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Genetic Contributions to Antisocial Personality and Behavior: A Meta-Analytic Review From an Evolutionary Perspective

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ABSTRACT. Evidence from behavioral genetics supports the conclusion that a significant amount of the variance in antisocial personality and behavior (APB) is due to genetic contributions. Many scientific fields such as psychology, medicine, and criminal justice struggle to incorporate this information with preexisting paradigms that focused exclusively on external or learned etiology of antisocial behavior. The current paper presents a meta-analytic review of behavioral genetic etiological studies of APB. Results indicated that 56% of the variance in APB can be explained through genetic influences, with 11% due to shared non-genetic influences, and 31% due to unique non-genetic influences. This data is discussed in relation to evolutionary psychological theory.

Keywords: antisocial behavior, evolution, violence

THE DEGREE WITH WHICH ANTISOCIAL, aggressive, and violent behavior can be explained through evolved genetic factors continues to be debated in the social sciences. This paper concerns itself with identifying the degree of variance in antisocial behavior that can be explained through genetic and non-genetic factors in order to better inform this debate. It is my argument that the existing "Standard Social Science Model" (SSSM; Tooby & Cosmides, 1992) has, despite vaguely acknowledging the importance of "nature" in human behavior, persisted in focusing primarily on social or environmental explanations of behavior at the expense of genetic explanations of antisocial behavior. This may be, in part, a practical concern wherein genetics is difficult for most social scientists to control. Yet this practical concern appears to filter down into theory and intervention priorities. Ultimately, social science may be experiencing a problem related to "levels of analysis" (Tinbergen, 1963). Specifically, the SSSM, due to practical

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limitations, may have become accustomed to viewing behavior through "proximal" or immediate causes or even mere correlates. For example, relationships between being abused as a child and acting violently as adults, though not always large, are commonly found in the literature (e.g. Ferguson et al., 2008). However, it remains unclear if this relationship between family violence and violent crime is due to learning/socialization factors or genetic factors or some combination of the two. Specific genes or socialization influences that contribute to behavior may be considered proximal or immediate causes of behavior. The ultimate cause of behavior, however, may more likely be the evolutionary process, which caused those specific genes to be selected due to specific environmental pressures, or which caused the ability to learn aggressive behavior to become an adaptive trait. It is the position of this article that understanding evolution as an ultimate cause of antisocial behavior is an important part of the discussion on antisocial behavior and violence that, as of yet, has not received adequate attention. Thus, this paper will examine the evidence for genetic contributions to antisocial behavior and explain these contributions from an evolutionary framework.

The Genetics of APB

In the current article, I refer to antisocial personality and behavior (APB) as the main variable of interest. This variable is defined as including both the innate traits and motivation that direct individuals toward antisocial behavior (i.e., antisocial personality disorder, psychopathy) and antisocial behavior itself (i.e., aggression, violence, lying, stealing, etc.). Use of the term *APB* is intended to reflect the range of dependent measures used in relevant studies, some of which examine personality factors (i.e., Psychopathy Checklist), and others of which examine behaviors (i.e., arrest records, self-reported criminal activity, etc.) Evidence for a genetic (or partially genetic) etiology for APB comes from two main sources. Specifically, these are studies investigating the relationship of specific genes and APB, and behavioral genetics research, which attempts to determine the variance in APB that is due to genetic and non-genetic influences.

Regarding specific genes, understanding of the human genome and its influences on human behavior remains preliminary. However research on specific genes in human populations has begun to provide some evidence for how genes may contribute in the development of APB. For example, in one study of 240 children with attention deficit hyperactivity disorder, children with the valine/methionine variant in the catechol O-methyltransferase (COMT) gene showed greater antisocial behaviors than those without this variant (Thapar et al., 2005). This variant of the COMT gene also appeared to interact with neonatal risk to increase APB. The COMT gene variant may have influenced the development of the prefrontal cortex, potentially reducing control over aggressive impulses.

Caspi et al., (2002) used a longitudinal design to examine the impact of the MAOA gene located on the X-chromosome and its interaction with exposure to

maltreatment in the family. Results indicated that males with both a low-MAOA activity genotype and exposure to maltreatment were significantly more likely to exhibit child conduct disorder and adult antisocial behavior, including criminal arrests, than were high-MAOA activity genotype males who had been similarly maltreated. Although the low-MAOA genotype on its own did not increase APB, it appears that its presence places individuals at risk for APB, which can be triggered by maltreatment in the family. The presence of this gene on the X-chromosome may also help explain why males are more aggressive, on average, than females, particularly if the low MAOA activity genotype is recessive in nature. This finding related to the low-MAOA genotype has been replicated in other studies (e.g., Nilsson et al., 2007; Kim-Cohen et al., 2006).

