Disordered Views of Aggressive Children

A Late Twentieth Century Perspective[a]

JOHN E. RICHTERS[b]

Child and Adolescent Disorders Research Branch
National Institute of Mental Health
5600 Fishers Lane, Room 18C-17
Rockville, Maryland 20857

INTRODUCTION

American society made an unannounced site visit to the social and behavioral sciences recently in the midst of a rising national epidemic of youth violence.[1-3] The unusual visit took place in hundreds of public and private forums around the United States as major institutions of local, state, and federal government turned to individual researchers, their professional organizations, and their funding agencies for science-based insights and solutions to the violence crisis. Although unannounced in the conventional sense, the collective visit certainly was not unexpected. As the National Research Council's Committee on Basic Research in the Behavioral and Social Sciences concluded almost a decade ago, "few demands on the behavioral and social sciences are more insistent than the call for knowledge on how to reduce the threat of these dangerous acts."[4] Indeed, the United States has invested heavily during the past half century in scientific approaches to understanding, curbing, and preventing childhood aggression. Thus, the national violence crisis provided a natural occasion and very specific agenda for seeking a return on that investment.

Those in search for an *immediate* solution to the violence crisis found little guidance from the scientific community. What they did find, however, was a rich foundation of data useful for thinking about solutions on a longer time horizon. Emerging from decades of scientific evidence concerning risk factors, concurrent correlates, sequelae, and life course patterns of aggression and antisocial behavior, several robust trends stood out as particularly salient.[5,6] First, the majority of children engage in some form of aggressive and/or antisocial behavior during their adolescent years but desist following a limited period of experimentation. Second, the most persistent and severe patterns of antisocial and aggressive behavior often originate much earlier in life among a much smaller group of children. Third, these latter children are significantly more likely than the others to come from disadvantaged backgrounds characterized by multiple risk factors, and to perform less well on measures of cognitive, emotional, social, and nervous system functioning. Fourth, once the aggressive and antisocial lifestyles of these children develop and stabilize, they tend to be remarkably refractory to interventions. There remains

[a] Sections of this paper are based on a more elaborate discussion of the conduct disorder issue.[6]

[b] Tel: (301) 443-5944; e-mail: jrichter@nih.gov.

little in the way of scientific consensus about *why* some children become persistently aggressive and *why* their antisocial trajectories are so difficult to deflect once stabilized.[6] Nonetheless, for many within and outside the scientific community these general trends provided a rationale for investing in early childhood prevention initiatives, focusing especially on children at highest risk for developing persistent problems of aggression and antisocial behavior.

The Federal Violence Initiative Controversy

As the national dialogue turned to a consideration of specific strategies for prevention, however, a firestorm of protest erupted over allegations that the federal government intended to harness these general trends as justification for adopting a draconian biomedical approach to violence prevention.[7] While ignoring social influences such as poverty, racism, poor education, poor parenting, according to critics, the so-called federal Violence Initiative would focus on biological causes of violence by seeking to identify and treat "violence-prone" children on the basis of biological markers for aggression. Moreover, the government's justification for this approach would be a science-based interpretation of persistent childhood aggression as a form of mental disorder — conduct disorder.[8]

Not surprisingly, the allegations elicited images of Huxley's *Brave New World,* charges of racism, eugenics, coercive biological approaches to social control, and an immediate wave of public protest. Similar fears had been stirred almost 20 years ago when a medical advisor to President Richard Nixon recommended a government-sponsored mass screening of all 6- to 8-year-olds in the United States to detect and correct "violent and homicidal tendencies."[9] Even though Dr. Hutschecker's ill-fated proposal was rejected and harshly criticized at the time, it was soon dismissed in the public mind as misguided advice from an isolated individual who happened to have access to the president. The current proposal, however, could not be as easily dismissed. A detailed rationale for the disorder-based biomedical strategy had been put forward this time by the eminent biological psychiatrist Dr. Frederick Goodwin, the first scientist to demonstrate the therapeutic antidepressant effects of lithium and an internationally recognized expert on manic-depressive illness.[10] Even more important to critics, Goodwin was also the federal government's highest ranking psychiatrist and head of its lead agency for mental health policy, services, and research.[7] Thus, in contrast to Hutschnecker's memo, Goodwin's proposal seemed to carry the combined authority of government endorsement and scientific justification.

