined by the set of experiences that someone has been exposed to, and it can be difficult to make links between particular forms of maltreatment and later effects.[144]

Antisocial behavior is a complex phenomenon whose nature and severity are also highly variable. One distinction that is often drawn is between "life-course-persistent" and "adolescent-limited" antisocial behavior.[145] We need to develop a more fine-grained description of different adverse childhood experiences and later antisocial behaviors if we want to better understand the relationships between them.[146]

We know that a small number of individuals are responsible for a disproportionate amount of offending. Moffitt and others found that 10 percent of "life-course-persistent" antisocial males were

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139. *See generally* Kent W. Nilsson et al., *Gene-Environment Interaction of Monoamine Oxidase A in Relation to Antisocial Behaviour: Current and Future Directions*, 125 J. NEURAL TRANSMISSION 1601 (2018).

140. *See id.* at 1601, 1610, 1616.

141. *See* J. Wertz et al., *Genetics and Crime: Integrating New Genomic Discoveries into Psychological Research About Antisocial Behavior*, 29 PSYCH. SCI. 791, 791 (2018) (discussing a genome-wide association study which allows scientists to identify many different genetic variants that might be associated with a particular trait).

142. *See* Nilsson et al., *supra* note 139, at 1614-15.

143. *Id.* at 1614.

144. *Id.* at 1614-15.

145. *Id.* at 1602.

146. *See* David D. Vachon et al., *Assessment of the Harmful Psychiatric and Behavioral Effects of Different Forms of Child Maltreatment*, 72 JAMA PSYCHIATRY 1135, 1140-41 (2015).

responsible for over 50 percent of violent offending at age twenty-six.[147] Despite the limitations of our present knowledge, it seems clear that genetic factors and gene-environment interactions are playing important causal roles in antisocial behavior. It is likely that a better understanding of these roles will contribute to our understanding of such persons, and will help us to mitigate, and perhaps prevent, the harms they cause to other people and themselves.

II. BRAIN DYSFUNCTION AND CRIME

In 1848, Phineas Gage was twenty-five years old and working as a construction foreman for a railway company.[148] He sustained a severe injury when a tamping iron weighing more than thirteen pounds was blown into his face by an explosive detonation.[149] It entered just below his left cheekbone, traversed his skull, and exited through the top of his head, landing more than one hundred feet away, covered in brains and blood.[150] Amazingly, Gage did not lose consciousness.[151] He recovered from this injury with no obvious neurological deficit.[152] Since this event, he has become one of the most famous case studies in cognitive neuroscience.[153]

The reason for this is the profound impact of the injury on Gage's personality and moral character. Before the injury, he was said by Dr. John Harlow to have had "temperate habits," "considerable energy of character," and "a well balanced mind."[154] After the accident, the "equilibrium or balance, so to speak, between his intellectual faculty and animal propensities" had been lost.[155]

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147. Terrie E. Moffitt et al., *Males on the Life-Course-Persistent and Adolescence-Limited Antisocial Pathways: Follow-Up at Age 26 Years*, 14 DEV. & PSYCHOPATHOLOGY 179, 179, 187, 191 (2002).

148. ANTONIO R. DAMASIO, DESCARTES' ERROR: EMOTION, REASON, AND THE HUMAN BRAIN 3 (1994).

149. *Id.* at 4, 6.

150. *Id.* at 4.

151. *Id.*

152. *Id.* at 6-7.

153. Mo Costandi, *Phineas Gage and the Effect of an Iron Bar Through the Head on Personality*, THE GUARDIAN (Nov. 8, 2010, 7:04 AM), https://www.theguardian.com/science/blog/2010/nov/05/phineas-gage-head-personality [https://perma.cc/E248-X6XB].

154. DAMASIO, *supra* note 148, at 8.

155. *Id.*

> [He became] fitful, irreverent, indulging at times in the grossest profanity which was not previously his custom, manifesting but little deference for his fellows, impatient of restraint or advice when it conflict[ed] with his desires, at times pertinaciously obstinate, yet capricious and vacillating, devising many plans of future operation, which are no sooner arranged than they are abandoned.[156]

He was fired shortly after returning to his former employment.[157] He died in 1861 after suffering a prolonged epileptic seizure.[158]

The main brunt of the injury suffered by Phineas Gage was borne by the ventro-medial prefrontal cortex (VMPFC).[159] The prefrontal cortex subserves "executive" functions, such as thinking, planning, problem-solving, and emotional and behavioral control.[160] Damage to the orbito-frontal cortex (OFC) leads to impairment of response inhibition.[161] This can lead to disinhibited behaviors, labile emotions, impulsivity, and lack of concern for others.[162]

The frontal cortex has been a focus of research into the links between brain dysfunction and crime for many years. Early studies by Raine and others used positron emission tomography to study convicted murderers.[163] They were divided into two groups.[164] The first was termed "affective" murderers.[165] In these cases, the crime was impulsive or "hot blooded" and was usually carried out in response to provocation by the victim or other persons.[166] The second group

156. *Id.*
157. *Id.*
158. *Id.* at 10.
159. *Id.* at 32.
160. *See* Shintaro Funahashi & Jorge Mario Andreau, *Prefrontal Cortex and Neural Mechanisms of Executive Function*, 107 J. PHYSIOLOGY – PARIS 471, 471-72 (2013).
161. *See generally* Daniel W. Bryden & Matthew R. Roesch, *Executive Control Signals in Orbitofrontal Cortex During Response Inhibition*, 35 J. NEUROSCIENCE 3903, 3903 (2015).
162. *See* EDMUND T. ROLLS, THE ORBITOFRONTAL CORTEX 139 (2019).
163. *See generally* Adrian Raine et al., *Reduced Prefrontal and Increased Subcortical Brain Functioning Assessed Using Positron Emission Tomography in Predatory and Affective Murderers*, 16 BEHAV. SCIS. & L. 319 (1998).
164. *Id.* at 319.
165. *Id.* at 320.
166. *Id.*

comprised "predatory" murders.[167] Here, the violent act was controlled, purposeful, and "cold blooded."[168]

The affective murderers had lower levels of functioning in the prefrontal cortex compared to controls, and high levels of activity in subcortical areas involved with emotional responsiveness.[169] The predatory murderers had similar high levels of subcortical activity but had prefrontal functioning that was close to normal.[170]

The authors suggested that a high level of subcortical activity predisposes the person to aggressive behavior.[171] In the predatory group, there was sufficient prefrontal activity to regulate and control these impulses until an opportune moment to strike arose.[172] In the affective group, prefrontal activity was insufficient to contain the aggressive drive, and the result was impulsive violence.[173]

The idea of violence arising from a combination of over-activity in emotion-generating areas of the brain and under-performance in areas involved in self-control has been a main focus of research into antisocial behavior and violence. The subcortical area that has received the most attention is the amygdala.[174] This is a complex structure, present in the anterior temporal lobes, which has wide connections to other brain structures.[175] It has reciprocal connections to areas involved in the reception and processing of sensory information.[176] It also connects to the brainstem and plays a role in the physiological responses to emotion-generating stimuli.[177] Finally, it has connections to the VMPFC and the OFC.[178]

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167. *Id.*
168. *Id.*
169. *Id.* at 324-25.
170. *See id.*
171. *Id.* at 319.
172. *Id.* at 329.
173. *Id.* at 328.
174. *See, e.g.*, *id.* at 321.
175. *See id.* at 321, 328-29; Nilsson et al., *supra* note 139, at 1611.
176. *See* Raine et al., *supra* note 163, at 329; Nilsson et al., *supra* note 139, at 1611.
177. Yiran Gu et al., *A Brainstem-Central Amygdala Circuit Underlies Defensive Responses to Learned Threats*, 25 MOLECULAR PSYCHIATRY 640, 640-41 (2020).
178. *See* R. J. R. Blair, *Traits of Empathy and Anger: Implications for Psychopathy and Other Disorders Associated with Aggression*, 373 PHIL. TRANSACTIONS ROYAL SOC'Y BIOLOGICAL SCI., no. 20170155, 2018, at 1, 3; Abigail A. Marsh et al., *Reduced Amygdala-Orbitofrontal Connectivity During Moral Judgments in Youths with Disruptive Behavior Disorders and Psychopathic Traits*, 194 PSYCHIATRY RSCH.: NEUROIMAGING 279, 279 (2011).