Retz, Retz-Junginger, Supprian, Thome, and Rosler (2004) found a relationship between the serotonin transporter promoter gene (5-HTT) and impulsive violence in a forensic sample of 153 males. Specifically, a deletion/insertion polymorphism on this gene predicted impulsively violent behavior within this population. In and of itself, polymorphism on this gene was not able to explain the majority of violent behavior, but it appears that this gene may be one part of a larger genetic puzzle.

It is important to point out that these single genes do not appear, in and of themselves, to deterministically cause APB in the same sense that the HD gene invariably leads to Huntington's disease. Rather, these genes likely interact with each other in ways that remain poorly understood. Further, there are likely numerous other genes that are involved either directly or indirectly (i.e. via interactions) that have yet to be identified. Finally, these studies demonstrate that genetic vulnerability and exposure to family violence interact to lead to APB.

These studies are unable to inform us regarding the relative contribution of genetic and non-genetic influences in explaining proportions of variance in the etiology of APB. For this purpose, behavioral genetic procedures such as twin and adoption studies have been employed as a means of gauging or estimating the relative contribution of genetic and non-genetic influences in APB etiology.

Differences in Evolutionary and Behavioral Genetic Approaches to APB

On the surface, evolutionary and behavioral genetic approaches to understanding APB (and other phenomenon) would appear to be directly compatible. Specifically, for genes that promote certain behavior to exist within individuals, they must have developed through the process of natural selection. Similarly for behaviors to have evolutionary origins, they must be coded for in genes that are passed down through sexual reproduction. However, it has been observed (e.g. Horgan, 1999) that evolutionary psychologists and behavioral geneticists oftentimes distance themselves from one another. Both fields may appear to benefit from distancing themselves from unpopular elements of the other field. For instance, in downplaying evolution, behavioral geneticists can avoid the controversy of

evolutionary theories. Some behavioral geneticists also may mollify critics by placing emphasis on gene-environment interactions or environmental effects (e.g. Moffit, 2005) rather than the direct effect of genes. Horgan (1999) argues that evolutionary psychologists also avoid specific genetic causal explanations for differences in behavior by focusing on commonalities in behavior rather than differences between individuals.

The result of the chasm between evolutionary psychology and behavioral genetics is that theoretical differences in the explanation of behavior arise. Tinbergen (1963) suggested that there are multiple levels at which behavior can be explained. Arguably, behavioral geneticists prefer to focus on proximate explanations for behavior (i.e. specific genes), whereas evolutionary psychologists look at ultimate causes of behavior (i.e., natural selection). It is not too much of a leap perhaps, to suggest that ultimate causes lead to proximal causes, which in turn lead to behavior. In other words, behavioral genetics research actually provides evidence in support of the mechanism through which the natural selection of evolutionary psychology influences human behavior, both in regards to commonalities and differences.

Behavioral Genetics and APB

Stated briefly, behavioral genetics studies attempt to ascertain the relative contribution of genetic and non-genetic influences in explaining traits, characteristics, or patterns of behavior at the population/sample level. Given that identical (monozygotic, MZ) twins share all of their genetic material and fraternal (dizygotic, DZ) twins share approximately half of their genetic material, but (it is assumed) share environments to similar degrees, it is concluded that the correlated behavior of MZ twins should be twice that of DZ twins (Moffitt, 2005). Any variations from this observation can be attributed to non-genetic effects.

Meta-analytic reviews of behavioral genetic studies for APB suggest that approximately 40%–50% of the variance can be explained through genetic influences (Rhee & Waldman, 2002; Miles & Carey, 1997). These results suggest that genetic influences provide a significant contribution to APB. These reviews, while providing important evidence for the genetic basis are not without some limitations. Miles and Carey (1997), for instance, discuss laboratory measures of aggression that have not been properly validated in regards to APD (Tedeschi & Quigley, 1996; Ferguson, 2007). Rhee and Waldman (2002) include a wide range of studies, which is both a strength (for inclusion and breadth) and a weakness (as arguably more recent studies may have improved on deficiencies from previous studies). Neither of the previous meta-analyses attempts to understand the results from behavioral genetics studies of APB from a theoretical framework that would explain the development of genetic predisposition for APB and understand how genetic risk is influenced by environmental threats. Nonetheless, the behavioral genetics data provides evidence for proximal genetic causes of APB. Yet it

is important to understand proximal genetic traits by examining evolution as an ultimate cause of those genetic traits.