In reality, neither perception was true. Goodwin's proposal had not received serious consideration in government planning, and it was neither compelled nor justified in the eyes of other scientists by a dispassionate assessment of the scientific evidence. The so-called biological proposal outlined by Goodwin was very much his own initiative and had been put forward in an effort to win a more central role for his agency in the govenrment's violence prevention planning.[7] Moreover, as the Secretary of Health and Human Services would later acknowledge, the so-called Violence Initiative with a capital *V* and a capital *I* never existed.[11] What did exist was an earnest long-range planning effort — warranting only a lower case *v* and i — to better coordinate existing government programs and to determine whether additional programs were needed to stem the national tide of violence.

By the time this became clear to all but the most ardent of critics, however, considerable damage already had been done. The controversy stemming from

Goodwin's proposal had immediately sabotaged what had been a constructive national dialogue about science-based approaches to violence prevention strategies. Soon thereafter, it stirred a sufficient level of public tension to force postponement of a government-funded scientific conference on genetics and crime.[11] Finally, it triggered the appointment of a blue-ribbon panel to conduct an unprecedented assessment of the adequacy of NIH-funded violence-related research in addressing public health needs and to ensure that the research was being conducted in an ethically and socially responsible manner.[12]

The NIH Blue-Ribbon Panel Review

The NIH panel's review did much to allay public fears about the alleged but nonexistent biomedically based federal Violence Initiative, but it was less effective in relieving public misgivings about the contemporary emphasis in scientific discourse on explanations of persistent childhood aggression as a form of mental disorder. The panel did, early in its deliberations, vigorously debate the controversial question of whether chronic childhood aggression is a medical disorder, a learned phenomenon, or multicomponent behavior. Some members found objectionable the claim that chronic and severe antisocial behavior in childhood and adolescence is necessarily evidence of an underlying mental disorder and raised concerns that it represented a counterproductive "medicalization" of a burgeoning and multidetermined social crisis *(i.e.,* antisocial behavior and violence) in the United States. No one on the NIH panel objected, in principle, to the notion that some chronic forms of violence and antisocial behavior might be products of underlying dysfunctions within individuals, warranting a mental disorder diagnosis and intervention. Rather, their comments stemmed from concerns about (1) the *overinclusiveness* of the criteria for conduct disorder, and (2) its potential for being misused to diagnose, label, and treat children and adolescents for a mental disorder when their behavior may instead be the product of deviant environments and/or subcultures. Some expressed the additional concern that the conduct disorder diagnosis and its consequences might be applied to disproportionate numbers of minority children.[12]

Far from arriving at anything approaching consensus on these concerns, the panel found itself mired in definitional ambiguities about the scientific meaning of terms, such as *psychopathology, mental disorder,* and *dysfunction.* One member even characterized the panel's debate as a "stroll through a semantic jungle"[12] (p. 78). In the end, the panel settled on expressing a more general concern about NIH-funded research focused narrowly on causes of aggression within the individual and recommended a shift toward more multidisciplinary research focusing on a broader range of relevant social influences, such as family, neighborhood, community, and culture.

OVERVIEW

It is customary following more traditional scientific site visits for investigators to engage in a period of debriefing, often focusing on the merits and implications of issues uncovered by the review. There was certainly nothing traditional about the recent site visit, either its unusual collective nature, its focus on the emotionally charged issue of mental disorder and aggression, or its concluding NIH panel

review. Nonetheless, the issues it highlighted about the causes of childhood aggression and antisocial behavior are every bit as worthy of scientific debriefing. The discussion that follows is intended to initiate that process by reaching beyond the rhetoric and confusion of the recent controversy to consider the scientific merits of contemporary claims that persistent childhood aggression reflects an underlying mental disorder.

