



Incorporating Biological Science into Criminological Theory

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Abstract

Most criminological theory courses will typically discuss at some point the biological influences of crime. However, more often these discussions will start and end with reference to Cesare Lombroso, who wrote the book “The Criminal Man,” where he described that criminals all appear to have a similar physical morphology and they would display specific “atavisms” to indicate their propensity to crime. This research was later debunked, but at its time it helped to spur on the debate for the eugenics movement—a political movement that took off in the early 1900s that justified the euthanizing or sterilization of the developmentally delayed, criminal, and mentally ill. Much of this history is often brought up by individuals within criminology to argue against the adoption of biological perspectives of crime.

However, this area of research did not stop at Cesare Lombroso, and much of this research has already been widely accepted into other fields. As such, the same old arguments against incorporating biological science into the field today may simply be misguided and potentially flawed due to misconceptions of the role played by biology, and much of the fear mongering may be misguided as fundamentally misunderstanding the strength of our legal institutions in this country to protect individuals from discrimination and unequal treatment. There will not be another eugenics movement. The time for that has past. So, isn't it about time for the field of criminology to get over this and start incorporating biological science into criminological theory and in our curriculum? As such, this article looks at three specific areas of biological research, regarding genetic influences, brain and neurochemical influences, and neonatal influences, to suggest a few ways that biology can be incorporated into our understanding of crime.

Keywords: The Criminal Man; Mesomorphic physique; The field of criminology.

Abbreviations: SSSM: Stand Social Science Methodologies; PET: Positron Emission Topography; MRI: Magnetic Resonance Imaging

Introduction

Sometimes advances in science are made in different fields, but the results of those studies wind up having a large influence in other areas. And sometimes theories that were long thought abandoned find a way to reemerge

due to these advances—even if slowly and (within some corners of the field) with a lot of protest. I am of course mentioning the resurgence of biological theories of crime and the recent development of biosocial criminology as a new area of research worthy of pursuit within the field of criminology. As such, I will attempt to (within the best of my ability) discuss the implications of three separate results from biosocial research and the potential implications it may have on theory development within criminology. I hesitate by saying “within the best of my ability” as many of the methodologies used to study

biological and genetic influences of crime are quite different from the methods taught in most criminology graduate programs. Most criminological theory courses will typically discuss at some point the biological influences of crime.

Often these discussions will refer to Cesare Lombroso, who wrote the book “The Criminal Man,” where he described that criminals all appear to have a similar physical morphology (i.e. a mesomorphic physique) and they would display specific “atavisms” to indicate their propensity to crime. These atavisms, which can be used as indicators of criminality, included several things from the length of their arms, the shape of their skull, or the shape of their ear lobes. This research was later debunked, but at its time it helped to spur on debate for the eugenics movement—a political movement that took off in the early 1900s that justified the euthanizing or sterilization of the developmentally delayed, criminal, and mentally ill.

Much of this history is often brought up by individuals within criminology to argue against the adoption of biological perspectives of crime. Much of these arguments may be misguided and potentially flawed due to misconceptions of the role played by biology, and much of the fear mongering may be misguided as fundamentally misunderstanding the strength of our legal institutions in this country to protect individuals from discrimination and unequal treatment (i.e. 14th Amendment). Regardless of this history of backlash mentioned quite frequently in biosocial texts, such as Kevin Beaver’s text “Biosocial Criminology” where he goes on at length about this debate, advances made in other fields by biological and genetic researchers continued unabated. As such, I will discuss three results that have had a profound impact upon the study of crime: (1) genetic influences, (2) brain and neurochemical influences, and (3) neonatal influences.

Genetic Influences on Crime

In a massive meta-analysis of twin studies over the last fifty years, Polderman et al. (2015), researchers from outside the field of criminology, discussed the potential role of genetics in particular traits. In general what they found was that the role of genetics in regards to predisposing individuals towards specific traits is complicated. While some traits, such as height (with an estimated genetic role of 0.908) and skeletal structure (with an estimated genetic role of 0.830) are highly correlated, other traits such as social values (0.489) and temperament (0.470) are much less correlated.