The Controversy Over Evolutionary Explanations of Human Behavior

Given that behavioral genetic studies of APB provide evidence for a significant genetic contribution, it is somewhat surprising to find considerable resistance to discussing or studying the evolution of APB or violence in the social sciences. Arguably, modern social science has focused on "pitfalls" of modern life such as media violence, toy guns, and Western values, although violence and homicide rates are found to be high among non-advanced cultures without access to these modern accruements (Buss & Shackelford, 1997). For instance, a brochure on youth violence available from the American Psychological Association's Web site states that "There is no gene for violence. Violence is a learned behavior . . ." (APA, 1996). Although the brochure goes on to suggest that genetically influenced factors such as learning disabilities and impulsivity may interact with learned violence, this brochure inaccurately suggests that genetics do not directly contribute to violence. The brochure is outdated also in suggesting that youth violence is on the rise, when in fact it has been on a precipitous decline for 15 years. Similarly, it has been noted that the National Institutes of Health have de-emphasized genetic, evolutionary, or other biological studies of violence behavior (Enserink, 2000). Critiques of biological theories of aggression are perhaps epitomized by Berkowitz (1993), who claims that aggression has not been linked to biological storage areas for an aggressive instinct, and that aggression is normally provoked by external stimuli. From this perspective, aggression would seem to only be biological if it were univariate, purposeless, and unprovoked. Tooby and Cosmides (1992) argue that perspectives such as those of Berkowitz are indicative of the "Standard Social Science Model" (SSSM), which postulates the brain as a general-purpose learning device, devoid of content at birth, with behavior solely a product of subsequent learning. By contrast, evolutionary psychology views the brain as having evolved through countless generations of environmental pressures, wherein certain largely pre-wired brain "modules" give organisms in-born mechanisms for dealing with likely environmental stressors.

As for the reluctance of the psychological discipline to embrace the contributions of evolutionary theory, three main reasons would appear to be relevant. The first of these is simply a matter of dogma and indoctrination. Prior to the mid-20th century, genetic explanations for behavior were common. However, difficulties in conducting genetics research in humans and abuses of evolutionary theory, including eugenics and the belief in racial differences in intelligence (Kamin, 1974) decreased the appeal of evolutionary explanations for many. As environmental explanations (particularly those focused on learning) gained prominence in psychology in the 20th century, genetic theories of human behavior were largely repudiated in favor of environmental theories. Arguably, this movement coincided with social changes in the United States

at that time, such as the increased influence of feminist theories. Which tended to focus on the innate "sameness" of individuals rather than on innate differences.

Secondly, misunderstandings about the nature of how evolutionary theory applies to human behavior may also increase resistance. Two common misconceptions about evolutionary psychology involve the "naturalistic fallacy" and concerns with regards to biological hard determinism. Briefly, the naturalistic fallacy is the belief (or fear) that if something is demonstrated to be due to biology, then this provides moral justification for the behavior. Similarly, biological hard determinism would imply that human behavior is due only to genetic (or other biological) effects and is not influenced by the environment, nor open to the effects of agency. However, evolutionary psychologists have indicated clearly that they do not endorse either the naturalistic fallacy or biological hard determinism (see Wilson, Dietrich, & Clark, 2003 for a discussion).

Finally, a qualitative difference exists between genetic causal influences and non-genetic causal influences. If non-genetic influences (whether biological or socialization) are demonstrated to cause a negative outcome, this observation provides an evident solution, namely that removing the causal influence will likely reduce the negative behavior. However, genetic influences that are shown to cause a negative outcome cannot logically or ethically be removed. This may be interpreted as suggesting that genetic causal influences offer no evident solution for negative behavior reduction; individuals may thus be reluctant to endorse such research under this misimpression, as it appears to offer little hope for behavioral change. However, blinding oneself to the influence of genetic elements on behavior, by necessity, blinds oneself also to gene/environment interaction effects, which may offer some solutions for the reduction of negative behavior. Understanding the genetic influences on behavior, and identifying these genetic risks within individuals, may result in treatments that theoretically could be targeted early and preventatively toward individuals who may have this genetic risk. Understandably, discussion of these possibilities raises considerable ethical concerns (see, for example, Williams, 1994). This is not to say that such techniques may not prove to be useful in the future, but that great care must be taken to ensure that any behavioral or medical interventions for violence prevention are undertaken only under strict ethical guidelines. Put directly, we enter problematic ethical territory once individuals are punished for crimes they have not yet committed. Ultimately, it may be decided that such approaches are either unpractical or wholly unethical. Nevertheless, even if that should be true, observations regarding the limits of the "practicality" of genetic effects on violence should not be intertwined with discussions of the "truthfulness" of such effects.

