CRIMINOLOGY

ABANDON TWIN RESEARCH? EMBRACE EPIGENETIC RESEARCH? PREMATURE ADVICE FOR CRIMINOLOGISTS*

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In their original article, Burt and Simons (2014) argued that heritability studies should be abandoned because twin and adoption research is a fatally flawed paradigm. They pointed optimistically to epigenetics research as the way forward. In our view, both recommendations are hasty. This commentary will put forward two contrarian opinions. First, twin and adoption studies still have a lot to offer criminologists who seek the social causes of crime. Second, epigenetics research has very little to offer yet for criminologists who seek the social causes of crime.

WHAT CAN TWIN AND ADOPTION RESEARCH OFFER CRIMINOLOGISTS?

Twin and adoption studies are informative about environmental crime causation. In 2005, Terrie E. Moffitt reviewed the contribution of twin and adoption research to the study of antisocial behavior in a paper titled, "The New Look of Behavioral Genetics," which subsequently became the basis of a lecture for the Stockholm Criminology Prize (Moffitt, 2005a; see also Moffitt, 2005b). The message was that the most remarkable contribution of behavioral genetic studies to science is the robust and compelling evidence base documenting *environmental* causes of behavior, especially *social causes of antisocial and criminal behaviors*. Quantitative twin studies have documented that environmental causes generate at least half of population variation in antisocial behaviors and crime. Twin and adoption studies also have shown that the influence of numerous specific risk factors on crime is environmentally mediated. These unprecedented contributions have gone virtually unnoticed by many criminologists.

The 2005 review included a 20-page description of publications that have applied twin and adoption designs to make rigorous (and successful) tests of the environmental causation of antisocial and criminal outcomes (Moffitt, 2005a). The end of that article (p. 548) made this prediction: "Twin and adoption designs are likely to prove very useful

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for a long time." In the aftermath of the announcement that the human genome had been solved, many pundits speculated that the need for quantitative behavioral-genetic twin and adoption studies has ended. To the contrary, there are many essential roles for quantitative analyses in genetically sensitive designs, even as researchers work more with measured genes. Designs that can control for genetic influence will be essential for showing whether a putative environmental variable really affects behavior environmentally. Dizygotic twins are ideal for testing what factors explain behavioral differences between siblings who are matched for age, sex, ethnic background, and most early rearing experiences. Discordant monozygotic twins are ideal for studying environmentally induced variation in the behavior of siblings matched even further, for genotype (Moffitt, 2005a). The prediction remains true today.

It may seem counterintuitive to use behavioral-genetic designs to test environmental influences on crime, but this is one of their strongest applications. Quantitative behavioral genetics studies can help rule out the most pernicious challenge to environmental causation theories: selection effects. Selection occurs because individuals rarely are randomly assigned to criminogenic social environments. Some third variable, potentially a heritable one, can generate propensity for crime and at the same time can lead individuals to create, seek, or otherwise end up in criminogenic environments. Antisocial behavior "creates" social reactions from others when aggressive toddlers evoke harsh discipline, when bullies evoke rejection by peers, when young shoplifters evoke processing by the justice system, or when abusive husbands evoke divorce by their wives. Individuals "seek" environmental settings consonant with their propensity to offend when aggressive children gravitate toward a delinquent peer group, when offenders pair off with girlfriends and wives who tolerate or encourage antisocial behavior, or when a pedophile seeks volunteer work with children. People who have behaved antisocially "end up in" criminogenic environments selectively when misbehaving children are tracked into special classes for disturbed pupils, when troubled teenagers are recruited by gangs, when violent young men are imprisoned with hardened offenders, or when parolees find nothing but unskilled jobs available to them. Thus, selection effects account for much of the observed correlations between crime and many of its putative environmental risk factors.

Behavioral genetic studies are one of criminology's few options for disentangling selection effects from social causation effects. Ordinary observational studies cannot test whether a particular risk factor is truly causal because they cannot disentangle social causation from selection effects. Experimental random assignment can rule out selection effects, but it is unethical to assign research participants randomly to experimental conditions expected to induce aggression or criminal offending. Criminologists have a limited arsenal of methods for testing social causation: 1) natural-experiment studies of within-individual change, 2) randomized treatment experiments, and 3) behavioral-genetic designs (Moffitt, 2005b). None of these three designs alone provide decisive proof of social causation, but if all of them supply corroborative empirical evidence by ruling out selection effects on a risk factor, then a strong theoretical case for social causation can be made. As a result of this paucity of methods for testing causation, if criminology abandoned twin and adoption research, then our field would lose one of the three chief methodological weapons in our arsenal for testing social causation. Criminologists' capacity to study the social and environmental causes of crime would be, sadly, reduced.