In a paper reviewing this subject, Blair proposed that abnormal aggression arises from a combination of increased anger and decreased empathy for the distress of the victim.[179] Irritability and an increased risk for reactive aggression are associated with "(i) heightened responsiveness of the amygdala and [the peri-acqueductal gray area] in response to threat and social provocation; (ii) heightened responsiveness of the striatum to negative prediction errors (the unexpected absence of reward) and (iii) dysfunction in potential anger regulatory roles [in the VMPFC and lateral frontal cortex.]"[180]

The propensity to cause harm to another person is increased if there is a breakdown in empathy with the victim.[181] Empathy draws on a range of capacities. These include inhibition of aggression in response to the distress of the victim; learning the negative effects of actions, such as aggression, that create distress in others; and reasoning about actions that cause distress.[182] The amygdala is known to respond to distress cues and can act to freeze aggressive actions.[183] If this response is impaired, the risk of aggression is increased.[184] These capacities are also vulnerable to damage to the frontal areas, and such damage will further increase the risk of violence.[185]

Acquired brain injury (ABI) is a blanket term that covers any disability arising from brain damage acquired after birth,[186] or in the course of prenatal development.[187] Causes include traumatic injury, malnutrition, environmental toxins such as lead, abuse of alcohol and drugs, hypoxic injury such as opiate overdose, stroke, brain tumors, and emotional neglect and abuse.[188] Prenatal brain

179. Blair et al., *supra* note 178, at 1-2.

180. *Id.* at 5.

181. *Id.* at 3.

182. *Id.* at 2-3.

183. *Id.* at 2.

184. *Id.*

185. *See id.*

186. *Brain Injury Overview*, BRAIN INJ. ASS'N OF AM., https://www.biausa.org/brain-injury/about-brain-injury/basics/overview [https://perma.cc/9YYE-85PC].

187. *See* Adre du Plessis, *Brain Injury in the Fetus*, *in* ACQUIRED BRAIN INJURY IN THE FETUS AND NEWBORN 1, 1 (Michael Shevell & Steven Miller eds., 2012).

188. BRAIN INJ. ASS'N OF AM., *supra* note 186. Timothy M. Marshall et al., *Neurotoxicity Associated with Traumatic Brain Injury*, *Blast*, *Chemical*, *Heavy Metal and Quinoline Drug Exposure*, 25 ALT. THERAPIES 28, 28 (2019) (explaining that continuous exposure to toxins from the environment, such as lead, can result in brain damage); *Causes of Acquired Brain*

damage can arise from malnutrition and from exposure to drugs and alcohol.[189]

Traumatic brain injury can be caused by a single, severe injury, in which case the link between injury and behavioral change may be obvious.[190] In other individuals, the disability arises from the cumulative effects of a series of minor injuries.[191] This can happen with a child who is repeatedly battered or shaken, or in sportspersons who experience multiple head trauma.[192] In these cases, the behavioral changes are more insidious and may be incorrectly attributed to willful, antisocial conduct.[193]

The impacts of brain injury are highly variable and depend on a range of factors, such as the severity of the injury and location of the damage.[194] The sequelae of the initial injury can also contribute to the final outcome; for example, acquiring one brain injury increases the risk of subsequent brain injuries, perhaps due to impaired judgment arising from the initial injury.[195]

One problem in this area has been that lesions that precede the onset of criminal acts arise in different parts of the brain.[196] This seeming inconsistency can be resolved by the fact that brain structures do not act in isolation from each other.[197] Complex behaviors

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Injury, N. BRAIN INJ. ASS'N, https://www.nbia.ca/causes-brain-injury/ [https://perma.cc/ DS46-7KJ2] (discussing how brain tumors can cause acquired brain injury); WORLD HEALTH ORG., NEUROLOGICAL DISORDERS: PUBLIC HEALTH CHALLENGES 111-12 (2006) (explaining that malnutrition in children may lead to neurological disorders).

189. du Plessis, *supra* note 187, at 4, 9.

190. *See Traumatic Brain Injury*, MAYO CLINIC, https://www.mayoclinic.org/diseases-conditions/traumatic-brain-injury/symptoms-causes/syc-20378557 [https://perma.cc/JTS4-5DBQ].

191. *Chronic Traumatic Encephalopathy*, MAYO CLINIC, https://www.mayoclinic.org/diseases-conditions/chronic-traumatic-encephalopathy/symptoms-causes/syc-20370921 [https://perma.cc/DLM6-UM8D] (explaining that repeated head trauma can lead to brain injury known as chronic traumatic encephalopathy (CTE)).

192. *See id.*; T. Joyce et al., *Pediatric Abusive Head Trauma*, STATPEARLS (Aug. 26, 2021), https://www.ncbi.nlm.nih.gov/books/NBK499836/ [https://perma.cc/E2TV-57SF].

193. *See Traumatic Brain Injury*, JOHNS HOPKINS MED., https://www.hopkinsmedicine.org/health/conditions-and-diseases/traumatic-brain-injury [https://perma.cc/BQQ5-ACE4].

194. *Complications*, NAT'L HEALTH SERV., https://www.nhs.uk/conditions/severe-head-injury/complications/ [https://perma.cc/6X3E-M5MB].

195. *See generally* Oliver Lasry et al., *Epidemiology of Recurrent Traumatic Brain Injury in the General Population*, 89 NEUROLOGY 2198 (2017).

196. *See generally* R. Ryan Darby et al., *Lesion Network Localization of Criminal Behavior*, 115 PROC. NAT'L ACAD. SCIS. 601 (2018) (finding separate brain lesions associated with criminal behavior).

197. *See id.* at 602-05.

require the operation of networks of brain areas that act in concert.[198] Lesion-induced symptoms can arise from changes in function in areas connected to the lesion and not just from the site of the lesion itself.[199] Therefore, specific behavioral changes, such as criminality, may arise if any of the structures in a given network are damaged.[200]

Darby and others described seventeen cases in which there was a clear temporal relationship between developing a brain lesion and the onset of criminal behavior.[201] The lesions were located in a functional network comprising the medial prefrontal cortex, the OFC, and different parts of the temporal lobes.[202] No location was affected in all subjects, but all of the lesions were in areas that formed part of the same functional network.[203] This network was distinct from lesions causing other neuropsychiatric syndromes.[204] It involved regions known to have roles in moral thinking, value-based decision-making, and theory of mind, but not regions with roles in empathy and cognitive control.[205] The researchers then studied another group in which the temporal relationship between lesion and the onset of criminality was less certain, and obtained very similar results.[206]

These are intriguing findings. The fact remains that most patients with lesions in these areas do not turn into criminals. When it comes to human actions, however, there are few single causes that are both necessary and sufficient to produce the action. Most actions arise from a complex web of causation that includes immediate provocations and longer term predisposing factors. If it is clear that a brain lesion was a necessary causal factor, even if it was not sufficient, it should provide grounds for mitigation or even excuse.

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198. *Brain Basics: Know Your Brain*, NAT'L INST. OF NEUROLOGICAL DISORDERS & STROKE, https://www.ninds.nih.gov/Disorders/Patient-Caregiver-Education/Know-Your-Brain [https://perma.cc/6HTR-DESU].

199. Darby et al., *supra* note 196, at 601.

200. *Id.*

201. *Id.* at 605.

202. *Id.* at 601.

203. *Id.* at 602.

204. *Id.* at 603.

205. *Id.* at 601.

206. *Id.* at 604.

Brain injury can lead to cognitive impairments such as memory loss and poor concentration.[207] The prefrontal cortex serves functions such as forward planning, emotional and behavioral self-control, and problem-solving.[208] Damage to this area can cause impulsivity and impaired emotional regulation.[209] There may be loss of inhibition and the emergence of aggression and inappropriate behaviors.[210]

There are many studies of rates of brain damage or dysfunction in offender populations. Williams and others carried out a survey of adult male prisoners in the United Kingdom.[211] Sixteen percent had experienced moderate to severe traumatic brain injury and 48 percent had a history of mild injury.[212] Prisoners who had suffered head injuries were younger at entry into custodial systems, had higher rates of repeat offending, and had spent more time in prison in the previous five years.[213]

Brewer-Smyth and others found that *95 percent* of incarcerated female offenders showed evidence of some form of neurological abnormality that had predated the index offense.[214] Forty-two percent of subjects reported at least one brain injury with loss of consciousness.[215] Rates were higher in those convicted of violent offenses, who reported a mean of two brain injuries with loss of consciousness per subject.[216] Eighty percent of violent offenders had experienced physical abuse in childhood and seventy percent had been sexually abused.[217]

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207. MAYO CLINIC, *supra* note 190.

208. Shazia Veqar Siddiqui et al., *Neuropsychology of Prefrontal Cortex*, 50 INDIAN J. PSYCHIATRY 202, 204 (2008).

209. *Id.* at 206-07.