Understanding APB From an Evolutionary Framework

APB may be understood as a bi-product of normal human aggression. Here, use of the term "aggression" is similar to that proposed by Baron and Richardson

(1994). Specifically, aggressive behavior is here defined as intentional behavior produced to cause physical harm or humiliation to another person who wishes to avoid the harm. Violent antisocial behavior, by extension, is here defined as intentional behavior intended to cause physical harm or humiliation to another person who contrastingly wishes to avoid the harm *and the behavior is out of proportion with precipitating or provoking stimuli and carried out with disregard to the welfare or rights of others.* Thus all violent antisocial behaviors are aggressive, but not all aggressive behaviors are necessarily antisocial. For example, acting in self-defense in response to a threatening individual would be considered aggressive behavior, but not antisocial behavior.

Buss and Shackelford (1997) provide an excellent review of the underlying premises of an evolutionary understanding of aggressive behavior. Buss and Shackelford argue that aggression can be thought of as an adaptive response that can result in certain gains, such as co-opting or defending resources, increased mating options and mate fidelity and increased status. Aggression is not unitary or context-blind, but rather modular and context-specific, and one would expect aggression to be provoked by external stimuli. Our understanding of APB can be built upon several assumptions that are derived from evolutionary psychology models such as those provided by Buss and Shackelford. These are:

1. Human aggression is a normative and adaptive response that has a selective advantage for individuals (note this does not imply that it is morally desirable).
2. Restraining aggression (i.e., impulse control) is also a normative and adaptive response that has a selective advantage for individuals.
3. Aggressive and aggressive impulse control instincts respond to environmental stimuli, or catalysts, which are cognitively processed in order to select the most adaptive response to an environmental stressor.
4. The human brain has evolved separate systems or "devices" to manage separate aggression and aggression-reduction impulse control drives. These devices may at times compete, particularly when environmental catalysts are ambiguous.

From an evolutionary perspective, a behavior as ubiquitous as aggression is best understood as an adaptation to environmental pressures that provided a selective advantage to members of the species. That is to say, members of the species who possessed the genotype related to the production of aggressive behaviors were more likely to survive and produce viable offspring than members of the species with genotypes that were less likely to produce aggressive behaviors. The selective advantages provided by aggressive behaviors may be related to external pressures common to hominid species, such as the benefits of risk-taking during hunting, and fending off or attacking predatory species. Benefits to aggression may also include higher success regarding intraspecies

competitive pressures such as mate selection and supply of resources (see Sagan & Druyan, 1992 for a discussion).

Although aggression, in moderate amounts and in proportion to environmental threats, may be beneficial, high levels of aggression may clearly be "too much of a good thing" phenotypically speaking. High levels of aggression may place an individual at extreme risk for harm or may result in social rejection and depriving the individual of the benefits of social groups, the development of which also likely contributes to the survivability of individual hominid organisms. Therefore, an individual may benefit not only from being aggressive, but knowing when to be aggressive and when to restrain aggressive impulses. Just as an aggressive instinct may provide a selective advantage under some circumstances, so too an aggression-reduction instinct may provide a selective advantage under other circumstances. These ideas are consistent with Gray's theories on behavioral approach and inhibition systems (Gray, 1990). The aggression-reduction instinct may be synonymous with what is often referred to as "impulse control" or "executive functioning." Deficits in portions of the brain (i.e., frontal lobes of the cortex) related to executive functioning have been demonstrated to predict overly aggressive (i.e., antisocial) behavior (Mercer & Selby, 2005; Donovan & Ferraro, 1999; Soderstrom et al., 2002). Neuroimaging studies document that frontal cortex lesions are associated with impulsive aggression, and less-so with trait aggression (Leon-Carrion & Chacartegui-Ramos, in press). For example, Raine, Lencz, Bihrle, LaCasse, and Colletti (2000) found that individuals with APB have 11% less grey matter in the prefrontal cortex as compared to non-APB individuals. This was the case even in individuals without history of brain injury. Critchley et al., (2000) found similar results in violent individuals, as compared to non-violent controls, in the prefrontal cortex and in the amygdala and hippocampus. The numerous studies on frontal lobe functioning and violence are too numerous to summarize here in full, although several excellent review sources exist (e.g., Davidson, Putnam & Larson, 2000; Hare, 1993; Leon-Carrion & Chacartegui-Ramos, in press). Thus it is reasonable to conclude that an "impulse control device," such as would decrease the frequency of aggressive responses to ambiguous stimuli, may be located in the frontal lobes.

