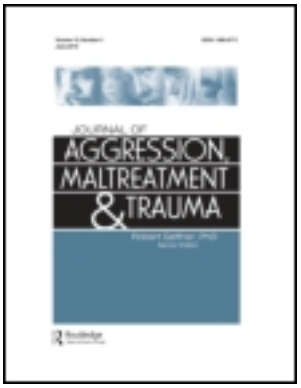

Journal of Aggression, Maltreatment & Trauma

Publication details, including instructions for authors and subscription information:
http://www.tandfonline.com/loi/wamt20

Genetic, Maternal, School, Intelligence, and Media Use Predictors of Adult Criminality: A Longitudinal Test of the Catalyst Model in Adolescence through Early Adulthood

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Published online: 17 May 2013.

To cite this article: Christopher J. Ferguson, James D. Ivory & Kevin M. Beaver (2013): Genetic, Maternal, School, Intelligence, and Media Use Predictors of Adult Criminality: A Longitudinal Test of the Catalyst Model in Adolescence through Early Adulthood, Journal of Aggression, Maltreatment & Trauma, 22:5, 447-460

To link to this article: http://dx.doi.org/10.1080/10926771.2013.785457

Journal of Aggression, Maltreatment & Trauma, 22:447–460, 2013

ISSN: 1092-6771 print/1545-083X online
DOI: 10.1080/10926771.2013.785457

Routledge
Taylor & Francis Group

RESEARCH ON BIOLOGICAL BASES AND COMPLEX PROCESSES OF BEHAVIOR

Genetic, Maternal, School, Intelligence, and Media Use Predictors of Adult Criminality: A Longitudinal Test of the Catalyst Model in Adolescence through Early Adulthood

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The catalyst model suggests that adult criminality arises from the interaction of genetic and proximal social influences such as family influences, but that distal social influences such as media exposure have only negligible influence. This article uses data from a 13-year longitudinal study of adolescent health to examine the catalyst model. As expected by the catalyst model, adult criminality was best explained by a confluence of genetic and proximal social risk factors. The influence of media exposure on adult criminality was negligible. Implications of these findings for both theory and policy are discussed.

Received 14 February 2012; revised 10 July 2012; accepted 11 July 2012.

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KEYWORDS adolescence, child development, computer games, criminal behavior, genetics, mass media, violence

The second half of the 20th century saw a pronounced increase in criminal behavior in the United States, particularly between the late 1960s and early 1990s (Federal Bureau of Investigation [FBI], 1951–2010). In the 1990s, this crime trajectory reversed, quickly returning rates of criminal behavior to 1960 levels. As of this writing, this downward trend continues (FBI, 1951–2010). This pattern has been observed for both nonviolent and violent criminal behaviors and, although less dramatic than in the United States, similar patterns have been identified in other industrialized nations across the same time frame (van Dijk, van Kesteren, & Smit, 2007). Not surprisingly, scholars have sought explanations both for the crime spike of the 1960s to the 1990s, as well as for the subsequent precipitous (and unexpected) decline. The crime wave of the 1960s through 1990s had been explained through racial tensions (Becker, 1968), mental health problems among youth (Anthony, 1968), increased dangerousness of mental health patients themselves (Cocozza, Melick, & Steadman, 1978), increasing population density (Spector, 1975), a purported belief in the rise of so-called juvenile "superpredators" (see Muschert, 2007), and the introduction of television (Centerwall, 1989). The subsequent decline in crime since the 1990s has generated almost as many speculative explanations, ranging from improved policing (Messner et al., 2007) to the *Roe v. Wade* theory, which speculates that the legalization of abortion resulted in the birth (or raising) of fewer antisocial children (Levitt & Dubner, 2005). Most scholars over the years have agreed that criminal behavior is difficult to explain in a compact and simple way, even with the best science available (Feshbach, 1971; Gottschalk & Ellis, 2010; Walsh & Ellis, 2007). This observation suggests that much research on the origins of criminal behavior might ultimately provide little guidance to policymakers about "what went wrong" during most of the latter part of the 20th century or "what went right" from the 1990s onward.