This is because traits such as temperament and social values are highly complex traits that are a product of both nature and nurture. Instead, the researchers found that traits that follow the often predicted effect of genetics in which dizygotic twins (i.e. fraternal twins) share half the same genes and may thus share half of the same traits and where monozygotic twins (i.e. identical twins) share all of the same genes and thus should share all of the same traits ($2_{dz} = 1_{mz}$) seem to cluster into specific types. For example for some traits, such as those affecting the neurological, ear, nose and throat, cardiovascular, and ophthalmological domains, these traits closely match the predicted $2_{dz} = 1_{mz}$ model. However, for the more complex traits such as conduct disorders, higher-level cognitive functions, hyper-activity, and anxiety, for example, both genetic and non-genetic (shared and non-shared environmental) factors are necessary to explain them. According to Kevin Beaver [1], “Discussions of heritability often produce confusion over what heritability estimates mean and what heritability estimates can reveal. ... Heritability estimates cannot be extrapolated to the individual—that is, heritability can only be applied to group-level variance.”

What this means is that just because 50 percent of the variance in a particular phenotype is due to heritability does not necessarily mean that we can say that 50 percent of an individual person’s phenotype is due to genetic factors. In other words, a lot of things go into explaining why certain individuals have certain traits. According to Beaver [1], genetics much like many other biological processes are malleable—meaning they change. Often times these changes can be due to an individual’s environment or different exposures which may turn on and off different parts of a person’s genetic code. While some complex traits then can be affected by genetics to a certain proportion at a certain scale (usually at an aggregate level) individuals may vary widely on those shared traits. According to Beaver and others, the proportion of variance that is not due to genetics is of course due to the environment, which is separated into two types: (1) the shared environment, which refers to the things that the siblings share in common (same household, similar upbringing, same schools, etc.), and (2) the non-shared environment, which refers to the things that the siblings do not share in common, such as friends, for example.

The genetic factor in heritability is often denoted as h^2 , while the environmental factors are denoted as c^2 (shared environment) and e^2 (non-shared). According to Beaver, the proportion due to each of these different factors can be derived by knowing the specific proportions of the shared variance of the traits from monozygotic and

dizygotic twin pairs, such that $h^2 = 2(rMZ - rDZ)$, where rMZ represents the “cross-twin correlation” due to monozygotic twins and rDZ represents the cross twin correlation due to Dizygotic twins. Further, $c^2 = 2rDZ - rMZ$ and $e^2 = 1 - (h^2 + c^2)$. Further, according to findings from a twin study conducted by Terrie Moffitt for antisocial traits, the percentage of variance due to heritability is roughly 50%, shared environmental factors is 20%, and non-shared environmental factors is 30%. All of this appears to make some intuitive sense. However, it may certainly have some unappealing implications, such as the small percent of variance due to shared environmental factors. This would seem to suggest that there was little that parents can do to modify their parenting practices, for example, to influence the potential antisocial behaviors in their children. This, of course, tees biosocial criminologists up for a lot of criticism [2].

For example, according to Walters in 38 twin based studies up to that point many of the methodologies used in the studies were flawed in some way, such as family studies. As such, they believed that the actual heritability due to genetics lies somewhere between zero and the higher estimate predicted by the family based studies. However, overall, due to some of the studies with larger sample sizes, they still found that genetics contributes to some extent to influence particular traits (although weak) [3].

Further, according to Walters and White [4] “[T]he large number of methodological flaws and limitations (such as sample size, sampling bias, generalizability, etc.) in the research should make one cautious in drawing any causal inferences at this point in time.” However, they were also referring to the studies conducted prior to [5] and the methodologies being used have only advanced since then. Another common attack, according to Beaver [1], is the equal –environment assumption (EEA) and the look alike theory. According to the EEA attack, many critics contend that biosocial criminologists assume that both monozygotic and dizygotic twin pairs share similar environments; however, they contend that the environments shared by monozygotic twins are more similar than the environments shared by dizygotic twins, partially because they look more alike, are dressed the same, or are raised in a more similar fashion.

If true then the EEA assumption would mean that the estimate for heritability is over inflated. However, according to Arthur Jensen, responding to the look alike theory purported by critics, in explaining the role of genetics on intelligence stated, “If those who really believe that the IQ correlation between MZ twins is better

explained in terms of their physical similarity than in terms of their genetic correlation, they should go out and find unrelated people who look alike, such as movie stars and their doubles, and determine the correlation between their IQs,” [6]. This would seem ironic that many critics of biosocial influences are making similar arguments as Cesare Lombroso to discredit biosocial results.