This gene/environment interaction is important to emphasize. The antisocial genotype, as with any genotype, is unlikely to produce a static array of behaviors across all environmental situations. Rather, genotype produces a behavioral range or range of behaviors in order to allow the individual to adjust to differing environmental threats. Environments with low threat or strain are less likely to elicit antisocial behavioral responses than are environments with high threat or strain. Understanding what environmental situations are likely to produce antisocial behaviors from individuals high in antisocial personality may provide promising avenues for prevention and intervention, with interventions targeted at increasing the behavioral range of antisocial individuals to

include more non-aggressive behavioral options. From an evolutionary prospective, the way in which genes and environment interact renders the individual more flexible in dealing with a host of potential environmental threats. A more behaviorally flexible organism is inherently more adaptive than a behaviorally rigid one.

The Current Study

The current meta-analysis will seek to explain the etiological origin of genetic effects, as well as gene-environmental interactions from an evolutionary framework. As such, the present study will provide an ultimate causal explanation for the proximal influences of specific genetic traits. Consistent with both previous research and an evolutionary perspective, it is hypothesized that a significant amount of the variance, most likely the largest single variance component, will be explainable through genetic influences. It is further hypothesized that genetic influences will be found to be highest in children and to diminish slightly across age, due to individuals accumulating non-genetic biological and non-biological influences, experiences, and injuries across time.

Method

Study Selection and Categorization

PsycINFO was searched for all articles published between the years of 1996 and 2006 (this criterion discussed below) that included the following search terms: (adopt* or twin or heritabil* or behavior genetic* or behavioral genetic*) and (violen* or violent crim* or crim* or aggress* or antisocial). The references of primary sources revealed in this search were also examined for studies that were not discovered during this initial search.

Articles were judged relevant if they met the following criteria:

(a) Articles had to have been published between the years of 1996–2006 (effectively an 11-year publication period). Limiting meta-analyses to more recent research allows for an examination of recent trends in the literature, in which methods may have improved over past years.
(b) Outcome variables had to clearly measure some element of antisocial, violent, or aggressive behavior. Criteria were essentially identical to those Rhee and Waldman (2002) discuss in depth.
(c) Articles had to include methodology (i.e., twin or adoption) for determining relative contributions of genetic and non-genetic influences in APB.

A total of 38 published studies comprised of 53 separate observations (some studies broke down results by gender, some did not) were found that met the

above criteria. The combined sample size for the included articles was 96,918. Articles in the current study were coded for the presence of several potential moderator variables, namely: (a) Age (below age twelve, twelve to eighteen and above eighteen), (b) Sex, (c) Whether the outcome variables clearly used measures of antisocial personality disorder according to DSM-IV criteria, indices of violent behavior such as police reports or self-reported violent criminal activity, or broader measures of violent or aggressive behaviors as found in clinical measures of behavior disorders related to aggression.

Calculating Effect Size Estimates and Statistical Analyses

Pearson's *r*, a flexible and easily interpreted index of effect size, was used as the effect size estimate in this study. Correlation coefficients were transformed to Fisher's *z*, weighted, averaged and transformed back to a pooled *r*, denoted r_+. The Comprehensive Meta-Analysis (CMA) software program was used to fit fixed effects models. Once the combined effect sizes were calculated for genetic, shared non-genetic and unique non-genetic variance components, these were transformed back to (h^2), (c^2) and (e^2) variance estimates. Due to rounding during the effect size calculation process it is likely that the final total variance may differ slightly from 100%. Table 1 presents all included studies, effect sizes for (h^2), (c^2) and (e^2) in terms of *r* and characterization on moderator variables.

Results

Table 2 presents the results from the meta-analysis on the entire group of studies broken down by genetic, shared non-genetic, and unique non-genetic variance components. As can be seen, genetic influences account for the largest component of the variance in APB, with 56% explained, shared non-genetic influences explaining 11% of the variance in APB, and unique non-genetic influences explaining 31%. These results indicate that genetics is a significant contributor to APB, but that non-genetic influences also remain important. Tests of homogeneity indicate the presence of moderator variables.