210. *Id.*

211. W. Huw Williams et al., *Traumatic Brain Injury in a Prison Population: Prevalence and Risk for Re-Offending*, 24 BRAIN INJ. 1184, 1185 (2010).

212. *Id.* at 1184.

213. *Id.*

214. Kathleen Brewer-Smyth et al., *Physical and Sexual Abuse, Salivary Cortisol, and Neurologic Correlates of Violent Criminal Behavior in Female Prison Inmates*, 55 BIOLOGICAL PSYCHIATRY 21, 21 (2004).

215. *Id.* at 28.

216. *Id.* at 26.

217. *Id.* at 24.

A study by Lansdell and others highlighted the importance of ABI in perpetrators as a risk factor in domestic violence.[218] Victims, usually a female partner, are often subjected to blows to the head, or to nonlethal strangulation, which can cause hypoxic brain injury.[219] Brain-damaged perpetrators can produce brain-damaged victims. Violence is usually repeated, sometimes over long periods of time, and sometimes at escalating levels of severity.[220] Children can be traumatized by witnessing violence perpetrated by one parent against the other,[221] and by the other effects of having one, or perhaps two, brain-damaged parents.[222]

III. TRAUMATIZATION AND CRIME

The third area that I wish to explore is the relationship between psychological traumatization and crime. This is important in itself, but it also provides a framework for bringing together and understanding some of the research discussed above.

First, it helps us to understand the relationship between MAOA-L and crime. There is considerable evidence of over-activity of the sympathetic nervous system and excess release of neurotransmitters, such as noradrenaline, in patients with post-traumatic stress disorder (PTSD).[223] The person with low-activity MAOA may be more susceptible to the impact of this, because of impaired ability to break down these transmitters.[224] A drug known as prazosin, which blocks receptors for norepinephrine, is an effective treatment

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218. *See generally* Gaye T. Lansdell et al., *Strengthening the Connection Between Acquired Brain Injury (ABI) and Family Violence: The Importance of Ongoing Monitoring, Research and Inclusive Terminology*, J. FAM. VIOLENCE, May 2021, at 1.

219. *Id.* at 2.

220. *Id.* at 1, 3, 8.

221. Jayne O'Donnell & Mabinty Quarshie, *The Startling Toll on Children Who Witness Domestic Violence Is Just Now Being Understood*, USA TODAY (Jan. 31, 2019, 7:03 PM), https://www.usatoday.com/story/news/health/2019/01/29/domestic-violence-research-children-abuse-mental-health-learning-aces/2227218002/ [https://perma.cc/YT7Q-LF9M].

222. Linda F. Pessar et al., *The Effects of Parental Traumatic Brain Injury on the Behaviour of Parents and Children*, 7 BRAIN INJ. 231, 231 (1993).

223. Roger K. Pitman et al., *Biological Studies of Post-Traumatic Stress Disorder*, 13 NATURE REVS. NEUROSCIENCE 769, 775 (2012).

224. Dean A. Stetler et al., *Association of Low-Activity* MAOA *Allelic Variants with Violent Crime in Incarcerated Offenders*, 58 J. PSYCHIATRIC RSCH. 69, 69 (2014).

for PTSD symptoms such as physiological over-arousal and nightmares.[225]

Second, it helps explain the link between violence and the combination of over-activity in emotion-generating areas of the brain and under-activity in areas, such as the frontal lobes, that are to do with self-control. The amygdala plays a central role in the detection of threat, the expression of fear, and the heightening of memories for emotional events.[226] Functional neuro-imaging studies in PTSD patients have demonstrated exaggerated amygdala and decreased VMPFC responses to trauma-related stimuli.[227] The failure of the VMPFC to inhibit the amygdala may lead to increased attentional bias to threat, impaired extinction of traumatic memories, and deficits in emotion regulation.[228]

A third factor is that environments in which there is a risk of PTSD are environments in which there is also a risk of traumatic brain injury. The child who is physically abused will be at risk of cumulative brain damage from blows to the head.[229] The child who is born with Fetal Alcohol Spectrum Disorder will likely grow up with a mother who is abusing alcohol and will be exposed to all the risks of neglect and abuse that arise from this.[230] Soldiers in combat are at risk of both PTSD and traumatic brain injury.[231] This can include damage to the VMPFC and other frontal areas. These injuries are neurologically "silent," meaning they do not lead to obvious symptoms such as muscle weakness or paralysis.[232] Damage may therefore not be detected.[233]

A final complication can arise when the traumatized person uses alcohol or other drugs as a form of self-medication. This can further

225. Pitman et al., *supra* note 223, at 775.

226. *Id.* at 772.

227. *Id.*

228. *Id.* at 773.

229. *See* T. Joyce et al., *supra* note 192.

230. *See* Alan Price et al., *Prenatal Alcohol Exposure and Traumatic Childhood Experiences: A Systematic Review*, 80 NEUROSCIENCE & BIOBEHAVIORAL REVS. 89, 89-90 (2017).

231. Caitlin Kennedy & Sandy Wang, *Traumatic Brain Injury and Post-Traumatic Stress Disorder in Military Veterans: When Two Problems Collide*, NAT'L CTR. FOR HEALTH RSCH., https://www.center4research.org/traumatic-brain-injury-post-traumatic-stress-disorder-military-veterans-two-problems-collide/ [https://perma.cc/H72N-NA54].

232. *See* Lansdell et al., *supra* note 218, at 3.

233. *See id.*

weaken inhibitions against violence.[234] Alcohol abuse renders the person vulnerable to brain injury from accidents or violence.[235] A damaged brain can cause disabilities, such as lack of foresight and loss of inhibitions.[236] It can expose the person to risk of further harm, for example, by repeated injury.[237] A vicious circle is created when it becomes increasingly difficult for the person with a damaged brain to control an addiction to alcohol.

Research into the relationships between traumatic experiences and subsequent criminality has been carried out for many years. According to one authority, traumatization followed by the victimization of other people is "a major cause of violence in society."[238]

When one considers the biographies of those who have been responsible for murder and mayhem on the grand scale, it is striking how often one finds a history of early traumatization and abuse. Adolf Hitler's father was an authoritarian, overbearing, and irritable drunkard.[239] He was repeatedly violent to his wife and children.[240] According to his sister, the young Adolf "got his sound thrashing every day."[241] On one occasion, his father beat him so badly that he left him for dead.[242] Joseph Stalin's father was a violent, drunken cobbler who "savagely beat" his wife and child.[243] Stalin's mother in turn, as he later recalled, "thrashed him mercilessly."[244] The traumatized child will sometimes grow up and take a terrible revenge on the world.

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234. *See Alcohol Alert*, NAT'L INST. ON ALCOHOL ABUSE & ALCOHOLISM (Oct. 1997), https://pubs.niaaa.nih.gov/publications/aa38.htm [https://perma.cc/R4G8-VXF2] (explaining that alcohol "weakens brain mechanisms that normally restrain impulsive behaviors").

235. *See Alcohol*, WORLD HEALTH ORG. (Sept. 21, 2018), https://www.who.int/news-room/fact-sheets/detail/alcohol [https://perma.cc/TU69-CAVT].

236. NAT'L HEALTH SERV., *supra* note 194.

237. *See id.*

238. Bessel A. van der Kolk & Alexander C. McFarlane, *The Black Hole of Trauma*, *in* TRAUMATIC STRESS: THE EFFECTS OF OVERWHELMING EXPERIENCE ON MIND, BODY, AND SOCIETY 3, 11 (Bessel A. van der Kolk et al. eds., 2007).

239. IAN KERSHAW, HITLER, 1889-1936: HUBRIS 11-12 (1998).

240. *Id.* at 13.

241. *Id.*

242. JONATHAN H. PINCUS, BASE INSTINCTS: WHAT MAKES KILLERS KILL? 188 (2001); *see also* Suzy Hansen, *The Mind of A Killer*, SALON (July 27, 2001, 7:00 PM), https://www.salon.com/2001/07/27/killers_3/ [https://perma.cc/UJ8G-SNPN] (describing an interview with Dr. Pincus).

243. SIMON SEBAG MONTEFIORE, STALIN: THE COURT OF THE RED TSAR 26 (2003).

244. *Id.*

A common consequence of traumatization is the development of PTSD.[245] Other psychiatric syndromes can also arise, such as depression, substance misuse, and personality disorder.[246] The salient features of PTSD fall into three groups.