Regardless, there is still an effect due to the potential critical standing that biosocial criminology has in the field. For example, in an article by Barnes et al. [7], “On the consequences of ignoring genetic influences in criminological research,” they found that standard social science methodologies (SSSM) that do not take into account potential biological influences of crime are producing biased estimates. This was especially problematic when the specific heritability for antisocial traits was stronger or equal to the influence of the variables being studied (sans-genetic factors). The problem was so bad that the researchers stated, “As the correlation between genetic factors and the criminological variable increased, the degree to which the effect of the criminological variable on antisocial behavior was confounded increased as well. In some of the most extreme examples, 100% of the association between the criminological variable and antisocial behavior was explained away due to genetic confounding,” [7].

If so, then the implications for genetic influences in criminology may have profound effects for many different criminological theories—potentially making many of them obsolete and due to be abandoned, much like Cesare Lombroso’s theory was many years before.

Brain and Neuro Chemical Influences on Crime

Another finding from biosocial criminology and in particular researchers such as Adrian Raine are the implications due to findings regarding brain and neuro chemical functioning. Most of the studies regarding brain formation and crime include methods using positron emission topography (PET) scanning and magnetic resonance imaging (MRI) scans. These studies are quasi-experimental in nature (meaning they should be given wider credit than say many cross sectional correlational studies popular in criminology) in that they generally involve two groups being examined: a criminal group composed of criminally disposed individuals recruited for the study and a control group of non-criminally disposed individuals. The scientists would typically then compare PET and MRI scans to the other members in the group to find similarities and then to the other group to see if they differ. According to Rowe [8] the way PET scans work is

that the individual will be injected with some form of sugar with radioactive isotopes that can be picked up on the scanner. (This radioactivity is generally harmless in small quantities).

As the brain consumes the sugar due to the isotopes the areas that exhibit higher uptake levels would brighten more on the PET scanner than the areas of the brain that don't absorb as much. This gives the scientists an indication of what parts of the brain for that particular individual are more active than others. In a 1993 study, Adrian Raine performed PET scans on 41 individuals (22 murderers and 19 non-criminal control subjects matched on sex and age). Raine found that the murderers were shown to have much lower sugar metabolism in their pre-frontal cortexes than the controls, with one glaring exception. The exception was a serial murderer who had killed roughly 45 people.

Raine hypothesized that this individual needed higher prefrontal cortical ability to be able to plan out many of his murders. However, in general, individuals with lower pre-frontal cortical activity are said to be impulsive and find it more difficult to plan and control their emotions. Raine, Buchsbaum, and LaCesse [9] replicated this study with twice the participants and found similar results regarding lower pre-frontal cortical activity. They also found higher activity in the lower portions of the brain primarily responsible for aggression, such as the amygdala.

Much of these results would seem to harken back to the famous case of Phineas Gage. The way the story is usually told is that in 1848 Phineas Gage was working as a construction foreman for the Rutland & Burlington Railroad in Vermont. The crew's job was to use explosives to blast holes in the side of rock to help clear a path for where the railroad would be laying down tracks. However, as sometimes happens with explosives, there was a premature blast that caught many of the individuals off guard. This blast ended up lodging a steel rod through Gage's head. Based on medical evidence, we know that the rod was sent through the side of his face and then through his prefrontal cortex causing massive damage to that portion of his brain. Gage, however, was not killed, and he was able to stand up to ride to the hospital to eventually have the rod removed. According to historical accounts, after the incident Gage's personality changed.

Where he had been a hardworking and conscientious person before (having been promoted to foreman), after his incident he was described as moody, irritable, and quick to anger. He was also described as being "unpredictable and prone to impulsive fits of violence,

which ultimately led to his being let go by the Railroad Company" [10]. Similar results due to traumatic brain injuries have also been found with soldiers who sustained injuries due to explosive blasts [11] Grafman et al., [12,13]. Further, children less than four years old may be more susceptible to brain injuries and possible neglect [14]. This finding may be incredibly important in regards to the section I will discuss next on neonatal influences.

Studies conducted by experimental psychologists have also found interesting links between certain neurochemicals and aggression [15]. The neurotransmitters serotonin and dopamine have been found to be tied to the behavioral trait of aggression, more specifically impulsive aggression. More specifically, lower levels of serotonin has been found to be associated with impulsive aggression, and they found an interesting interaction with another neurotransmitter, dopamine, in which an impairment in serotonin can lead to a dysregulation of dopamine. Dopamine is the neurotransmitter that is involved in the modulation of aggression. In animal studies, hyperactive dopamine levels have been associated with increases in impulsive aggression [16]. Serotonin, on the other hand, is the neurotransmitter more closely associated with planning and self-regulation, and a reduction of which can result in an inability to hold back aggressive tendencies when placed in coercive situations [17]. According to Moffitt [18], certain neuropsychological deficits such as serotonin in combination with family risk factors has been found to be associated with the most persistent, serious, violent offenders.