Analyses of Moderator Variables

Bivariate correlations were used to examine the effect of moderator variables on the effect size of the three variance components for APB. Results for genetic influences indicated that effect size was moderated by type of measurement used ($r = .38$, $p \leq .01$) with broader measure of aggression obtaining higher effect sizes than measures limited to DSM-IV criteria for antisocial personality disorder. Similarly, age was correlated ($r = -.40$, $p \leq .01$) with effect size, suggesting that genetics is a more powerful predictor of APB in younger individuals than in adults. Sex was not a significant moderator ($r = .06$, $p \geq .05$).

TABLE 1. Included Studies in Current Meta-Analysis

Study		*r*	Age	Sex	Outcome
Arseneault (2003)	h^2	.91	Child	Mixed	Externalizing
	c^2	.00			
	e^2	.42			
Bartels (2003) 1	h^2	.83	Teen	Male	Externalizing
	c^2	.10			
	e^2	.55			
2	h^2	.85	Teen	Female	
	c^2	.00			
	e^2	.53			
Blonigen (2006) 1	h^2	.70	Adult	Male	Antisocial
	c^2	.00			
	e^2	.72			
2	h^2	.70	Adult	Female	
	c^2	.00			
	e^2	.72			
Blonigen (2005)	h^2	.70	Teen	Mixed	Externalizing
	c^2	.00			
	e^2	.71			
Brendgen (2006)	h^2	.64	Child	Mixed	Violent
	c^2	.00			
	e^2	.77			
Brendgen (2005)	h^2	.79	Child	Mixed	Violent
	c^2	.00			
	e^2	.62			
Bullock (2006)	h^2	.53	Child	Mixed	Externalizing
	c^2	.53			
	e^2	.66			
Button (2005)	h^2	.65	Child	Mixed	Externalizing
	c^2	.14			
	e^2	.75			
Cleveland (2003)	h^2	.64	Teen	Mixed	Violent
	c^2	.24			
	e^2	.73			
Coccaro (1997)	h^2	.69	Adult	Male	Violent
	c^2	.00			
	e^2	.73			
Eley (2003)	h^2	.80	Child	Mixed	Externalizing
	c^2	.56			
	e^2	.33			

(Continued)

TABLE 1. ***(Continued)***

Study		*r*	Age	Sex	Outcome
Eley (1999) 1	h^2	.83	Child	Male	Externalizing
	c^2	.23			
	e^2	.50			
3	h^2	.83	Child	Female	Externalizing
	c^2	.23			
	e^2	.50			
Gelhorn (2006)	h^2	.78	Teen	Mixed	Antisocial
	c^2	.00			
	e^2	.62			
Gjone (1997)	h^2	.85	Child	Mixed	Externalizing
	c^2	.00			
	e^2	.53			
Goldstein (2001)	h^2	.56	Adult	Female	Antisocial
	c^2	.31			
	e^2	.77			
Hicks (2004)	h^2	.89	Teen	Mixed	Antisocial
	c^2	.00			
	e^2	.45			
Hudziak (1999) 1	h^2	.85	Child	Male	Externalizing
	c^2	.00			
	e^2	.52			
2	h^2	.83	Child	Female	
	c^2	.00			
	e^2	.56			
Hudziak (2003) 1	h^2	.78	Child	Male	Externalizing
	c^2	.45			
	e^2	.43			
2	h^2	.78	Child	Female	
	c^2	.46			
Jacobson (2000)	h^2	.57	Adult	Male	Antisocial
	c^2	.56			
	e^2	.60			
Jacobson (2002)1	h^2	.54	Adult	Male	Antisocial
	c^2	.39			
	e^2	.75			
2	h^2	.64	Adult	Female	
	c^2	.24			
	e^2	.75			

(Continued)