Burt and Simons (2014) urged abandoning adoption studies because they think adoption studies are fatally flawed. However, an adoption study of crime was the first in the

behavioral sciences (to my knowledge) to rule out potential genetic confounding while supporting a hypothesis of social causation. Van Dusen et al. (1983) used data from the Danish Adoption Study to disentangle the socioeconomic circumstances of adoptees' conception (their biological father's occupational status) from the socioeconomic status in which they were reared (adoptive father's status). The results showed that biological inheritance could not explain the majority of the class–crime connection, supporting the inference that the social class in which people grow up has a direct environmental effect on their probability of criminal offending (Van Dusen et al., 1983).

Burt and Simons (2014) also suggested that the twin design is irreparably flawed. However, one of the most exciting future uses of twin data for criminology is the study of discordant twins (Boomsma, Busjahn, and Peltonen, 2002; Kendler, 2001). Because twins in a monozygotic pair are never perfectly identical in their offending behavior despite sharing all their genes, this gives criminologists a special opportunity to study what experiences reduce co-twins' behavioral similarity. In one example by Caspi et al. (2004), the Environmental Risk Longitudinal Twin Study (E-Risk) used 600 monozygotic twin pairs to test the hypothesis that hostile and cold parental treatment causes children to become more aggressive. Caspi et al. exploited discordant parental treatment of co-twins and tied this to the co-twins' discordant behavioral outcomes. They found that within pairs, the twin who received relatively more maternal negativity and less warmth developed the most aggressive behaviors (Caspi et al., 2004). To capture subtle but important differences in environment between co-twins, audiotapes were made of the mother describing each of her twins. Negativity and warmth were measured by coding tone of voice and speech content, according to the established "expressed emotion" paradigm. The children's behavior was measured using teacher reports to rule out the possibility that a mother's negativity toward a child could bias her reports about the child's aggression. The 5-year-olds whose mothers were most negative toward them showed increasing levels of aggression when followed up as 7-year-olds. Studying monozygotic twin pairs ruled out the possibility that any genetically transmitted liability had caused both the mother's hostile parenting and her child's aggression and ruled out the possibility that a genetically influenced child effect provoked mothers to hostility.

Twin discordance can be studied for most of the leading causal variables in criminology. Criminogenic experiences unique to the individual and not shared with his or her sibling include being the target of child sexual abuse, falling in with a gang of delinquent friends, dropping out of high school, sustaining a head injury, developing an addiction, or suffering long-term unemployment. To date, discordant twin designs in developmental criminology have been limited mainly to studying childhood aggression. However, enough twin samples have reached adulthood that it is now possible to apply the discordant twin design to testing causes of adult crime and causes of desistence from crime. Imagine testing whether prison causes crime by comparing twin siblings who go to prison against their co-twins who do not. Research attempting to test the iatrogenic influence of incarceration on postrelease criminal recidivism is bedeviled by the possibility that longer prison sentences might selectively be given to individuals carrying greater criminal propensity (Nagin and Snodgrass, 2013). Discordant twin designs are a natural solution. Imagine testing theories of criminal desistence using desistence-promoting experiences unique to the individual and not shared with his or her sibling, such as moving to a new location, getting a good job, bonding with a prosocial spouse, having a first child, or becoming born again. In my view, criminologists need behavioral genetic designs and should exploit them more in the future.

WHY SHOULD CRIMINOLOGISTS BE SKEPTICAL ABOUT EPIGENETIC RESEARCH?

Many social scientists embrace the new epigenetics research because it has been billed as evidence that environment trumps genes. There is much excitement about this approach, which promises to capture a biological signature left behind by environmental adversity. However, our reading, and that of many biologically oriented scientists, is that epigenetics has been wildly oversold, particularly in the media. Many of our expert epigenetics research colleagues are deeply embarrassed by the warm, uncritical response their work has attracted from the social sciences. A biologist attendee at a July 2014 Washington, DC workshop on the social and behavioral implications of epigenetics gasped, "The biologists there were horrified at the thought … we really don't understand the basic biology well enough yet to do this!" A social scientist attendee agreed, "After the meeting I got the feeling the popular media has sold us a false bill of goods." Here, we briefly summarize several cautions but refer interested readers to more in-depth discussions and sobering views by real experts in epigenetics (Heijmans and Mill, 2012; Juengst et al., 2014; Mill and Heijmans, 2013).