The first are intrusive memorizations.[247] These can take the form of flashbacks, in which the patient reexperiences the traumatic event, along with its associated emotions, and bad dreams or nightmares.[248] Flashbacks may be so intense as to be frankly hallucinatory.[249] These may be precipitated by triggering events, which remind the patient of the traumatizing event.[250]

The second group of symptoms arises from physiological overarousal.[251] If bad things happen to people, they can develop a pervasive sense that the world is a dangerous, threatening place.[252] The body responds to threat by going into *fight or flight mode*, meaning a state of readiness to fight off the threat or run away from it.[253] This causes symptoms such as anxiety, tension, insomnia, poor concentration, increased startle response, irritability, and quickness to anger.[254] The link between a "fight" reaction to stress and an increased likelihood of violence is an obvious one.[255] The person in this frame of mind is on a physiological hair-trigger.

The person may be constantly scanning the environment for threat.[256] There may be hostile attribution errors, such as seeing a

245. SUBSTANCE ABUSE & MENTAL HEALTH SERVS. ADMIN., U.S. DEP'T OF HEALTH & HUM. SERVS., A TREATMENT IMPROVEMENT PROTOCOL: TRAUMA-INFORMED CARE IN BEHAVIORAL HEALTH SERVICES 10 (2014).

246. *Id.* at 4.

247. *Id.* at 82.

248. *Id.*

249. *Id.*

250. *Id.*

251. *Id.* at 82-83.

252. *See id.*

253. *See Causes: Post-Traumatic Stress Disorder*, NAT'L HEALTH SERV., https://www.nhs.uk/mental-health/conditions/post-traumatic-stress-disorder-ptsd/causes/ [https://perma.cc/H5TA-Z6AS] (explaining that those with PTSD are found to produce heightened adrenaline levels even when not in physical danger).

254. *See* Valerie Rosen & Gayle Ayers, *An Update on the Complexity and Importance of Accurately Diagnosing Post-Traumatic Stress Disorder and Comorbid Traumatic Brain Injury*, 15 NEUROSCIENCE INSIGHTS, Jan.-Dec. 2020, at 1, 2-3.

255. *See* SUBSTANCE ABUSE & MENTAL HEALTH SERVS. ADMIN., *supra* note 245, at 83.

256. *See id.*

threat where it does not exist.[257] High levels of emotional arousal can diminish frontal cortex activity.[258] The end result is someone who is prone to hostility and suspiciousness.[259] All of this may be exacerbated if the person uses alcohol and drugs as a way of achieving temporary relief from his distress.[260]

The third group of symptoms arises from the need of the sufferer to find a safe space.[261] The nature of this need will depend on the traumatizing event or events. It may comprise avoidance of places or situations that provoke recollections of the trauma.[262] There may also be a general social withdrawal, estrangement from people, and restricted emotions.[263]

A more sinister development is when the traumatized person becomes a perpetrator.[264] In the words of the poet W. H. Auden, "[t]hose to whom evil is done[, d]o evil in return."[265] One mechanism for this is "identification with the aggressor."[266] The victim of abuse feels helpless, weak, and humiliated.[267] In contrast, his abuser is strong, powerful, and in control.[268] The victim deals with his helplessness by assuming the mantle of the powerful abuser.[269] This provides an outlet for his anger and desire for revenge, and often involves feelings of contempt for his victims. In these cases, the propensity to harm does not arise from a failure of empathy. These offenders well understand the distress that they are causing, and this is the whole point of what they do.

The relationship between childhood maltreatment and juvenile delinquency has been a subject of research for many years. High rates of severe traumatization and PTSD have been found in

257. *See id.*
258. *See* Kolla & Bortolato, *supra* note 67, at 2.
259. *See* SUBSTANCE ABUSE & MENTAL HEALTH SERVS. ADMIN., *supra* note 245, at 83.
260. *See id.* at 89.
261. *See id.* at 82.
262. *See id.*
263. *See id.*
264. *See, e.g.*, Yael Lahav et al., *Identification with the Aggressor and Inward and Outward Aggression in Abuse Survivors*, J. INTERPERSONAL VIOLENCE, July 2020, at 1, 2.
265. W.H. AUDEN, SEPTEMBER 1, 1939, at l.21-22 (1939).
266. *See, e.g.*, Lahav et al., *supra* note 264, at 4.
267. *See id.* at 4-6.
268. *See id.*
269. *See id.* at 5.

offender populations.[270] Fondacaro and others reported that 40 percent of male inmates had experienced childhood sexual abuse.[271] Cauffman and others found that only 12 percent of female juvenile offenders had *no history* of traumatization.[272] Half of this cohort met criteria for PTSD at the time of the study and 65 percent had a lifetime history of PTSD,[273] these rates being nearly six times higher than the general population.[274] Dixon and others found rates of PTSD of 37 percent in female juvenile offenders, with sexual abuse being the precipitating event in 70 percent of these.[275]

Lynch and others found a prevalence of PTSD of 53 percent in incarcerated female offenders, compared to 10 percent in the general population.[276] Furthermore, women in the United States prison system are more likely than men to experience sexual assault by fellow inmates and by staff.[277] The high rates of traumatization and PTSD in female offenders should be a matter of particular concern in the United States, where the number of women incarcerated increased by *750 percent* between 1980 and 2017.[278]

Braga and others carried out a meta-analysis of prospective, longitudinal studies of the link between maltreatment and juvenile antisocial behavior in both males and females.[279] Maltreatment

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270. *See, e.g.*, Karen M. Fondacaro et al., *Psychological Impact of Childhood Sexual Abuse on Male Inmates: The Importance of Perception*, 23 CHILD ABUSE & NEGLECT 361, 361-62 (1999).

271. *Id.* at 366.

272. Elizabeth Cauffman et al., *Posttraumatic Stress Disorder Among Female Juvenile Offenders*, 37 J. AM. ACAD. CHILD & ADOLESCENT PSYCHIATRY 1209, 1215 (1998).

273. *Id.* at 1212.

274. *Id.* at 1210, 1212 (comparing the 1 percent and 14 percent incidence in general population with the 65 percent incidence in study subjects).

275. Angela Dixon et al., *Trauma Exposure, Posttraumatic Stress, and Psychiatric Comorbidity in Female Juvenile Offenders*, 44 J. AM. ACAD. CHILD & ADOLESCENT PSYCHIATRY 798, 801 (2005).

276. Shannon M. Lynch et al., *A Multisite Study of the Prevalence of Serious Mental Illness, PTSD, and Substance Use Disorders of Women in Jail*, 65 PSYCHIATRIC SERVS. 670, 673-74 (2014).

277. ALLEN J. BECK ET AL., U.S. DEP'T OF JUST., SEXUAL VICTIMIZATION REPORTED BY ADULT CORRECTIONAL AUTHORITIES, 2009-11 1, 9, 12 (2014), https://bjs.ojp.gov/content/pub/pdf/svraca0911.pdf [https://perma.cc/MV49-QKAQ].

278. STORM ERVIN ET AL., URBAN INST., ADDRESSING TRAUMA AND VICTIMIZATION IN WOMEN'S PRISONS: TRAUMA-INFORMED VICTIM SERVICES AND PROGRAMS FOR INCARCERATED WOMEN 1 (2020), https://www.urban.org/research/publication/addressing-trauma-and-victimization-womens-prisons [https://perma.cc/S86R-TKRM].

279. Teresa Braga et al., *Unraveling the Link Between Maltreatment and Juvenile Antiso-*

was defined as neglect, physical abuse, sexual abuse, emotional abuse, or overall maltreatment.[280] The main findings were that maltreatment is associated with higher rates of general antisocial behaviors and aggression.[281] Physical and sexual abuse were more strongly linked to aggression than to general antisocial behavior.[282] Young people who were neglected were at greater risk of antisocial behaviors.[283] There are many factors that increase the risks of both maltreatment and delinquency.[284] These include family stress, family configuration, parent-child relationships, child-rearing skills, and parental psychopathology.[285]

The association between maltreatment and delinquency may arise from these common causes, as well as maltreatment directly causing delinquency.[286] Studies that controlled for family dysfunction found weaker links between maltreatment and delinquency.[287] Although common causal factors had a moderating effect on this association, there was a residual direct relationship between maltreatment and delinquency.[288]

Adolescence is the stage in which the risks of violent offending and violent behaviors are at their peak.[289] Wojciechowski described a technique known as group-based trajectory modeling and its use in investigating the relationships between traumatization and violence in juvenile offenders.[290] These are young people who have come to the attention of the criminal justice system and who have therefore shown some propensity for antisocial behavior.[291]

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cial Behavior: A Meta-Analysis of Prospective Longitudinal Studies, 33 AGGRESSION & VIOLENT BEHAV. 37, 37, 40 (2017).

