

Coming to grips with the cycle of violence

A commentary on: 'Violent crime runs in families: a total population study of 12.5 million individuals'
by Frisell *et al.* (2010)

C. J. Patrick* and U. Vaidyanathan

Department of Psychology, Florida State University, Tallahassee, FL, USA

Received 10 March 2010; Accepted 15 March 2010; First published online 29 April 2010

Key words: Epidemiology, heritability, violence.

Frisell *et al.*'s (2010) paper is a landmark effort that is sure to be influential and highly cited. It documents the phenomenon of familial transmission of violent behavior in concrete numeric terms using data from a nationwide sample of individuals. It provides compelling support for widely accepted, but still controversial, premises (e.g. concordance for violent behavior among family members reflects constitutional as well as experiential influences). It also presents evidence in support of newer emerging perspectives (e.g. the notion that heritability for specific behavioral proclivities can vary across differing environments – in the current instance, across differing social strata). In addition, it highlights intriguing new points – for example, the notion that particular forms of antisocial deviance (within Frisell *et al.*, specific subtypes of violent crimes) show heightened rates of family transmission, with greater behavioral specificity, than others.

All empirical studies, including this one, have methodological limitations that constrain inferences and interpretations. For example, while the use of matched controls comprises a strength of Frisell *et al.* (2010), effects of confounds cannot be ruled out entirely in a matched design. In particular, comparisons involving adoptive samples, which were utilized in Frisell *et al.* to infer genetic contributions to transmission of violence, carry risks of confounding due to the non-randomness of the adoption process (Cadoret, 1985; Billings *et al.* 1992). However, rather than focus on methodological points here, we focus instead on two major substantive issues highlighted by Frisell *et al.* (2010). One is the question of what etiologic factors underlie the heightened incidence of violent behavior observed among family members of violent

individuals. The other is what can be done to reduce their impact.

What violence-promoting characteristics are transmitted within families?

While presenting compelling evidence that violent behavior does indeed run in families, and that both genes and environment are likely to be involved in this transmission, Frisell *et al.* (2010) leave unresolved the question of what exactly is transmitted. Clearly, this question is enormously complex and many years of systematic research will be required to answer it. Nonetheless, recent advances in the literatures on aggression and antisocial behavior more broadly provide the basis for a coherent perspective on this question that can help to guide investigative efforts. This perspective emphasizes: (1) probabilistic liability as opposed to deterministic causation; (2) interplay between genes and environmental events (including the phenomenon of assortative mating) in the emergence of behavioral dispositions; (3) gender and development as moderators of genetic and environmental influence; (4) a hierarchical approach to conceptualizing problem behaviors and affiliated traits; and (5) neurobehavioral phenotypes as targets of study in research on risk for behavioral pathology.

'Born vulnerable' versus 'born violent'

Research on the biology of aggression has remained controversial over many years because of concerns that research of this type will lead certain individuals in society to be branded as 'violent' from birth. However, this concern is based on the outdated notion that specific genes give rise directly and inexorably to distinct phenotypic outcomes. The prevailing perspective in modern science, with respect to physical diseases as well as mental disorders, is that instances of direct biological determinism are the exception

* Address for correspondence: Dr C. J. Patrick, Department of Psychology, Florida State University, Tallahassee, FL, USA.
(Email: cpatrick@psy.fsu.edu)

rather than the rule. For the most part, genes establish liability or vulnerability to pathology, with multiple rather than single genes contributing to liability for pathologies of most types.

In Frisell *et al.* (2010), odds ratios for violent behaviors were elevated amongst family members of individuals exhibiting violence, but rates of violence among family members of affected persons were still low in absolute terms. Consistent with this, for child and adult antisocial behavior more broadly, rates of concordance between monozygotic co-twins (who possess identical genotypes) are far from perfect (Rhee & Waldman, 2002). These findings are in accordance with the idea that what is transmitted across generations within families is a heightened *propensity* for aggression.

Gene–environment interplay and assortative mating

While inferring a role for genetic factors in violent behavior, Frisell *et al.* (2010) also make a compelling case for the importance of environmental influences. In particular, effects of shared family environment were inferred from elevated rates of violence among unrelated adopted offspring of violent offenders, and from higher rates of violence among maternal as compared with paternal half-siblings of violent probands.