ALL HAVE WON AND MUST HAVE PRIZES?

We suspect that, if pressed, most psychologists and criminologists would tacitly agree with the general view that both biology (i.e., genetics in particular) and social environment can contribute in meaningful ways to criminal behavior. This general view is referred to by many, as we do here, as the nature–nurture compromise. In research practice, however, the application of this tacit view to theoretical frameworks and research designs is typically incomplete, imperfect, and vague. Many scholars might agree with the nature–nurture compromise in general principle, yet focus on only

social-environment-based explanations for criminal behavior and object to or ignore the application of genetic explanations, in particular to theories of criminal behavior, despite strong data indicating genetic contributors to criminality (Ferguson, 2010; Rhee & Waldman, 2002). For example, an American Psychological Association (APA, 1999; Reddy et al., 2001 noted this was done in collaboration with MTV) pamphlet on warning signs for youth violence states unequivocally that "violence is a learned behavior" and subsequently lists several social risk factors for violence with no mention of genetics. This preference among many scholars for models of behavior that focus on social environment rather than genetics is not a recent trend; rather, it has likely been advanced by various historical trends in scholarship ranging from fears that biological explanations for behavior might encourage eugenic abuses to the influential Chicago School's preference for environmental models of behavior beginning in the early 20th century (Sherry, 2004).

Thus we raise the concern that the nature–nurture compromise might, at times, offer a fig leaf for scholars to attack data that conflict with their ideological beliefs about social risk factors as primary culprits for criminal behavior[1] while maintaining a veneer of theoretical flexibility about the role of genetics. In other words, some scholars might take the vocal position that is essentially, "I like genetic theories generally, but I hate every one individually." Naturally, we do not mean to imply all scholars take such positions, merely that such issues remain an obstacle to the integration of genetic and evolutionary models of criminal behavior with more traditional social science models of the same behavior (Frisell, Lichtenstein, & Långström, 2011).

Conversely, we observe that the relative paucity of efforts to integrate genetic and social research limits the utility of existing data addressing causes of criminal behavior. In this sense, genetic and social research continues on parallel paths; further, even divergent strands of social research might continue on parallel paths, with individual scholars pursuing research programs dedicated to specific risk factors of interest to them. Failure to integrate divergent strands of genetic and social research might result in spurious claims of importance for each purported risk factor to which such strands of research are dedicated. Alternately, more studies that investigate and control for more genetic and social risk factors simultaneously might separate the wheat from the chaff in terms of which risk factors are of real importance. For example, correlations between media violence viewing and criminal behavior tend to be very small to negligible (Ferguson & Kilburn, 2009; Savage, 2008; Sherry, 2001). It is thus reasonable to suggest that such correlations might be weakened further in multivariate analyses that include more social and genetic risk factors. For example, boys tend to view more violent media

[1] We hold the belief that scholars are human, and, as human beings, tend to interpret data in accordance with their pre-existing beliefs. As such we do not intend this comment as unduly critical or challenging.

and engage in more frequent acts of aggression (Olson, 2010). Controlling for gender alone in studies of media effects on aggression and violence could substantially reduce any observed link between media violence and crime; this possibility would be particularly overlooked in meta-analyses, which traditionally remain dependent on analysis of bivariate relationships. Allowing research on individual risk factors to continue progressing largely on separate parallel paths might artificially inflate the alleged explanatory power of all such risk factors.

As a result, increasingly it has been acknowledged that multivariate analyses are the "gold standard" in research with criminal behavior as outcome (Savage, 2004). Further, increasingly scholars understand that considering genetic and social risk factors in combination is important to truly understand which risk factors for criminal behavior are of the greatest importance (Frisell et al., 2011).