Neonatal Influences on Crime

The final result I want to discuss that may have implications for theory is the potential effect of neonatal influences due to exposure to toxins, such as drugs, alcohol, tobacco, and lead while pregnant. For a quick anecdotal aside, back when I was working as a child protective investigator I remember one of the first cases where I had to remove a child from a home. The family had been struggling with an addiction to methamphetamine, and after being caught by their sponsor with methamphetamine he reluctantly called in an anonymous report (hint nothing is ever actually anonymous). When I went to the house, the family was informed that they were caught with drugs. They had had several reports against them in the past, but this time the State had had enough and I was told to help remove their 1 and half year old son from their care. What is most memorable to me was the physical and temperamental reactions of the child. I specifically remember that while the child cried at some point half of his face would droop

and he would seem to glitch (like a robot in a weird sci-fi cartoon).

This would also result in a strange scream that would change octaves half way through and then go back to normal, like a Doppler effect. One of his eyes was also slightly crossed eyed. I just remember thinking, "That's not right." I could only imagine what things this child was exposed to. After the child was taken into foster care, the other children avoided him, because he had a tendency to run up to them and bite them. Several other investigations I had involving removals concerned children born in hospitals suffering from neonatal abstinence syndrome or withdrawals. Essentially, the children were born addicted to methamphetamine or heroine that they had been receiving through their mother's blood stream while in-utero.

After being born they would go into massive withdrawals and they often shook due to massive seizures. According to Dodge and Pettit [19], "Fetuses exposed to opiates or methadone are at a heightened risk for conduct problems 10 to 13 years later, as are fetuses exposed to alcohol, marijuana, and cigarette by-products during pregnancy." According to Kevin Beaver [1], "Prenatal exposure to toxins represents one of the most salient environmental factors that can interfere with normal brain development." Some of the results due to exposure to various chemicals, such as cigarettes, has been found linked to numerous maladaptive outcomes, such as reduced cognitive abilities (Sexton, Fox, and Hebel, 1997), hyperactivity [20], and early antisocial behavior [21].

According to Adrian Raine [22], "The effects of fetal exposure to alcohol in increasing risk for conduct disorders is well known, but recently a spate of studies has established beyond reasonable doubt a significant link between smoking during pregnancy and later conduct disorder and violent offending." According to Mayes [23], prenatal exposure to cocaine has been linked to poor emotional and attention regulation in infants and preschool-aged children. Further, another potential risk of exposure is due to lead. According to the Federal Interagency Forum on Child and Family Statistics [2], children exposed to higher levels of lead have been shown to suffer from cognitive and developmental impairments and troubling behavior affects. This adds increased interest as to what will happen to the millions of children exposed to incredibly high levels of lead found in the drinking water of Flint Michigan [13]. While the evidence due to neonatal exposure to toxins and later association to criminal activity is inconclusive [10], it is worth considering the implications.

Many of the results I have discussed can easily tie back into other criminological theories, besides biosocial criminology. For example, many of the factors discussed can easily be applied as just another cumulative disadvantage that can be tied into Sampson and Laub's Life Course Developmental Theory [24]. Even if the effects due to genetics, brain formation, neurotransmitter levels, and toxic exposure are small, it is important to remember the effect they may have over the course of an individual's life.

Back when I studied mutual and hedge fund regulations, one of my law school professors discussed the seemingly small fees often paid to financial managers for facilitating trades. Usually the fees were incredibly small, representing 0.3% of the cost of the trade, for example. However, while this amount seemed small, when those fees were added up over the course of many trades and when that amount was multiplied due to the effects of compound interest, the amount lost over the course of an individual's life or up until their retirement was huge. These biological factors may have a similar effect—while initially they seem small, compounded over the course of an individual's life, they may lead that person to take vastly different life paths.