Beyond a role for environment *per se*, however, contemporary research findings point to moderating effects of parental behavior and other environmental influences on the phenotypic expression of particular underlying genotypes. This has been illustrated in the animal literature by research demonstrating intergenerational transmission of brain anomalies to be dependent on levels of maternal care exhibited toward offspring early in life (Liu *et al.* 2000). Of specific relevance to violence in humans, Caspi *et al.* (2002) reported evidence of a moderating effect of environment on the occurrence of aggressive behavior in relation to a specific gene polymorphism: Individuals with a genotype coding for diminished levels of monoamine oxidase A (MAOA) in the brain showed heightened rates of aggressive antisocial behavior only if exposed to maltreatment during childhood. Subsequent research has established the importance of gene \times environment interactions in behavior disorders of various types (e.g. Bakermans-Kranenburg & van IJzendoorn, 2006; Hicks *et al.* 2009). From this standpoint, variations in genetic makeup can be viewed as affecting mental health outcomes by either raising or lowering susceptibility to the impact of adverse environmental experiences on brain and behavioral function.

Additionally, the striking effects for assortative mating found in Frisell *et al.* (2010) replicate those

reported in previous work (e.g. Hicks *et al.* 2004) and highlight this as one key factor contributing to the perpetuation (if not escalation) of violent tendencies across generations within families. Regarding assortative mating and its role in violence transmission, it seems reasonable to suppose that genetic and environmental influences contribute hand-in-hand both to the systematic pairing of aggressive individuals (i.e. to the extent that preferences and opportunities reflect dispositions shaped by gene–environment interplay) as well as increased rates of aggression among offspring of such pairings (i.e. children in such cases are likely to experience more aggression in the home as well as carry greater constitutional liability for aggressive behavior).

Gender and development

A further point to consider in research on family transmission of violent behavior is the crucial role of gender and development as moderators of genetic and environmental influences. Generally, women tend to be less physically aggressive than men (e.g. Crick & Grotpeter, 1995) and engage in far fewer violent crimes (e.g. Pastore & Maguire, 2000; also see Table 1 in Frisell *et al.*). In addition, there is extensive evidence that gender moderates the association between stress and aggression, with females less prone to respond aggressively when stressed (Verona & Kilmer, 2007). Given these findings, it is highly intriguing that in Frisell *et al.* (2010), the most salient effect of gender was that female relatives of violent female probands showed greatly enhanced rates of violent crime compared with male relatives of violent male probands. The authors posited that environmental as well as genetic factors are likely to have contributed to this increased transmission for females. In particular, they highlighted social modeling as one likely source of environmental influence. Although specific constitutional factors were not discussed, a plausible hypothesis, considering that certain factors (probably constitutional as well as sociocultural) appear to mitigate against aggression in women as a whole, would be that those women who do behave violently are more likely than their male counterparts to lie toward the extreme of polygenic-constitutional liability toward aggression – with their close relatives more extreme also, on average.

With regard to development, the maturation of brain regions crucial to behavioral control continues well into adulthood, with particular genes exerting varying levels of influence at differing points in the maturational process. From the standpoint of genes as moderators of environmental experience, this means that the pathogenic impact of adverse environmental

events can also vary across stages of development. As a concrete illustration, recent evidence indicates that the expression of genetic liability to alcoholism is dramatically influenced by age of initial exposure to ethanol (e.g. Agrawal *et al.* 2009). Within Frisell *et al.* (2010), the potential role of developmental factors was illustrated by the finding of a moderating effect of probands' age at first conviction on odds ratios for violence among family members.

Hierarchical conceptualization of aggression and related problems

Traditionally, behavioral problems of differing types have been conceptualized as separate entities with distinctive etiologies. However, this way of thinking is challenged by the well-established finding of systematic co-morbidity among particular sets of disorders. For example, disorders involving prominent anxiety and mood symptoms form one such subset, and disorders entailing aggressive antisocial behavior and substance-related problems another (Krueger, 1999; Kendler *et al.* 2003). Hierarchical models (e.g. Mineka *et al.* 1998; Krueger *et al.* 2002) have been formulated to account for this systematic overlap among disorders. The essence of these models is that families (spectra) of interrelated disorders can be conceptualized in terms of broad entities (factors) reflecting their overlap, along with narrower variables reflecting the unique, non-overlapping features of each. Variables of each type (i.e. common factors, disorder-specific variables) represent coherent targets for etiological research.