Before turning to that agenda, however, it is worth noting what the conduct disorder controversy is *not* about. Namely, there is no question that persistent aggression and antisocial behavior in childhood is objectionable and intolerable on social, ethical, moral, and public health grounds. It places children themselves at high risk for a disturbing array of harmful outcomes, ranging from tremendous personal distress and social-emotional problems, to physical danger, imprisonment, and even early death. Moreover, these harms tend to radiate outward and afflict all those who come into contact with antisocial children — individuals, families, neighborhoods, institutions, and ultimately society itself. Thus, there has never been controversy about the social, clinical, and public health significance of the childhood aggression problem, only about the validity and implications of claims that it is caused by a mental disorder. In fact, it is precisely because of the social and public health significance of childhood aggression that a proper understanding of its origins and causes is considered so important.[12]

THE ASCENDANCY OF CONDUCT DISORDER

The notion that persistent childhood aggression and antisocial behavior reflects an underlying mental disorder within the child has existed in the literature of psychiatry for more than 200 years.[13] Moreover, it has existed in one form or another in the scientific literature for most of the twentieth century, ranging from specific concepts of disorder to more general models of social-emotional maladjustment.[2] Its controversial claim to scientific and public health legitimacy, however, is of more recent origin, dating back only to 1980 when persistent childhood aggression was officially classified as *conduct disorder,* a mental disorder of childhood, by the American Psychiatric Association.[6,13]

Diagnostic Criteria

The significance of this event lies in the ambitious and dubious causal claim on which the conduct disorder diagnosis rests. According to the current edition of the APA's *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV), all children (under age 18) who engage in at least 3 of 15 antisocial behaviors over a 12-month period, with at least one during the past six months, resulting in clinically significant impairment in social, academic, or occupational functioning, suffer from *conduct disorder,* a major mental disorder of childhood.[8] If there is evidence that at least one of the 15 antisocial behaviors was present before age 10, the child is further classified as having *conduct disorder, childhood-onset type*; the remaining children are classified as having *conduct disorder, adolescent-onset type.* The diagnosis ignores all contextual information about the child as possible influences on and/or alternative explanations for the antisocial behavior, including personal and family background, previous experiences, and current circumstances. The fact that a child has engaged in a clinically significant pattern of antisocial

behavior as defined by the behavioral diagnostic criteria is ipso facto evidence that he or she is suffering from a mental disorder. The DSM manual offers no defense or rationale for the disorder claim and does not specify exactly — or even generally — what has gone wrong within the child to cause the antisocial behavior. It is explicit only in claiming that the behavior pattern is a consequence of a dysfunction *within* the child.

Clinical Caveats

The DSM-IV diagnostic criteria reflect only modest changes from the criteria first published in 1980. In contrast to previous editions of the DSM, however, the DSM-IV included for the first time a vaguely worded but telling cautionary suggestion to clinicians: "It may be helpful for the clinician to consider the social and economic milieu in which undesirable behaviors occur before making a diagnosis."[8] The basis for this cautionary note, according to the manual, is that "concerns have been raised that the conduct disorder diagnosis may at times be applied to individuals in settings where patterns of undesirable behavior are sometimes viewed as protective *(e.g.,* threatening, impoverished, high crime)," further noting the example of "immigrant youth from war ravaged countries" (p. 88) whose behavior might have had survival value.[8] Despite the obvious intention of this "helpful suggestion," however, it is *only* a suggestion. It is not a requirement, it offers no guidelines for distinguishing between disordered and nondisorder variants of childhood antisocial behavior, and it is not reflected in the formal diagnostic criteria described above. Thus, whatever impact it may or may not have on the diagnostic decisions of practicing clinicians, it carries no weight in how the diagnosis is applied by researchers and epidemiologists, who study and estimate the prevalence and incidence of conduct disorder strictly on the basis of the formal criteria.