280. *Id.* at 39.

281. *Id.* at 46.

282. *Id.*

283. *Id.*

284. *Id.* at 38.

285. *Id.*

286. *Id.*

287. *Id.* at 41 ("[C]ontrolling for family functioning yield[ed] a smaller effect size compared to not controlling for this characteristic.").

288. *Id.* at 47-48.

289. Thomas W. Wojciechowski, *PTSD as a Risk Factor for the Development of Violence Among Juvenile Offenders: A Group-Based Trajectory Modeling Approach*, 35 J. INTERPERSONAL VIOLENCE 2511, 2517 (2020).

290. *Id.* at 2513.

291. *Id.* at 2512.

This study was part of a Pathways to Desistance project.[292] It recruited 1,354 adolescents who were aged between fourteen and eighteen at the time of the index offense and followed them for seven years.[293] All study participants were assessed in relation to symptoms of PTSD.[294] With regard to violent offending, adolescents fell into one of four groups.[295] The first and largest of these showed little or no violent offending in the course of the study.[296] The second showed a moderate level of violence that remained stable.[297] The third showed high levels of violence at the start, followed by desistance and a decline in violence to very low levels over seven years.[298] The final group exhibited high levels of violence which showed only a small decline over time.[299] A history of PTSD more than doubled the risk of adolescents falling into one of the violent groups, compared to the group that exhibited little or no violence.[300]

The links between traumatization and violence in military personnel have also been extensively studied.[301] MacManus and others described a meta-analysis of seventeen studies of violence in United Kingdom and United States military personnel who had deployed to Iraq and Afghanistan.[302] Violent behavior was common with a pooled estimate of physical assault of 10 percent in the previous month and 29 percent for all types of physical aggression.[303] The majority of studies found an association between combat exposure and post-deployment violence and aggression.[304] Several studies concluded the risk of violence increased with the "intensity and frequency" of traumatic experiences in combat.[305]

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292. *Id.* at 2513.
293. *Id.* at 2517.
294. *Id.* at 2518-19.
295. *Id.* at 2523-25.
296. *Id.* at 2523-24.
297. *Id.*
298. *Id.* at 2524.
299. *Id.* at 2524-25.
300. *Id.* at 2525-26.
301. *See, e.g.*, Deirdre MacManus et al., *Aggressive and Violent Behavior Among Military Personnel Deployed to Iraq and Afghanistan: Prevalence and Link with Deployment and Combat Exposure*, 37 EPIDEMIOLOGIC REVS. 196, 197 (2015).
302. *Id.* at 197-98.
303. *Id.* at 205-06.
304. *Id.* at 208.
305. *Id.*

One study looked at violence in United Kingdom military personnel who served in Iraq in 2003.[306] Violence was more likely in those who had a combat role and the experience of multiple traumatic events in the course of deployment.[307] PTSD symptoms and heavy drinking were also both strongly associated with post-deployment violence.[308]

How should criminal justice systems respond to offenders who suffer from psychiatric syndromes that have been caused by prior traumatization? A baseline principle of medical ethics is "first, do no harm."[309] Traumatized offenders should be identified, and the system should ensure that inmates are not exposed to further assault and harm that will perpetuate and exacerbate existing damage. This should be motivated not only by basic humanity, but also by the need to reduce the risks of recidivism.

Although much is being done,[310] services are often poorly funded and inadequate. One report on female offenders in the United States calls for prison services to be *trauma-sensitive* (that is, for staff to be aware of the high levels of traumatization in offenders); *trauma-informed* (aware of the impacts of trauma on people); *trauma-responsive* (services should have policies and practices that diminish harm and create opportunities for change); and *traumaspecific* (that is, provision of treatments to "promote healing and recovery").[311]

If traumatization is such an important cause of crime, why do we not do more to alleviate its impacts on offenders? One reason is that trauma-related disorders generally do not meet criteria for legal excuse[312] and will, at best, constitute grounds for mitigation.[313] The

306. D. MacManus et al., *Violent Behaviour in UK Military Personnel Returning Home After Deployment*, 42 PSYCH. MED. 1663, 1664 (2012).

307. *Id.* at 1669.

308. *Id.* at 1666.

309. Robert H. Shmerling, *First, Do No Harm*, HARV. HEALTH BLOG (June 22, 2020), https://www.health.harvard.edu/blog/first-do-no-harm-201510138421 [https://perma.cc/P8UF-VCYH].

310. ERVIN ET AL., *supra* note 278, at 25.

311. *Id.* at 6.

312. *See, e.g.*, MODEL PENAL CODE § 4.01 (AM. L. INST. 1985) (stating that to excuse responsibility, an individual must "lack[] substantial capacity either to appreciate the criminality (wrongfulness) of his conduct or to conform his conduct to the requirements of law"); M'Naghten's Case, 8 Eng. Rep. 718, 722-23 (H.L. 1843) (holding to excuse responsibility, an individual's affliction must cause them to not understand either the nature of their action or

person is still considered responsible for his wrongdoing.[314] It can be argued that not everyone who is traumatized ends up with a mental disorder,[315] and even fewer commit serious crimes. The correct riposte to this might be to adduce the many other factors, also without the control of the offender, which contributed to his crime (such as a damaged brain). Instead, we assume that, despite all of the trauma, the person is still deserving of punishment.[316]

A more fundamental problem is that prisons are not therapeutic institutions; they are institutions of punishment.[317] The conditions of life are, often by design, harsh, austere, and degrading.[318] The organizational culture of punishment is quite different from the culture of therapy.[319] To punish people is to inflict harm, and this can be difficult to reconcile with the impulse to confer benefit. If therapy is being offered, this entails a recognition that the crime was the result of factors beyond the conscious control of the offender. If this is recognized, how do we justify the harm that we are inflicting on people?

I would argue that what is needed is not more therapy grafted on to institutions of punishment. A skin graft or a transplanted organ will be rejected by the immune system of the host, unless specific measures are taken to prevent this.[320] Something analogous can

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that their action itself was right or wrong).

313. MODEL PENAL CODE § 4.02(2) (AM. L. INST. 1985) (stating "mental disease or defect" is applicable as a mitigating factor in capital cases).

314. *Id.* § 4.01.

315. *See Post-Traumatic Stress Disorder*, NAT'L INST. OF MENTAL HEALTH,, https://www.nimh.nih.gov/health/publications/post-traumatic-stress-disorder-ptsd [https://perma.cc/Y2QZ-CV4U].

316. WAYNE R. LAFAVE, SUBSTANTIVE CRIMINAL LAW § 7.1(d) (3d ed. 2020) ("A number of informed observers believe that it is therapeutically desirable to treat behavioral deviants as responsible for their conduct rather than as involuntary victims playing a sick role.").

317. Alexis B. Apel & James W. Diller, *Prison as Punishment: A Behavior-Analytic Evaluation of Incarceration*, 40 BEHAV. ANALYST 243, 244 (2017) (stating the purpose of imprisonment in the United States is "operant punishment").

318. *Id.* at 245-46 ("[S]ome [governments] even advocate for intentionally harsh conditions during incarceration under the philosophy that they deter offenders from committing future crimes.").

319. Etienne Benson, *Rehabilitate or Punish?*, AM. PSYCH. ASS'N (July/Aug. 2003), https://www.apa.org/monitor/julaug03/rehab [https://perma.cc/THD7-M34V] (finding that prisons are "much less likely to rehabilitate their inhabitants" after the pivot to punishment theory in the 1970s).

320. JULIA DEATHRIDGE, BRIT. SOC'Y FOR IMMUNOLOGY, TRANSPLANT IMMUNOLOGY 1 (2017), https://www.immunology.org/sites/default/files/Transplant%20Immunology.pdf

happen to therapy when it encounters the skepticism, cynicism, and authoritarianism that can dominate punitive environments. What is needed is a wholesale paradigm shift in how we understand and respond to crime in our societies.

IV. CRIMINAL JUSTICE AND PARADIGM SHIFT

Thomas Kuhn argued that science proceeds in two distinct ways.[321] In "normal" phases, there is a steady accumulation of knowledge and the growing approximation of theories to underlying reality.[322] This depends on the commitment of a given scientific community to a shared "paradigm," in other words, a particular set of theories, beliefs, techniques, and methodologies.[323] The paradigm refers to the "shared elements [that] account for the relatively unproblematic character of professional communication and the relative unanimity of professional judgment."[324] The paradigm provides a framework in which scientific observations are interpreted and understood.[325] The inculcation of this paradigm becomes part of the mindset of the scientist.[326]

This process continues until anomalies arise that cannot be understood by the underlying paradigm.[327] At first, these may be ignored or explained away.[328] As anomalies accumulate, the underlying paradigm becomes increasingly insecure.[329] These lead to a point of "paradigm shift" at which it becomes necessary to find a new paradigm that better explains empirical findings and does a better job of solving scientific problems.[330] In addition, Kuhn argues that a paradigm shift leads to new observations.[331] The reason for this is that a given paradigm creates an observational perspective

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[https://perma.cc/XV8Z-CQUK].