References

1. Beaver K (2009) Bio social criminology: A primer. Dubuque: Kendall hunt publishing company.
2. Moffitt T (2005) Genetic and environmental influences on antisocial behaviors: evidence from behavioral-genetic research. *Advances in genetics* 55: 41-104.
3. Walters G (1992) A meta-analysis of the gene-crime relationship. *Criminology* 30(4): 595-614.
4. Walters G, White T (1989) Heredity and crime: Bad genes or bad research? *Criminology* 27(3) 455-485.
5. Sexton M, Fox NL, Hebel JR (1990) Prenatal exposure to tobacco: II Effects on cognitive functioning at age three. *International Journal of Epidemiology* 19(1): 72-77.
6. Miele F (2002) Intelligence race and genetics: Conversations with Arthur R, Jensen Boulder CO: Westview.
7. Barnes JC, Boutwell BB, Beaver KM, Gibson CL, Wright JP (2014) On the consequences of ignoring genetic influences in criminological research. *Journal of Criminal Justice* 42(6): 471-482.

8. Rowe D (2002) *Biology and crime*. Los Angeles CA: Roxbury P:(174).
9. Raine A, Buchsbaum M, LaCasse L (1997) Brain abnormalities in murderers indicated by positron emission tomography. *Biological psychiatry* 42(6): 495-508.
10. Bartol A, Bartol C (2011) *Criminal behavior: A psychological approach*. 9th Ed. Boston: Pearson.
11. Barrash J, Tranel D, Anderson SW (2000) Acquired personality disturbances associated with bilateral damage to the ventromedial prefrontal region. *Developmental neuropsychology* 18(3): 355-381.
12. Grafman J, Schwab K, Warden D, Pridgen A, Brown HR, et al. (1996) Frontal lobe injuries violence and aggression: a report of the Vietnam Head Injury Study. *Neurology* 46(5): 1231-1238.
13. Hanna-Attisha M, LaChance J, Sadler R, Champney Schnepf A (2016) Elevated blood lead levels in children associated with the Flint drinking water crisis: a spatial analysis of risk and public health response. *American journal of public health* 106(2): 283-290.
14. Thompson R, Nelson C (2001) Developmental science and the media: Early brain development. *American Psychologist* 56(1) 5-15.
15. Seo D, Patrick CJ, Kennealy PJ (2008) Role of serotonin and dopamine system interactions in the neurobiology of impulsive aggression and its comorbidity with other clinical disorders. *Aggression and violent behavior* 13(5); 383-395.
16. Harrison A, Everitt BJ, Robbins TW (1997) Central 5-HT depletion enhances impulsive responding without affecting the accuracy of attentional performance: interactions with dopaminergic mechanisms. *Psychopharmacology* 133(4): 329-342.
17. Siever LJ (2008) Neurobiology of aggression and violence. *American Journal of Psychiatry* 165(4) 429-442.
18. Moffitt T (1993) *Adolescence-Limited and Life-Course-Persistent Antisocial Behavior: A Developmental Taxonomy*. *Psychological Review* 100(4) 674-701.
19. Dodge K Pettit G (2003) A biopsychosocial model of the development of chronic conduct problems in adolescence. *Developmental psychology* 39(2): 349-371.
20. Huijbregts SC, Séguin JR, Zoccolillo M, Boivin M, Tremblay RE (2007) Associations of maternal prenatal smoking with early childhood physical aggression hyperactivity-impulsivity and their co-occurrence. *Journal of abnormal child psychology* 35(2): 203-215.
21. Gatzke-Kopp LM, Beauchaine TP (2007) Direct and passive prenatal nicotine exposure and the development of externalizing psychopathology. *Child psychiatry and human development* 38(4): 255-269.
22. Raine A (2002) Annotation: The role of prefrontal deficits low autonomic arousal and early health factors in the development of antisocial and aggressive behavior in children. *Journal of Child Psychology and Psychiatry* 43(4): 417-434.
23. Mayes LC (1999) Developing brain and in utero cocaine exposure: effects on neural ontogeny. *Development and psychopathology* 11(4): 685-714.
24. Sampson RJ Laub JH (1997) A life-course theory of cumulative disadvantage and the stability of delinquency. *Developmental theories of crime and delinquency* 7: 133-161.
25. Federal Interagency Forum on Child and Family Statistics. (2005). *America's Children: Key National Indicators of Well-being 2005*. Washington DC: US Government Printing Office.
26. Lombroso C (1876) *The Criminal Man*. 1st Ed. Milan: Hoepli.
27. Polderman T J, Benyamin B, De Leeuw, CA Sullivan PF, Van Bochoven A (2015) Meta-analysis of the heritability of human traits based on fifty years of twin studies. *Nature genetics* 47: 702-709.
28. Raine A (1993) *Psychopathology of crime*. San Diego: Academic Press.