THE CATALYST MODEL

Ferguson and colleagues (Ferguson & Beaver, 2009; Ferguson et al., 2008) introduced the catalyst model as a theoretical model to help explain the interaction between genetic risk and specific social risk factors for antisocial acts. The catalyst model approaches criminal behavior from a public health perspective, treating criminal behavior, in essence, as a behavior disorder. Involving a diathesis-stress approach, the catalyst model builds on previous work on gene–environment interactions (e.g., Caspi et al., 2002; Ellis, 1991; Ellis & Walsh, 1997; Moffitt, 2005) and seeks to extend this work by examining the ways in which genetic risk interacts with social risk factors as well as stress in the environment. The catalyst model is also specific in stating that not all risk factors are equal—that there is a need to separate the wheat from the chaff, as it were. For example, the catalyst model (see Figure 1)

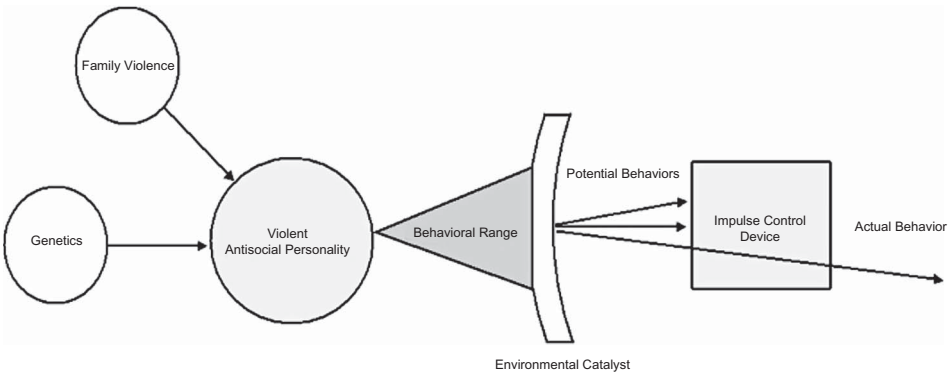

FIGURE 1 The catalyst model.

proposes that proximal social risk factors such as family environment and peer delinquency interact significantly with genetic risk, whereas more distal influences such as media use have negligible impact.

The catalyst model is thus essentially a diathesis-stress model (Zubin & Spring, 1977), albeit with some greater specificity about which social risk factors are of importance and which are not. Put briefly, the catalyst model posits that the interaction of genetic risk with early influences such as parental support, nurturance, or abuse produces personality profiles that are more or less prone to antisocial behavior. Actual antisocial behavior, according to the catalyst model, is also most likely to occur during times of stress. Antisocial behavior is inhibited through executive functioning and behavioral inhibition centers located in the prefrontal cortex, as damage to these areas is known to exacerbate antisocial behavior (e.g., Brower & Price, 2001; Mercer & Selby, 2005).

Initial testing of the catalyst model has demonstrated its superiority to standard social risk models of aggressiveness (Ferguson et al., 2008) and the model presents a set of testable pathways for future research. In this article, we propose to examine the efficacy of the catalyst model in predicting adult criminal behavior as measured through arrest history. The following hypotheses were tested: (a) a significant proportion of the variance in youth's subsequent adult arrest history will be predicted by genetic factors; (b) genetic factors will interact with parental factors, in this case maternal nurturance, in predicting adult criminality; (c) experiencing periods of stress, in this case as indicated by school stress and failure, will predict adult criminality; and (d) more distal risk factors, in this case media use, will not predict adult criminality.

This study builds on previous research in several ways. First, relatively few studies have considered both genetic effects and a broad variety of social risk factors (here, parental warmth, school problems, and media use) in combination. Second, this study uses a longitudinal design with data collected from participants during a period lasting from adolescence through early adulthood. Third, the main outcome measure will be participants' arrests, maintaining source independence between the outcome data and the predictor data (Baumrind, Larzelere, & Cowan, 2002).