321. THOMAS S. KUHN, THE STRUCTURE OF SCIENTIFIC REVOLUTIONS 5-6 (4th ed. 2012).

322. *Id.* at 24.

323. THOMAS S. KUHN, THE ESSENTIAL TENSION: SELECTED STUDIES IN SCIENTIFIC TRADITION AND CHANGE 294, 296 (1977).

324. *Id.* at 297.

325. KUHN, *supra* note 321, at 11.

326. *Id.*

327. Ian Hacking, *Introductory Essay* to KUHN, *supra* note 321, at xxvi.

328. *Id.*

329. *Id.* at xxvii.

330. *Id.* at xxiii.

331. *Id.* at xxxiv.

and expectation of what is out there, and this leads to focusing on observations that fit with the paradigm and neglecting those that do not.[332]

Kuhn also argued that different paradigms are *incommensurable*.[333] This means that the language and theories of one paradigm cannot be translated to the other and that paradigms cannot be rationally evaluated against each other.[334] It becomes possible for scientists to make and understand certain statements only after a new paradigm has been introduced.[335] Under the old paradigm, these statements may be nonsensical.[336]

An oft-quoted example of paradigm shift is the Copernican Revolution.[337] Pre-Copernican astronomy was based on concepts formulated by Aristotle.[338] This was a geocentric cosmology, in which the Earth was at the center of the universe and celestial bodies revolved around it in different ways.[339] Observations of stars and planets revealed that these did not move in the ways predicted by the Aristotelian model.[340] In the second century, Ptolemy introduced new ideas in an attempt to explain these anomalies, but these were in turn refuted, mainly by Islamic astronomers.[341]

Copernicus set out his heliocentric theory in writings that date from the early sixteenth century.[342] This was based on the need for a theory that provided a more satisfactory explanation of astronomical observation.[343] Copernicus argued that the apparent motions of the sun and stars were explained by the rotation of the Earth, its orbit around the sun, and the annual changes in axial

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332. KUHN, *supra* note 321, at 24.

333. Eric Oberheim & Paul Hoyningen-Huene, *The Incommensurability of Scientific Theories*, STAN. ENCYCLOPEDIA OF PHIL. (Sept. 4, 2018), https://plato.stanford.edu/entries/incommensurability/ [https://perma.cc/ZY75-QSSG].

334. *Id.*

335. *Id.*

336. *Id.*

337. *See, e.g.*, Sheila Rabin, *Nicolaus Copernicus*, STAN. ENCYCLOPEDIA OF PHIL. (Sept. 13, 2019), https://plato.stanford.edu/entries/copernicus/ [https://perma.cc/ATP9-X3FM].

338. *Id.* § 2.1.

339. *Id.*

340. *Id.*

341. *Id.*

342. *Id.* §§ 2.2, 2.6.

343. *See id.* § 2.2.

inclination.[344] The apparent movements of the planets arise from a combination of the movement of the Earth and the movements of the planets.[345]

The need for an astronomical model that matched reality had a practical importance that extended well beyond the world of astronomy. The great voyages of oceanic exploration that set out from Europe used navigational techniques that depended on observations of the sun and stars.[346] In the twentieth century and onwards, space travel obviously depended on advanced astronomy.

Criminal justice doctrine and practice revolve around a paradigm comprised of beliefs and values, such as moral realism, free will, desert, and retribution.[347] The main theme of this Article is that there is a need for a shift to a new paradigm based on recent research in neuroscience and other areas of criminology.

This need arises for two main reasons. The first is that the traditional model essentially has no capacity to explain criminal behavior, beyond evoking vague metaphysical concepts such as good and evil.[348] Its foundational beliefs are insecure and prescientific. Furthermore, there is no capacity within the existing paradigm for development or improvement.

The second reason is that the present system is failing to solve the problems that it exists to address.[349] This should come as no surprise. NASA would never have entrusted its mission to fly to the moon to people who believed that the Earth was flat and the moon revolved around it, attached to a solid sphere. In the same way, a criminal justice system will only succeed in addressing the problem of crime if it has understandings of human nature and criminality that are based on solid empirical foundations.

Despite the existence of a vast and expensive apparatus of criminal punishment, we have a seemingly intractable problem of crime. Furthermore, the attempt to control this under the present

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344. *See id.*

345. *See id.*

346. *See, e.g.*, *The Ages of Exploration: Mariner's Astrolabe*, THE MARINERS' MUSEUM & PARK, https://exploration.marinersmuseum.org/object/astrolabe/ [https://perma.cc/NJ9C-8PHK].

347. *See* Callender, *supra* note 12, at 43.

348. *See, e.g.*, CALLENDER, *supra* note 55, at 29.

349. *See id.* at 85 (discussing why the use of punishment as deterrence often fails to solve the problem it is allegedly supposed to be addressing).

paradigm requires the inflicting of incalculable suffering on offenders, and enormous costs on society. In contrast, scientific research is producing an ever-growing body of knowledge that is explaining why criminality arises, and which is able to assess the effectiveness of the many ways in which societies respond to crime.[350]

I believe that these two paradigms are incommensurable in the ways described above. One obvious example of this is free will. This is a complex and much-debated concept. There are many interpretations of what free will is, or is not, and the implications that these interpretations have for moral and criminal responsibility.[351]

One interpretation is *libertarian* free will. This proposes that there are actions that are free in the sense that they are not determined by prior causes.[352] The causal chain begins with the choices and decisions of the agent.[353] It is this interpretation that underpins retributive punishment, that is, the idea that the wrongdoer deserves to be punished in proportion to his crime, and that this punishment is morally required, regardless of any consequence that might flow from it.[354]

The idea that someone should be punished on this basis depends on the belief that the commission of the crime arose from a free and conscious decision on the part of the offender, and that he had the capacity to choose not to commit the crime.[355] If instead the commission of the crime can be attributed to causes beyond the control of the offender, there is no justification for retribution.[356]

There are many *a priori* objections to libertarian free will. In the present context, the important point is that it is quite incommensurable with a scientific approach to human behavior. It is in the nature of science to look for causes and explanations of the phenomena that it studies. There is nothing in psychology, neuroscience, or

350. *See, e.g.*, Callender, *supra* note 12, at 52 (describing the effectiveness of a study where offenders were randomly selected to participate in an alternative justice procedure).

351. *See* CALLENDER, *supra* note 55, at 1.

352. *Id.* at 10.

353. *Id.*

354. *See* Oliver Burkeman, *The Clockwork Universe: Is Free Will an Illusion?*, THE GUARDIAN (Apr. 27, 2021, 1:00 PM), https://www.theguardian.com/news/2021/apr/27/the-clockwork-universe-is-free-will-an-illusion [https://perma.cc/93H4-7HPB].

355. *See id.*

356. *See id.*

elsewhere in science that allows for free (in the libertarian sense), uncaused actions. There has never been an observation that can only be explained by free will.

It is argued by some that quantum theory has revealed that reality is not always governed by deterministic rules and that this creates space for libertarian free will.[357] There are two objections to this. The first is that we have no reason to believe that quantum level phenomena have any relevance for human choice and decision-making. The second is that, even if this were the case, it creates only randomness, and not the free will required for attributions of responsibility.

It is sometimes said that these causal factors, to use a phrase of Leibniz, "incline without necessitating."[358] Thus, it may be acknowledged that there were forces at work that had a malign influence on the actions of the offender. These may be excusing in the small number of cases that amount to legal insanity. If they are not excusing, they may be mitigating, and hence lead to a more lenient punishment. The belief remains, however, that they do not entirely remove the capacity for choice, and that the core of free will continues to operate.

Thus, we might acknowledge that the offender was a peaceful, law-abiding citizen and only became violent after he sustained a traumatic brain injury to his frontal lobes, but still insist that this did not remove criminal responsibility. We might take a similar view if the same hitherto peaceful, law-abiding citizen became violent only after he developed the paranoid delusion that the police were trying to kill him.

If we insist, despite it all, that the action that he performed was an action that was also in his power not to perform, we introduce something mysterious and inexplicable into the causal matrix. It follows from this that there are actions that cannot be subsumed under any causal principle and that there will never be a complete science of human behavior.