The hierarchical nature of behavioral pathologies is illustrated by findings of Frisell *et al.* pertaining to risk for specific subtypes of violence as compared with violence of any type. Generality of transmission was evidenced by increased rates of violence of any type among family members of individuals displaying violent acts of each specific type. At the same time, specificity of transmission was evidenced by the fact that violent behaviors of particular types (e.g. arson, homicide) were associated with greatly elevated risk for violence of the same type among family members. This pattern of results can be interpreted within the framework of hierarchical models. Some forms of violence (e.g. assault) are primarily expressions of a broad disposition toward behavioral deviancy; other forms reflect specific etiologic influences more so than this broad dispositional factor.

This hierarchical perspective on behavioral pathology provides a framework for thinking about what might be transmitted across individuals within families that promotes violent behavior. Part of the story appears to lie in the broad externalizing factor that reflects the systematic overlap between antisocial

tendencies and substance-abuse proneness. Behavior genetic research has shown that individual differences in general externalizing proneness are highly heritable (Krueger *et al.* 2002) and that this heritable propensity accounts substantially for parent-to-child transmission of antisocial and substance-related problems (Hicks *et al.* 2004). The role of general externalizing liability in family transmission of violent antisocial behavior could be evaluated by performing analyses similar to those undertaken by the authors, with measures of alcohol and drug-related problems included alongside indices of violent behavior. Work of this kind would provide insight into the extent to which concordance for violence among family members reflects transmission of a general externalizing propensity, as opposed to other etiologic influences that either shape the expression of this general propensity toward aggression (see Krueger *et al.* 2007) or operate separately, to promote specific forms of violent behavior (for crimes such as arson and homicide, see Frisell *et al.* 2010).

Neurobehavioral phenotypes as targets for research

A major challenge for future research on the etiology and prediction of violence will be to identify aspects of brain processing that correspond to broad *versus* narrow aggressive propensities (e.g. general proneness to aggressive acts *versus* specific proclivities toward fire-setting, lethal attack, etc). Regarding broad dispositional factors contributing to aggression, recent research has shown that common brain response measures such as the P300 and error-related negativity operate as reliable indicators of general externalizing propensity (Patrick, 2008; Nelson *et al.* in press). This work in conjunction with other lines of evidence points to a dispositional weakness in inhibitory control, traceable to anomalies in frontal brain function, as the neuropsychological basis of this general propensity (Patrick & Bernat, 2009).

The construct of deficient inhibitory control represents an example of a neurobehavioral construct – that is, a conceptual entity with direct referents in neurobiology and behavior. Systematic research on constructs of this type, operationalized either behaviorally or through physiological response measures, is needed to advance our understanding of how genetic and environmental influences operate to produce individual differences in aggression proneness (Patrick & Bernat, 2010).

What can be done to reduce the transmission of violent tendencies?

The article by Frisell *et al.* (2010) takes a public health perspective on the problem of violent behavior in

society. From this perspective, like cancer, heart disease, or suicide, proneness to violence can be viewed as a condition that arises from the joint impact of constitutional and environmental influences across time on the physiological systems of individuals. As with cancer or heart disease, the nature and degree of constitutional vulnerability varies from individual to individual, so that in some cases limited environmental adversity is required for aggressive tendencies to emerge; in cases like this, special precautionary measures (e.g. limiting early exposure to violent media; psychosocial or pharmacological interventions to heighten emotional sensitivity) would be required to prevent the underlying vulnerability from being actuated. In other cases where the vulnerability is less pronounced or less specifically aggression-oriented, measures aimed at preventing exposure to extreme violence-promoting experiences (e.g. physical or sexual abuse) or decreasing hostile-aggressive interactions within families may be sufficient to prevent violent tendencies from arising. At the broader societal level, a public health perspective on violence encourages policies and programs aimed at expanding the range of support resources available to individuals from at-risk families, and promoting more adaptive decision-making in domains related to partner selection, family planning, child care, etc.

In any case, a crucial first step toward addressing the problem of violence transmission within families is to recognize that the problem in fact exists. Frisell *et al.* (2010) have performed an important public service by presenting the most compelling empirical account to date of the existence and extent of the problem. It is hoped that their work will serve as inspiration for systematic research efforts aimed at delineating constitutional and environmental factors contributing to aggression proneness, and encourage national funding agencies to support research of this kind in the interests of public health and welfare.

Declaration of Interest

None.

References

- Agrawal A, Sartor CE, Lynskey MT, Grant JD, Pergadia ML, Grucza R, Bucholz KK, Nelson EC, Madden PAF, Martin NG, Heath AC (2009). Evidence for an interaction between age at first drink and genetic influences on DSM-IV alcohol dependence symptoms. *Alcoholism: Clinical and Experimental Research* 33, 2047–2056.
- Bakermans-Kranenburg MJ, van IJzendoorn MH (2006). Gene-environment interaction of the dopamine D4

receptor (DRD4) and observed maternal insensitivity predicting externalizing behavior in preschoolers. *Developmental Psychobiology* 48, 406–409.