METHODS

Participants

This study makes use of data from the National Longitudinal Study of Adolescent Health (Add Health; Resnick et al., 1997). The Add Health data set includes a multiwave longitudinal nationally representative sample of U.S. adolescents. Specific details on the general sample and recruitment can be found in the original publication of the Add Health data (Resnick et al.,

1997). Of interest to this study were the hundreds of monozygotic (MZ) and dizygotic (DZ) twin pairs included in the sample, with a careful selection of only same-sex twin pairs to avoid conflating heritability with gender differences in the DZ twin pairs. Both twin siblings from a total of 288 MZ and 292 same-sex DZ twin pairs were thus included in this study. Given that MZ twins share all genetic variation, whereas DZ twins share only a part of their genetic variation (similar to nontwin siblings) but share upbringing environment to the same degree as MZ twins, an examination of twins is one method to attempt to parse genetic from environmental influences and examine their interaction.

This analysis made use of data collected during three waves of the Add Health Study: (a) when adolescents were first recruited into the Add Health study in 1994–1995; (b) the Wave 3 follow-up, which occurred approximately 7 years later in 2001–2002; and (c) the Wave 4 data collected in 2008, approximately 13 years after initial recruitment. The average age of the teenagers was approximately 16 ($SD = 1.75$) at the time of recruitment for the study and the initial wave of data collection. Regarding race, 61.4% of the sample identified as Caucasian, 23.1% as African American, 3.6% as Native American, 7.6% as Asian American, and 9.4% as other. (The percentages add up to more than 100% as biracial participants were allowed to self-identify into more than one category.) Regarding ethnicity, 17% identified as being of Hispanic ethnicity. The current twin sample includes 292 (50.3%) male and 288 (49.7%) female twins. As noted earlier, all twin pairs selected are same-sex twins. These data are summarized in Table 1.

Measures

Maternal warmth

The maternal warmth scale consisted of five items related to the individual's perception that his or her mother was kind, loving, and supportive. Sample

TABLE 1 Sample Characteristics of Twin Pairs

Gender (twin pairs)	
Male	292 (50.3%)
Female	288 (49.7%)
Mean age	16 ($SD = 1.75$)
Race	
Caucasian	61.4%
African American	23.1%
Native American	3.6%
Asian American	7.6%
Other	9.4%
Hispanic ethnicity	17%
Twin status	
Monozygotic	288 (49.7%)
Dizygotic	292 (50.3%)

items include "Most of the time, your mother is warm and loving toward you" and "You are satisfied with the way your mother and you communicate with each other." Coefficient alpha for these items was .84. This scale was administered during Wave 1, and no corresponding scale for fathers was included in the Add Health data set.

School problems

The school problems scale consisted of 10 items related to difficulties getting along with teachers, getting along with other students, paying attention, and feeling happy and safe at school. Sample items include "During the 1994–1995 school year, how often did you have trouble getting along with your teachers?" and "During the 1994–1995 school year, how often did you have trouble paying attention in school?" Coefficient alpha was .76, and this variable was assessed during Wave 1.

Media use

During Wave 1 data collection, participants reported on the frequency of their television use and computer game use. Scholars have described the media, including both television and video games, as saturated with violent images and have proposed that frequency of media use might be one cause of criminal behavior. As such, this variable is included in his analysis. In this sample, television and computer use were moderately correlated ($r = .32$, $p < .01$). Frequency of television use and computer game use were summed to generate an overall frequency of media use variable.

Adolescent delinquency

The adolescent delinquency scale consisted of 15 items measuring unruly and antisocial behaviors during adolescence including fighting, selling drugs, stealing, and so on. These data were collected during Wave 1 of the Add Health Study. Sample items include "How often did you use or threaten to use a weapon to get something from someone?" and "How often did you drive a car without its owner's permission?" Coefficient alpha for this scale was .84.