This has an important consequence for any attempt to shift the paradigm. It entails that *no matter how much we learn* from

357. *See, e.g.*, Barry Loewer, *Freedom from Physics: Quantum Mechanics and Free Will*, 24 PHIL. TOPICS 91, 93 (1996).

358. William Rowe, *Divine Freedom*, STAN. ENCYCLOPEDIA OF PHIL. (July 31, 2007), https://plato.stanford.edu/entries/divine-freedom/ [https://perma.cc/8HVR-4JJ9].

neuroscience, psychology, sociology, and other disciplines about the causes of crime, "free will" is always there as a trump card that will win against anything that science can offer.

The belief that we are possessed of free will is one that accords with our everyday, unreflective, subjective sense of how we operate in the world. In the words of Dr. Samuel Johnson, "We *know* our will is free, and *there's* an end on't."[359] The idea that things are not as they appear and that we are moved by forces that operate below the level of conscious awareness is not an easy one to accept.

Nevertheless, the concept of free will has consequences that are pernicious in the context of criminal justice. It leads to a failure to understand the nature and causes of criminal behavior and the disabilities that many people experience in conforming their behavior to the law.[360] The result is punishments that are unjust and which often (or usually) fail to achieve any positive outcome for the offender, his victim(s), or society.[361] It can lead to offenders who are vulnerable because of mental illness, traumatization, or brain injury being placed in harsh, brutal environments that only make a bad situation worse.[362]

The accumulation of knowledge about the causes of criminal behavior is now at the point that it cannot be ignored. The criminal justice system must take this on board and modify its practices. It might be argued that there is no need for a wholesale paradigm shift and that the findings of neuroscience can be subsumed by existing doctrines of mitigation and excuse. There are reasons for doubt about this.

Behavioral genetic evidence has begun to appear in criminal courts.[363] The main interest has come from defense attorneys, who have argued that such evidence shows that the actions of the accused arise, at least in part, from genetic predisposition.[364] The

359. SAMUEL ARTHUR BENT, FAMILIAR SHORT SAYINGS OF GREAT MEN WITH HISTORICAL AND EXPLANATORY NOTES 300 (5th ed. 1887) (first emphasis added).

360. *See supra* Parts II-III.

361. *See infra* text accompanying note 403.

362. *See supra* text accompanying notes 317-20.

363. Nicholas Scurich & Paul S. Appelbaum, *Behavioural Genetics in Criminal Court*, 1 NATURE HUM. BEHAV. 772, 772 (2017).

364. *Id.*

corollary of this is that responsibility is diminished and the person is less deserving of punishment.[365]

In fact, the impacts of such evidence in the courts have so far been limited. At most, they have resulted in reduction of the severity of the charges faced by the accused.[366] Furthermore, many experiments have been carried out on judges and the lay public to assess the effects of behavioral genetic and other neuroscientific evidence on perceptions of responsibility and liability to punishment.[367] This research has found impacts that vary from marginal to nonexistent.[368]

Why should this be? I would suggest that scientific evidence will struggle to make an impact *if it is being applied in a nonscientific paradigm*, one in which free will and responsibility are accepted as facts, and punishment for wrongdoing is seen as intrinsically appropriate.

The reality is that the paradigm of medical-scientific explanation and the paradigm of free will, responsibility, and condign punishment are entirely separate and incompatible. Old concepts will have to be reappraised in the light of new scientific findings. One example is the person who is callous and unemotional. A traditional view might be that this is someone who is bad or even wicked. But if we accept that callous-unemotionality is a trait that is inherited and that a person has no choice in the matter, moral opprobrium becomes inappropriate. Callous-unemotionality becomes a problem to be managed. We need to find ways of helping such people live in society in ways that do not lead to harm to others. For example, it may be that callous-unemotional children are unlikely to respond to threats of punishment but will respond to positive reinforcement and appeals to self-interest.[369] It may even be possible to find ways in which this trait can be utilized for the benefit of society.

Science can no more diminish responsibility or mitigate punishment than it can prove the existence of angels or that Beethoven's Fifth Symphony is a great piece of music. What science states is

365. *Id.*
366. *Id.*
367. *See id.* at 773.
368. *See id.*
369. *See* Essi Viding et al., *Aetiology of the Relationship Between Callous-Unemotional Traits and Conduct Problems in Childhood*, 190 BRIT. J. PSYCHIATRY 33, 37-38 (2007).

that free will and responsibility are illusions and hangovers from a prescientific age. If we are going to make progress in criminal law, we need to dispense with illusions and hangovers and face reality.

What hope is there for a transformation such as this? The criminal justice system is powerful and deeply embedded in our societies. All powerful systems have enormous inertia and do not change easily or quickly. Its practitioners have not been educated in a scientific milieu or tradition. Max Planck pointed out that "a new scientific truth does not triumph by convincing its opponents and making them see the light, but rather because its opponents eventually die, and a new generation grows up that is familiar with it."[370]

Nevertheless, the new neuroscience has become a major part of the *zeitgeist* of the twenty-first century.[371] Younger people are more aware of neuroscience research and its implications than was the case a generation ago.[372] The road from where we are now to where we should end up in the future will be a long and hard one. But every journey has to start somewhere.

V. IMAGINING THE FUTURE

The application of medical-scientific explanation to criminality has a long and controversial history. At present, this model is applied only to a small group of offenders with serious mental illnesses, such as psychoses.[373] These are offenders who are deemed to be legally insane using criteria such as the M'Naghten Rule.[374]

The trial of Daniel M'Naghten in 1843 was arguably one of the most renowned trials in legal history. M'Naghten attempted to assassinate the British Prime Minister, Robert Peel.[375] As a result of mistaken identity, he shot Peel's secretary instead.[376] After a

370. KUHN, *supra* note 321, at 150 (quoting MAX PLANCK, SCIENTIFIC AUTOBIOGRAPHY AND OTHER PAPERS 33-34 (1949)).

371. *See* Richard E. Brown, *Why Study the History of Neuroscience?*, 13 FRONTIERS BEHAV. NEUROSCIENCE 1, 4 (2019).

372. *See id.*

373. *See M'Naughten Rule*, LEGAL INFO. INST., https://www.law.cornell.edu/wex/m%27naughten_rule [https://perma.cc/EY6Y-46WC].

374. *See id.*

375. *See id.*

376. *See id.*

lengthy trial, M'Naghten was found "not guilty, being insane" and sent to Bethlem Hospital.[377]

This acquittal led to an outcry in the press and the House of Lords.[378] A panel of judges was established to determine how the issue of insanity should be handled in courts.[379] This led to the "M'Naghten Rules."[380] The rules stated that in order to establish a defense on the ground of insanity:

> [I]t must be clearly proved that, at the time of the committing of the act, the party accused was labouring under such a defect of reason, from disease of the mind, as not to know the nature and quality of the act he was doing; or if he did know it, that he did not know he was doing what was wrong.[381]

This is a very restrictive definition, the specific intent of which was to ensure that any future M'Naghten would face the full severity of the law. It continues to be applied in many jurisdictions.[382] It is sobering to think that a criminal court in 1843 was taking a more expansive and sympathetic approach to a mentally disordered offender than many courts are taking in the twenty-first century.

What might a science-based criminal justice system look like? In the space of a single paper, it will only be possible to paint an outline in broad brush strokes. One model that already exists is mental health legislation.[383] This allows treatment without consent of people who are mentally ill and who are a threat to the safety of themselves or other persons.[384]

There are a number of factors that make mental health legislation a suitable model. The first is the main theme of this paper, the high prevalence of mental disorders in offender populations.

Second, most prisons are already significant providers of mental health care,[385] so the model and concepts should be familiar to those

377. DAVID W. JONES, DISORDERED PERSONALITIES AND CRIME: AN ANALYSIS OF THE HISTORY OF MORAL INSANITY 74 (2016).

378. *Id.* at 74-75.

379. RALPH SLOVENKO, PSYCHIATRY AND CRIMINAL CULPABILITY 19 (1995).

380. *Id.*

381. *See* M'Naghten's Case, 8 Eng. Rep. 718, 722 (H.L. 1843).

382. SLOVENKO, *supra* note 379, at 21-22.

383. *See generally* Mental Health (Care and Treatment) (Scotland) Act 2003, (ASP 13).

384. *See id.* § 236.