- Billings PR, Beckwith J, Alper JS (1992). The genetic analysis of human behavior: a new era? *Social Science and Medicine* 35, 227–238.
- Cadoret RJ (1985). Genes, environment, and their interaction in the development of psychopathology. In *Genetic Aspects of Human Behavior* (ed. T. Sakai and T. Tsuboi), pp. 165–175. Igaku-Shoin Ltd: Tokyo.
- Caspi A, McLay J, Moffitt TE, Mill J, Marin J, Craig IW, Taylor A, Poulton R (2002). Role of genotype in the cycle of violence in maltreated children. *Science* 297, 851–854.
- Crick NR, Grotpeter JK (1995). Relational aggression, gender, and social-psychological adjustment. *Child Development* 66, 710–722.
- Frisell T, Lichtenstein, Långström N (2010). Violent crime runs in families: a total population study of 12.5 million individuals. *Psychological Medicine*. Published online 25 March 2010. doi:10.1017/S0033291710000462.
- Hicks BM, DiRago AC, Iacono WG, McGue M (2009). Gene-environment interplay in internalizing disorders: consistent findings across six environmental risk factors. *Journal of Child Psychology and Psychiatry* 50, 1309–1317.
- Hicks BM, Krueger RF, Iacono WG, McGue MK, Patrick CJ (2004). The family transmission and heritability of externalizing disorders. *Archives of General Psychiatry* 61, 922–928.
- Kendler KS, Prescott CA, Myers J, Neale MC (2003). The structure of genetic and environmental risk factors for common psychiatric and substance use disorders in men and women. *Archives of General Psychiatry* 60, 929–937.
- Krueger RF (1999). The structure of common mental disorders. *Archives of General Psychiatry* 56, 921–926.
- Krueger RF, Hicks BM, Patrick CJ, Carlson SR, Iacono WG, McGue M (2002). Etiologic connections among substance dependence, antisocial behavior, and personality: modeling the externalizing spectrum. *Journal of Abnormal Psychology* 111, 411–424.
- Krueger RF, Markon KE, Patrick CJ, Benning S, Kramer M (2007). Linking antisocial behavior, substance use, and personality: an integrative quantitative model of the adult externalizing spectrum. *Journal of Abnormal Psychology* 116, 645–666.
- Liu D, Diorio J, Day JC, Francis DD, Meaney MJ (2000). Maternal care, hippocampal synaptogenesis, and cognitive development in rats. *Nature Neuroscience* 3, 799–806.
- Mineka S, Watson D, Clark LEA (1998). Co-morbidity of anxiety and unipolar mood disorders. *Annual Review of Psychology* 49, 377–412.
- Nelson LD, Patrick CJ, Bernat EM (in press). Operationalizing proneness to externalizing psychopathology as a multivariate psychophysiological phenotype. *Psychophysiology*.
- Pastore AL, Maguire K (2000). *Sourcebook of Criminal Justice Statistics 1999*. Washington, DC: U.S. Department of Justice, Bureau of Justice Statistics, U.S. Government Printing Office.
- Patrick CJ (2008). Psychophysiological correlates of aggression and violence: an integrative review.

Philosophical Transactions of the Royal Society B (Biological Sciences) **363**, 2543–2555.

Patrick CJ, Bernat EM (2009). From markers to mechanisms: using psychophysiological measures to elucidate basic processes underlying aggressive externalizing behavior.

In *Persistent Violent Offenders: Neuroscience and Rehabilitation* (ed. S. Hodgins, E. Viding and A. Plodowski), pp. 223–250. Oxford University Press: London.

Patrick CJ, Bernat EM (2010). Neuroscientific foundations of psychopathology. In *Contemporary Directions in*

Psychopathology: Scientific Foundations of the DSM-V and ICD-11 (ed. T. Millon, R. F. Krueger and E. Simonsen), pp. 419–452. Guilford Press: New York.

Rhee SH, Waldman ID (2002). Genetic and environmental influences on antisocial behavior: a meta-analysis of twin and adoption studies. *Psychological Bulletin* **128**, 490–529.

Verona E, Kilmer A (2007). Stress exposure and affective modulation of aggressive behavior in men and women. *Journal of Abnormal Psychology* **116**, 410–421.