First, although the term "epigenetics" can refer to virtually anything "above the DNA sequence," the measure that can be quantified with available technology is methylation. Methylation is often described as a chemical change induced by environmental experience that turns genes off, leading nonexpert readers to infer that methylation is only or primarily brought about by experience. In fact, methylation is ubiquitous and normative, and usually it has nothing to do with experience but is part of organism development that is, incidentally, under genetic control. Because the genome is identical in each of our cells, during normal development, most of our genes must be methylated, lest a kidney cell grow into an eye or a fingernail cell grow into an ear. Against this normative background, methylation marks that can be statistically associated with external experience are relatively rare, and effect sizes are expected to be small.

Second, methylation acts locally within particular tissues. Most tests in humans sample cells from saliva or blood, but making inferences about behavior tends to require brain cells, which are more difficult to come by (particularly from criminal offenders). Placenta, cord blood, and saliva have unique epigenetic signatures within the same child (Armstrong et al., 2104). There are even different methylation patterns among different kinds of cells within a blood sample.

Third, currently there is not enough information to point to where in the genome to look for putative experience-based methylation. It should be contingent on genomic location, but as we do not know the location of the genes associated with antisocial propensity, any search for methylation of them is not guided by hypotheses with strong prior probabilities. This leaves chance findings highly probable and demands extensive replication checks.

Fourth, laboratory assays for detecting epigenetic changes and statistical approaches to working with whole-genome epigenetic data sets are still in flux, which is making it difficult to accumulate comparable studies to gain a sense of what is known.

Fifth, although a small set of nonhuman studies provide initial evidence that experience can apparently alter methylation, it is far from clear that the detected methylation alterations have any consequences for health or behavior. Before methylation can affect health or behavior, it must alter expression of genes. Links from methylation data

forward to gene expression data are not yet known. Moreover, gene expression is merely the initial step. A cascade of subsequent contingent changes at the cellular and organ levels (all of which are probabilistic in nature and not yet understood) must occur en route to the level of observable health or behavior. Research testing for connections between methylation and gene expression is in its infancy, even in rodents. For criminology, research will need to show that stress and adversity predict methylation and methylation status predicts crime, and that methylation statistically mediates the pathway from stress and adversity to crime. This is a tall order.

Sixth, so far the most robust effects entail experimental nutritional or chemical exposures, whereas in contrast the literature on effects of parental care or social stress is less developed and still subject to replication confirmation.

Seventh, there is room for concern that public hype over epigenetics findings is already leading to a new vicious cycle of deterministic thinking, perhaps more dangerous than genetic determinism, because it points a finger of blame at parents who fail to prevent their children's genomes from becoming methylated (Juengst et al., 2014).

Of particular interest to criminologists reading the debate stimulated by Burt and Simons (2014), experts in epigenetic research point to the longitudinal twin design as the recommended approach to studying experiential effects on methylation in humans (Bell and Spector, 2012; Mill and Heijmans, 2013). Why twins? In addition to the aforementioned cautions, basic information about methylation is not known, such as sex or age differences, seasonal effects, circadian effects, or even whether the time-scale of change is typically minutes, days, weeks, or years. There are probably stages of development in the life course when normative methylation changes accelerate or slow, but these are as yet unknown. Thus, data are noisy in unknown ways. Because the epigenome is dynamic, not static like the DNA sequence, it is subject to all the confounds that plague any other outcome, including selection effects. For example, puberty undoubtedly entails epigenetic change, which could coincidentally (and artefactually) appear to be a correlate of delinquency initiation. Among the few established environmental sources of methylation change is tobacco exposure. Given the strong association of smoking and substance use with crime, establishing whether methylation findings in relation to criminal behavior reflect causal processes or spurious correlates will be tricky. To date, the most compelling reports of methylation effects have examined rodents (a key fact conveniently omitted in many popular media reports). In rodent models, all research participants are genetically identical, and everything in their environment is held constant across treatment and control groups except the experimental manipulation. This uniformity combined with experimental control over the severity of the treatment dose makes it relatively easy to detect effects in laboratory animals, compared with humans, who are characterized by staggering levels of both genetic and environmental diversity. As noted, ordinary observational studies cannot rule out selection effects, but the use of twins, especially longitudinal analyses in discordant monozygotic twins, can help to pin down and hold constant some of the complexity that will otherwise compromise translation of epigenetics research from rodent models to humans.

The current recommendation from the experts is, if you plan to study human epigenetics, then at least use twins. This judgment casts an interesting light on the recommendation of Burt and Simons (2014) that criminologists should embrace epigenetics and abandon twin studies. In any case, criminologists would be wise to maintain our usual critical thinking and skeptical scientific objectivity.

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