SCIENTIFIC AND PUBLIC HEALTH INFLUENCE

If the introduction of conduct disorder were simply a formalization of psychiatry's long-standing belief about the origins of childhood aggression, it might easily be ignored outside the narrow boundaries of the psychiatric profession. Since its introduction in 1980, however, the conduct disorder diagnosis has played an increasingly influential role in justifying, organizing, and interpreting government-funded research concerning risk factors, causes, preventions, and treatments for persistent childhood aggression. The magnitude of this impact is nowhere better reflected than in the change over two decades in the NIMH's posture concerning chronic aggression and mental disorder. In a 1968 statement, the NIMH conceptualized delinquent and criminal behaviors as "stemming from complex interactions of psychological, social, biological, and other factors," and specifically emphasized that the deviant behaviors warranted scientific study and not labels and definitions, such as *delinquency* and *mental disorder* [12](p. 148). By 1990, by contrast, research concerning the causes, prevention, and treatment of conduct disorder was given a high priority in the *National Institute of Mental Health's National Plan for Research on Child and Adolescent Mental Disorders."*

SIGNIFICANCE OF THE CONDUCT DISORDER CLAIM

There are a variety of reasons why the causal claim underlying the conduct disorder diagnosis warrants careful scientific scrutiny. First, as the recent controversy revealed, its current widespread usage in childhood aggression research is widely perceived as a tacit endorsement of the underlying causal claim. This perception in turn has considerable potential for inadvertently reinforcing stereotyped views of the causes of antisocial behavior that can restrict the range of intervention and prevention options considered by treatment providers and policy agencies. The Hutschnecker and Goodwin proposals were sobering reminders of how easily misguided solutions can spring from false beliefs about the causes of childhood aggression.

Second, as an ethical issue, the conduct disorder attribution carries with it the potential for long-term negative social consequences for those diagnosed. Two decades ago United States Senator Thomas Eagleton lost his bid for the vice presidency of the United States because of public concerns about his prior treatment for an affective disorder.[16] The comforting illusion that those days were well behind us was shattered again in 1996 when similar concerns were raised about the fact that the wife of General Colin Powell (retired) (himself a potential vice presidential nominee) had undergone treatment for an affective disorder. Unfortunately, stigmatization continues to play a significant role in the everyday lives of millions of Americans who are affected by mental disorders. These considerations are especially troubling at the close of the twentieth century, given the ever-expanding role of computer-aided access to information (including medical history) about the private lives of individuals. Ultimately, as in the case of all disorders, the most important question must be whether a diagnosis is scientifically valid and justified on the basis of its potential for conveying benefit to those diagnosed. That potential for benefit, however, must always be weighed against potential costs. It is not at all clear at present what those benefits are in the case of conduct disorder.

Third, as a social and public health issue, the conduct disorder attribution tends to focus attention on problems within the child and away from pathological conditions in the environment that may be largely responsible for his or her antisocial behavior. Finally, as a scientific matter, the assumption of underlying disorder tends also to have a powerful constraining influence on the questions that are asked and not asked by scientists about causes, treatments, and prevention strategies for childhood aggression.

IS PERSISTENT CHILDHOOD AGGRESSION A MENTAL DISORDER?

Negative Outcomes

By far, the most consistently cited justification for characterizing persistent childhood aggression as a form of mental disorder or psychopathology is the wealth of evidence showing that it is associated with significant levels of risk for a wide array of negative outcomes. For example, it has been amply demonstrated that, on average, children diagnosed with conduct disorder are much more likely to suffer from negative outcomes than children diagnosed with any of the other common behavioral syndromes and putative mental disorders of childhood. Beginning in preschool years, children who later display

serious conduct problems have been shown to manifest significantly higher levels of impulsivity, irritability, and inattention than their peers — characteristics associated with negative interactions with parents, peers, and teachers.[17-19] During middle childhood, children with conduct problems are more likely to be rejected by their peers," develop poor relationships with their teachers, engender less nurturance and support within the school setting,[18] and suffer from academic deficiencies." These factors, in turn, have been shown to be associated with an increased likelihood of deviant peer group affiliations,[22] which itself has been implicated as a risk factor for both substance abuse,[23] dropping out of school,[24] and criminal behavior.[25,26]