TABLE 1. ***(Continued)***

Study		*r*	Age	Sex	Outcome
Jaffee (2002)	h^2	.84	Child	Mixed	Externalizing
	c^2	.00			
	e^2	.54			
Koenen (2006) 1	h^2	.88	Child	Male	Externalizing
	c^2	.00			
	e^2	.47			
2	h^2	.88	Child	Female	
	c^2	.00			
	e^2	.48			
Larsson (2006)	h^2	.79	Teen	Mixed	Antisocial
	c^2	.00			
	e^2	.69			
Ligthart (2005) 1	h^2	.77	Child	Male	Externalizing
	c^2	.44			
	e^2	.46			
2	h^2	.79	Child	Female	
	c^2	.38			
	e^2	.47			
Malone (2004)	h^2	.57	Adult	Male	Antisocial
	c^2	.36			
	e^2	.74			
McGue (2006) 1	h^2	.65	Teen	Male	Antisocial
	c^2	.41			
	e^2	.64			
2	h^2	.69	Teen	Female	
	c^2	.35			
	e^2	.63			
O'Connor (1998)	h^2	.79	Teen	Mixed	Externalizing
	c^2	.47			
	e^2	.40			
Polderman (2006)	h^2	.70	Child	Mixed	Externalizing
	c^2	.00			
	e^2	.71			
Rushton (1996) 1	h^2	.86	Adult	Male	Antisocial
	c^2	.00			
	e^2	.51			
2	h^2	.00	Adult	Female	
	c^2	.80			
	e^2	.60			
Slutske (2001) 1	h^2	.82	Adult	Male	Externalizing
	c^2	.00			
	e^2	.57			

(Continued)

TABLE 1. ***(Continued)***

Study		*r*		Age	Sex	Outcome
Taylor (2003)		h^2	.62	Teen	Male	Antisocial
		c^2	.00			
		e^2	.78			
Thapar (1996)		h^2	.53	Child	Mixed	Antisocial
		c^2	.63			
		e^2	.57			
Tuvblad (2006)	1	h^2	.24	Teen	Male	Antisocial
		c^2	.72			
		e^2	.65			
	2	h^2	.77	Teen	Female	
		c^2	.41			
		e^2	.49			
Tuvblad (2004)	1	h^2	.52	Teen	Male	Externalizing
		c^2	.65			
		e^2	.56			
	2	h^2	.63	Teen	Female	
		c^2	.53			
		e^2	.57			
van Beijsterveldt (2003)	1	h^2	.82	Child	Mixed	Externalizing
		c^2	.44			
		e^2	.37			
	2	h^2	.74	Child	Female	
		c^2	.54			
		e^2	.41			
Viding (2005)		h^2	.75	Child	Mixed	Antisocial
		c^2	.41			
		e^2	.52			
Vierikko (2003)	1	h^2	.52	Child	Male	Externalizing
		c^2	.84			
		e^2	.30			
	2	h^2	.74	Child	Female	
		c^2	.56			
		e^2	.41			
Vierikko (2005)	1	h^2	.52	Teen	Male	Externalizing
		c^2	.79			
		e^2	.32			
	2	h^2	.84	Teen	Female	
		c^2	.42			
		e^2	.35			

Note. All studies are listed by first author last name and year. Manuscripts with multiple analyses (including analyses separated by gender) are delineated numerically after the date. Mixed = mixed sex sample.

TABLE 2. Meta-Analytic Results for Genetic, Shared Non-Genetic, and Unique Non-Genetic Variance Components in Antisocial Personality and Behavior

Component	r_+	95% C.I.	Homogeneity Test	Variance
Genetic	.75	(.74, .75)	$X^2(52) = 5813.8$, $p \leq .001$	56%
Shared Non-Genetic	.33	(.33, .34)	$X^2(52) = 7575.0$, $p \leq .001$	11%
Unique Non-Genetic	.55	(.54, .55)	$X^2(52) = 3550.8$, $p \leq .001$	31%

Note. r_+ = pooled correlation coefficient; C.I. = Confidence intervals; Variance = proportion of variance explained.

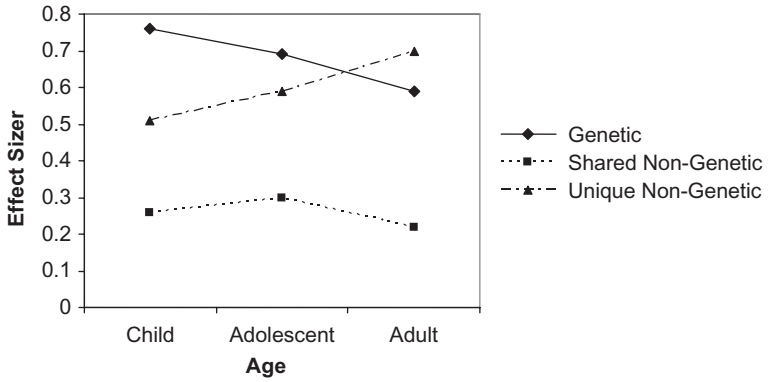

FIGURE 1. Age Trends in Genetic and Non-Genetic Influences in APB.