Intelligence

The Peabody Picture Vocabulary Test (Dunn & Dunn, 1981) was developed as a nonverbal test of intelligence. It is included in this study as a control variable given speculation that criminal behavior, or at least being arrested, is correlated with lower intelligence (Beaver & Wright, 2011).

Arrest history

The Add Health database included a history of arrests occurring through Wave 4. The use of arrest history as an outcome variable for criminal behavior has positive and negative issues. Positively, arrest history is independent of subjective individual interpretations of what might constitute "criminal" behavior in self-report surveys, and also removes the problem of positive image management issues and socially desirable responding. Using arrest data as the outcome also preserves independence between the predictor and outcome variables to reduce spurious correlations (Baumrind et al., 2002). However, we acknowledge that not all criminal behaviors result in arrests, leaving the possibility that our measure underestimates the actual criminal behaviors committed by participants in our sample.

Procedures

Specific procedures related to recruitment and data collection are included in the original Add Health report (Resnick et al., 1997). Analyses for this study were conducted with SPSS software. Genetically informative analyses were conducted using the DF Analysis Model, $K_1 = b_0 + b_1(K_2 - K_m) + b_2[R * (K_2 - K_m)] + e$. In this model, the individual's score (K_1) is related to his or her sibling's score on the same variable (K_2), the mean for the scale (K_m), and the degree of genetic relatedness (R) and residual variance (e). Entered into a regression equation the $(K_2 - K_m)$ refers to the shared environment term, $[R * (K_2 - K_m)]$ to heredity, and e refers to nonshared environment plus error variance (Rodgers & Kohler, 2005). Main predictive analyses were conducted using hierarchical multiple regressions. Variables were entered in the order from most proximal (biological sex, genetics, intelligence, previous delinquency) to midproximal (maternal influences and school stress) to most distal (media influences) in accordance with the catalyst model. An interaction variable between maternal warmth and the heredity estimate $[R * (K_2 - K_m)]$ was also included in the model to examine the hypothesized interaction between maternal warmth and heritability. Both variables were centered before creating the interaction term to reduce multicollinearity. Terms related to social risk factors (maternal warmth, school problems, media use) were transformed using difference scores between twins in the manner suggested by Purcell and Koenen (2005) to reduce bias and highlight nonshared, nongenetic variance. In this analysis, we did not consider all possible social risk factors such as race or socioeconomic status (SES) to avoid collinearity (SES, in particular, tends to covary highly with school issues, for instance) as well as reduced power in the regression equation due to multiple predictor variables. We do not mean to imply these variables are unworthy of consideration, but rather maintain the most parsimonious test of the catalyst model. Collinearity diagnostics revealed an absence of multicollinearity in the regression equation (lowest tolerance was .72, highest Variable Inflation Factor [VIF] was 1.38).

RESULTS

Heritability of Adult Criminality

Heritability analyses were conducted separately for males and females. Among females, genetic heritability (h^2) accounted for 58% (95% CI [52%, 63%]) of the variance in arrest history, with 16% (95% CI [8%, 24%]) due to shared nongenetic factors (c^2). The remaining 26% (95% CI [18%, 33%]) of the variance is due to nonshared nongenetic factors (e^2). For males, the picture was somewhat different, with genetic heritability accounting for 20% (95% CI [12%, 28%]) of the variance, shared nongenetic accounting for another 20% (95% CI [12%, 28%]) of the variance, and nonshared nongenetic factors accounting for the remaining 60% (95% CI [55%, 65%]). These data are presented in Figure 2.