385. *Criminalization of Mental Illness*, COOK CNTY. SHERIFF'S OFF.,

who work in criminal justice systems. For example, Chicago's Cook County Jail is one of the largest single-site jails in the United States.[386] Because around 50 percent of inmates suffer from mental disorders, it is also said to be the largest mental hospital in the state of Illinois, and one of the largest in the United States.[387] Many of these detainees have committed "crimes of survival."[388] In forty-four states in the United States, more people with mental illnesses are cared for in prisons and jails than in hospitals.[389]

Third, most societies have well-established bodies of legislation to cover the compulsory treatment of the mentally ill,[390] and these can provide a model for new approaches to offenders.

Fourth, because some offenders are already dealt with using mental health legislation, a precedent has been established.[391] I am proposing that this model becomes the norm, rather than one that is applied only to a small minority of offenders.

In Scotland, for example, the mentally ill person who is subject to compulsory powers must have a full diagnostic and risk assessment.[392] This must include the demonstration of significant risk to the person or others.[393] A treatment and management plan is prepared on the basis of this assessment.[394] It is necessary to state that the required treatment is available and that it cannot be provided without the use of compulsory measures.[395] This plan is submitted for independent scrutiny and approval.[396] The patient has rights of appeal against detention and treatment, and there are requirements for periodic mandatory reviews.[397] There are principles that have to

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https://www.cookcountysheriff.org/criminalization-of-mental-illness/ [https://perma.cc/G9AS-3GM2].

386. *Department of Corrections*, COOK CNTY. GOV'T, https://www.cookcountyil.gov/service/department-corrections [https://perma.cc/Y83Y-C7WV].

387. COOK CNTY. SHERIFF'S OFF., *supra* note 385.

388. *Id.*

389. *Id.*

390. *See, e.g.*, Mental Health (Care and Treatment) (Scotland) Act 2003, (ASP 13).

391. *See, e.g.*, *id.*

392. *See id.* §§ 63-65.

393. *See id.* § 64(5)(c).

394. *See id.* § 62.

395. *See id.* § 57(1).

396. *See id.* § 58(2).

397. *See id.* §§ 77-78, 324.

be applied; for example, that the aims of treatment should be achieved in the least restrictive manner.[398]

Coercive treatment of the mentally ill and coercive management of offenders have a common justification in the right of societies to protect their members from harm. Also, no society can survive without norms and standards and the power to protect itself from those who would flout these standards for personal gain.

No one can be subject to treatment under mental health legislation without a psychiatric diagnosis.[399] In the case of criminal wrongdoing, the equivalent of a diagnosis will be a trial of the facts to determine beyond a reasonable doubt that the accused committed the offense.[400]

A person who is the subject of coercive treatment under criminal law should have the equivalent of a full diagnostic assessment, which takes account of as many factors as possible. This might include the following:

> (1) Full mental health history (psychosis, brain injury, PTSD, personality disorder, and conduct disorder);
> (2) Full substance misuse history;
> (3) Assessment for specific disabilities (for example, ADHD, autism spectrum disorders, and dyslexia);
> (4) Occupational and educational history;
> (5) History of physical, emotional and sexual abuse, and neglect in childhood;
> (6) Family history of mental disorder and offending;
> (7) Scrutiny of school and social services records to confirm or ascertain evidence of abuse and neglect and conduct disorders;
> (8) Neurological assessment;
> (9) Neurocognitive testing;
> (10) Brain scanning;
> (11) Analysis of genetic risk factors (for example, MAOA-L);
> (12) Assessment for callous-unemotional traits;
> (13) Assessment of literacy and occupational skills;
> (14) Assessment of social circumstances (for example, housing, financial assets, and family support).

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398. *See id.* § 1.
399. *See* SLOVENKO, *supra* note 379, at 136.
400. *See id.* at 134-35.

This list should be used to prepare a causal account of the person's offending that is as full as possible. There should be consideration of the causal factors that are remediable and the ones that will be persistent. This should take account of factors such as neuropsychological deficits and the presence of multiple plasticity alleles. There should be an assessment of risk to public safety and the steps that can be taken to mitigate it.

Following this, a multi-professional panel should prepare a plan for rehabilitation. Where appropriate, "restorative" procedures could be applied—for example, involving the victim or victims of the crime in decisions about how to deal with the offender. This plan should be submitted for independent scrutiny and approval.

If treatment and rehabilitation are state-mandated, there is an obligation to the offender that these measures should be fully available and of high quality. Treatment should be offered for conditions such as PTSD, ADHD, psychosis, addiction, and personality disorder. Lack of basic life skills, such as literacy, should also be addressed. Offenders who are parents should be instructed in parenting skills and be given opportunities to sustain relationships with their children.

The primary aim should be reduction of recidivism. Secondary aims might include improvement of mental well-being and preparation for employment and other social roles, such as parenting. Achievement of these aims is worthwhile in itself. It will have the added benefit of reducing the long-term economic burdens of offenders on society.[401]

The duration of time in which compulsory measures will be in place should be determined by progress in completing the rehabilitation program and assessment of future risk. A principle of parsimony and least restriction should be applied. The use of coercive measures and restrictions on freedom should be the minimum required to meet the needs of rehabilitation. There should be regular independent review of the progress of offenders through the rehabilitation process.

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401. Gregg D. Caruso, *Free Will Skepticism and Its Implications: An Argument for Optimism*, *in* FREE WILL SKEPTICISM IN LAW AND SOCIETY: CHALLENGING RETRIBUTIVE JUSTICE 43, 64-65 (Elizabeth Shaw et al. eds., 2019).

It will never be possible for every convicted offender to be rehabilitated to the point at which risk to other persons is reduced to an acceptable level. The kinds of intensive assessment detailed above could contribute to assessment and management of risk. If prolonged detention is required in the interest of public safety, the restrictions on the offender should be the minimum required to ensure safety.[402] The environment in which the offender is detained should not be harsh or punitive. Efforts should be made to sustain relationships with spouses, children, and other family members.

A scientific approach does not preclude punishment in traditional forms, such as financial penalties or curtailment of liberty. There are some offenders who are simply rational economic agents, and who are not burdened with the kinds of disabilities described in this paper. These include drug traffickers, embezzlers, and the majority of "white collar" criminals. In these cases, the offender weighs the gains of crime against the costs of punishment (multiplied by the risk of being caught). A society that wishes to suppress these kinds of behaviors will have to impose costs that outweigh the gains and have effective systems to investigate crime and prepare cases for prosecution.

This approach will entail a major shift in the culture and practices of criminal justice. It is likely that there will be objections and opposition to it. One group that might be expected to have a negative view is victims of crime. In contrast to this expectation, a recent survey of crime victims in the United States found that "victims overwhelmingly prefer criminal justice approaches that prioritize rehabilitation over punishment and strongly prefer investments in crime prevention and treatment to more spending on prisons and jails."[403]

Another benefit of this approach is its relation to primary prevention of crime. The causes of acquired brain injuries and damage are well-known and widely recognized. The same applies to PTSD and other psychiatric syndromes arising from traumatization. All of these causes are either wholly or substantially preventable.

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402. *See id.* at 63-64.

403. ALL. FOR SAFETY & JUST., CRIME SURVIVORS SPEAK: THE FIRST-EVER NATIONAL SURVEY OF VICTIMS' VIEWS ON SAFETY AND JUSTICE 4 (2019), https://allianceforsafetyandjustice.org/wp-content/uploads/2019/04/Crime-Survivors-Speak-Report-1.pdf [https://perma.cc/S8PY-6EGU].

If we wish to reduce the burden of crime in our societies, we must reduce the traumatization of children, young people, and women. We know how to identify families in which there is a high risk of abuse, and we know that intervention and support can mitigate these risks.[404]

We must take all possible measures to protect the brains of our citizens from damage and injury. This is especially important when the brain is developing in fetal life and in childhood and adolescence. Possible measures include provision of universal high quality prenatal care, banning physical punishment of children, reducing risks of injuries in contact sports, improving road safety, improving occupational safety, reducing environmental pollution, reducing poverty and inequality, and reducing alcohol abuse and binge drinking.

Successful implementation of measures such as these will not only lead to reductions in crime, they will add enormously to the happiness and success of our societies.

CONCLUSION

There is now a substantial body of scientific knowledge on the causes of criminal behavior. The present paradigm, based on concepts such as free will, desert, and responsibility, has no explanatory power. It imposes incalculable suffering on offenders and enormous costs on society, but is of limited effectiveness in reducing the burden of crime in our societies.

It is time for this paradigm to give way to a model based on a realistic, empirical understanding of human nature. This proposal offers the hope of expanding our knowledge of criminality and of developing systems of criminal justice that are humane and effective.

404. Lansdell et al., *supra* note 218, at 10.