Results for shared non-genetic influences did not reveal significant moderating effects for measure used ($r = .06$), age ($r = -.05$) or sex ($r = .12$). Results for unique non-genetic influences did reveal significant moderators. Not surprisingly, these effects were opposite those for genetic influences with a significant correlation with type of measurement used ($r = -.54$, $p \leq .01$) and age ($r = .59$, $p \leq .01$). Sex was not a significant moderator ($r = -.06$, $p \geq .05$). Thus unique non-genetic influences are higher on measurements that use stricter DSM-IV criteria for antisocial personality disorder and for older individuals.

Results for the age moderator are particularly worthy of consideration. Figure 1 presents changes in effect size for genetic, shared non-genetic, and unique non-genetic effects across child, adolescent, and adult groups in terms of effect size r_+. As can be seen, the influence of both genetic and shared non-genetic influences decreases into adulthood, whereas the influence of unique

non-genetic influences increases into adulthood. As such, the proportion of variance in APB explained through genetic or shared non-genetic factors declines slightly across the lifespan. This is not surprising and likely reflects the gradual accumulation of non-genetic influences such as head injuries, infections, as well as socialization, and potentially increased agency (Bandura, 2006; Rychlak, 1999). It is possible as well that highly APB individuals are selected against by the environment. The likelihood of being killed or injured during an aggressive incident naturally increases across time, thus showing increased effect size for unique non-genetic effects. Thus it is important to understand that understanding the influence of genetic factors on APB depends somewhat at what point in the lifespan APB is examined. As unique non-genetic factors accumulated across time in the individual lifespan, naturally their influence on behavior increases. Genetic factors, being essentially unchanging, remain fairly static by comparison.

Discussion

Results from the current study highlight both the genetic effects and non-genetic effects on APB. As can be seen, genetic influences account for a considerable percentage of the variance. Understanding the development of these genetic factors from an evolutionary perspective allows us to understand the ultimate causal processes that lead to the development of APB. Shared non-genetic influences, which arguably are a reasonable indicator of family socialization (although other environmental influences that cause direct biological insults may also be part of this variance) has the smallest influence on APB. It is worth mentioning however, that 11% of the variance in APB is still a considerable percentage and worthy of attention, particularly as this portion of the variance may be particularly amenable to prevention or intervention.

Unique non-genetic influences accounted for about a third of the variance in APB. As this portion of the variance includes non-genetic biological influences such as head injuries and infections, non-family socialization processes, and potential agency effects (Bandura, 2006) this portion of the variance is most difficult to interpret. As such, until studies undertake to measure specific potential influences on APB, this portion of the variance may best be considered "unknown" variance, aside from the fact that we know it is variance that is unique to individuals and not due to shared influences. Utilizing specific measures of family violence exposure, family environment, peer relations, medical history, etc., as part of twin studies may help in elucidating the specific contributions of each of these potential non-genetic influences in the development of APB. Several studies have already begun to adopt such procedures (e.g., Caspi et al., 2004).

Results from this analysis are generally consistent with literature indicating that genetic contributions are an important influence in the development of APB (Larsson, Andershed, & Lichtenstein, 2006). As indicated earlier in the paper,

the presence of a significant genetic component to APB suggests evolutionary origins for this behavior. It would appear reasonable to conclude that aggressive behaviors have promoted the survivability of individual members in the species. Genes, thusly, are one predictor for individual variation (a component of natural selection) in this behavioral trait. Acknowledging this effect may be an important step in then examining specific gene-environment interactions that may promote APB.

An issue that bears mentioning is that of measurement standardization and validity. For instance, analyses have found that there is an inverse relationship between rigorous methodology and measurement in fields such as organizational psychology (Terpstra, 1981) and media violence effects (Ferguson, 2007), with higher effects reported in studies using poorly standardized and poorly validated instruments than for better instruments. Given criticisms of behavioral-genetics research, it is imperative that future research takes great care to use methods of the highest rigor and measurements with demonstrated reliability and validity.

Suggestions for future research involve including valid measures of non-genetic influences into twin study methodology to specifically examine the etiological contributions of these influences, once genetics has been controlled. Similarly, further research on gene/environment interactions would be helpful and likely offer the most positive route for intervention or prevention. Also, examining the catalytic impact of environmental strain on antisocial personalities and how the impact of this strain may be reduced would be a worthy avenue of research. Lastly, further research on the impulse control device theorized here may provide inroads in understanding how impulsive aggression, in particular, may be understood and treated.

AUTHOR NOTE

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