Longitudinal Predictors of Adult Criminality

The regression equation described earlier was significant through the final model, $R = .53$, R^2 adj $= .26$; $F(8, 256) = 12.59$. Among predictors included in the regression equation, male sex ($\beta = .17$; 95% CI [.09, .25]), adolescent delinquency ($\beta = .23$; 95% CI [.15, .31]), the heritability coefficient ($\beta = .31$; 95% CI [.24, .38]), and intelligence ($\beta = -.20$; 95% CI [–.12, –.28]), were all significant predictors of arrests at Wave 4. Maternal warmth was not predictive of arrest history ($\beta = -.04$) nor were school difficulties ($\beta = .07$), but the interaction between maternal warmth and heritability was ($\beta = -.10$; 95% CI [–.02, –.18]). Media use history was not predictive of arrest history ($\beta = -.01$)

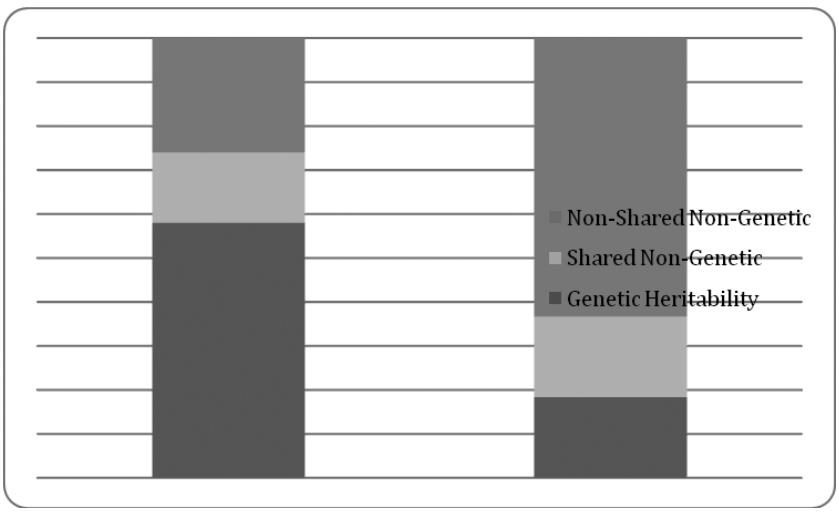

FIGURE 2 Heritability, shared nongenetic, and nonshared nongenetic influences on adult arrests.

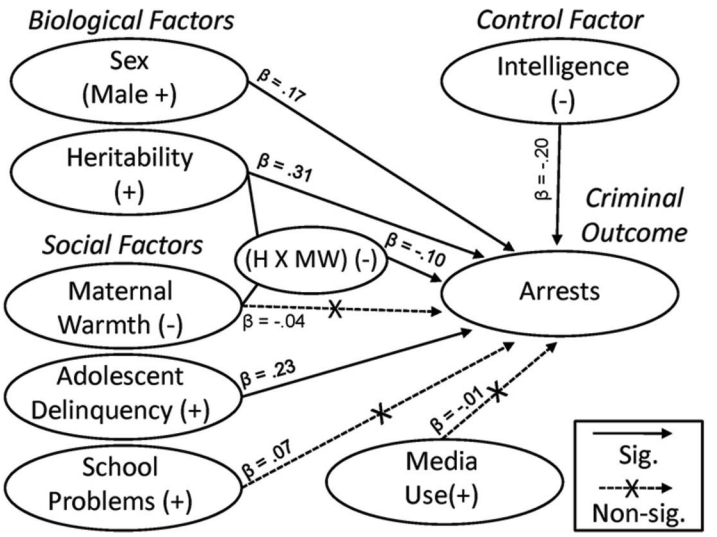

FIGURE 3 Visual representation of significant and nonsignificant predictors of adult arrests.

and ΔR^2 for the media use history step in the hierarchical regression was nonsignificant. Significant and nonsignificant predictors of adult arrests are presented in Figure 3.

DISCUSSION

In this longitudinal study employing data collected from participants during a period spanning adolescence through young adulthood, we examined the relative impact of genetics and several social risk factors on adult criminality as measured through arrest history. We found that arrest history was strongly related to heritability for females (58%) although somewhat less so for males (20%). Shared nongenetic factors, which tend to best represent identifiable social risk factors such as family environment, were comparable in females and males (16% and 20%, respectively). A considerable portion of the variance in adult criminality as measured through arrests, particularly for males, remains in nonshared nongenetic factors, essentially an "unknown" category consisting of idiosyncratic influences not shared between twins. This unknown category also presumably includes a certain amount of luck, particularly given that the measure is a measure of arrests to the exclusion of criminal behavior that did not result in arrests.

Once a heritability component had been controlled, male sex, a history of teen delinquency, lower intelligence, and a history of school problems all predicted adult criminality. Maternal warmth interacted with heritability such that maternal warmth functioned as a protective factor. Media use was not associated with either increased or decreased risk of adult criminality.

As a whole, these results fit reasonably well within the expectations of the catalyst model. School problems did not predict adult criminality but this might be because more immediate stress is more important than a history of past stress (e.g., see Ferguson, 2011). Aside from this, heritability interacted with maternal warmth in the expected manner. Further, the distal influence of media use was found not to predict adult criminality. These results suggest that the catalyst model is a good fit to the data and provides a reasonable theoretical template for understanding criminal behavior.

The strength of the catalyst model as we see it, and based on the data reported here, is that it fits well within a diathesis-stress or risk and resilience approach. However, it also acknowledges that not all risk factors are equal and that it is important to separate the wheat from the chaff so that scholarly efforts and public funding are directed in the most profitable directions. We observe that, at present, advocates of risk factors for criminality often assume an equivalence of value among social risks, which can result in a confused dispersal of limited resources. This equivalence of value approach tends to lead to potentially exaggerated statements regarding the importance of some risk factors, even when data from scholars' own studies suggests minor effects at most. The intrusion of "pet" risk factors and associated theories due to an assumption of equivalence might often be fed by particular political or activist agendas. For example, debates over the potential impact of media use have often been highly ideological and political (Grimes, Anderson, & Bergen, 2008; Williams & Skoric, 2005) and have been guided by an interest in the appropriateness of regulation and censorship (Livingstone, 1996). Our results here do not anticipate any particular gains made through pursuing policies based on the alleged effects of media on behavior, which appear in this instance to be quite minimal, particularly given that in the United States it can be difficult to legislate restriction of potentially harmful media content due to the high level of First Amendment–based scrutiny given to such restrictions (Levesque, 2007).

As noted previously, within recent generations criminality in the United States and other countries has experienced a precipitous climb followed by a remarkable fall. To date, our understanding of these trends remains limited (Fox & Piquero, 2003). We argue here that a complete understanding of criminal behavior cannot take place without a fuller understanding of the interaction of genetic and social risk factors for criminal behavior. Toward this goal, this study is envisioned as one small step.

Limitations and Conclusions

By working with a preexisting data set we did not have access to a full range of predictor variables. For instance, peer delinquency would have been a particularly important variable we would like to have considered. Similarly we would have liked to have had a better index of current environmental

stress than was available to us. Our measure of media exposure considered general exposure rather than exposure to violent material specifically. However, antimedia advocates routinely conclude that general media is saturated with harmful content and that exposure to general media content, thus, is itself harmful (e.g., APA, 1999; Center on Addiction and Substance Abuse, 2011). Therefore consideration of general media exposure is certainly of value, given these claims. Additionally, the arrest history outcome measure does not account for criminal behavior that did not result in arrest. We also urge caution in generalizing results to nontwin adolescents. Future research should explore the various ways in which the catalyst model can be used to predict other types of antisocial behaviors, including those that tap criminal behaviors that do not culminate in an arrest.

We hope that this article advances our understanding of genetic and social risk factors for criminal behavior, as well as their interaction. We believe this is a crucial direction toward a fuller understanding of criminal behavior that will allow more precise identification of risk factors, which in turn will allow these identified risk factors to receive attention from policy and social programs that is proportional to the risk factors' relative prominence rather than merely proportional to the attention given to them by isolated research agendas.

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