In the clinical domain, children diagnosed with conduct disorder have been found to suffer from a range of emotional adjustment problems, including attention deficit hyperactivity disorder, oppositional defiant disorder, anxiety, and depressive disorders. F inally, it has been shown that approximately 3 1% of those who warrant the conduct disorder diagnosis as children remain sufficiently antisocial through adulthood to qualify for the diagnosis of antisocial personality disorder after age 18, and to suffer as adults from a wide range of negative social, emotional, and health outcomes.[31,32]

Internal Functioning Deficits

Researchers in the domains of developmental psychopathology have amassed a wealth of data over the years showing that, on average, aggressive children tend to differ from nonaggressive children on a variety of measures of impulsivity, cognitive functioning, learning skills, emotion regulation,[33-37] and problem solving.[38] The most frequently cited basis for claims of an underlying disorder, however, has been the evidence that persistent aggression tends to be associated with putative deficits in neurological, biological, and/or psychophysiological functioning.[5] These findings have been discussed in detail elsewhere.[6] For present purposes, it will suffice to describe briefly the kinds of evidence cited.

For example, mild neuropsychological deficits have been found in numerous studies to be associated with delinquent and aggressive behavior in childhood, including deficiencies in attention modulation,[39,40] self-control and impulsivity,[41] verbal skills, memory, IQ, and visual-motor integration.[42] In the biochemical domain, children and adolescents hospitalized for disruptive disorders (including conduct disorder) compared to matched controls have been shown to have significantly lower levels of the serotonin metabolite 5-hydroxyindoleacetic acid (CSF 5-HIAA).[43] Moreover, these findings are generally consistent with earlier reports of an association between lower CSF 5-HIAA and both impulsivity and aggression in adults,[44,45] and between CSF 5-HIAA and aggression in free-ranging rhesus monkeys living under naturalistic conditions.[46] Finally, several studies have reported that electrodermal responses to external stimuli, an index of sympathetic activity reflecting processes related to anxiety and inhibition, tend to be diminished in groups of aggressive children and adolescents.[48,47] So-called undersocialized aggressive children especially have been shown to be more likely than controls to perseverate to their disadvantage with previously rewarded behaviors in the face of punishment.[48,49] These data converge with similar findings based on adult samples of incarcerated psychopaths, and with theoretical models of the role

played by passive avoidance learning deficits in acquiring antisocial behavior patterns.[50]

DISTINGUISHING BETWEEN DISORDER AND NONDISORDER

Plausible models and theories have been introduced over the years to account for how such characteristics might play direct and/or indirect causal roles in producing and/or sustaining antisocial behavior patterns, particularly those beginning in early childhood.[5] The crucial question, of course, is whether it is reasonable to interpret such descriptive findings as evidence for an underlying disorder or psychopathology. Many debates of the past concerning this question have been hampered both by limitations in the kinds of data available and by an absence of adequate theoretical models and frameworks for discriminating between disorder and nondisorder. During the past decade or so, however, there has been dramatic progress on both fronts. In the basic sciences there has been considerable progress in documenting and understanding the plasticity of the human nervous system, resulting in a much greater appreciation of the extent to which genetic, biological, and environmental factors interact in complex ways to influence human development.[51] Also, both reflecting and contributing to this progress, the hybrid discipline of developmental psychopathology has emerged as a powerful interdisciplinary framework for guiding research and theory concerning all aspects of human functioning and development.[52-54] Even more recently, there has been a resurgence of scholarly interest in defining the conceptual boundaries of mental disorder on the basis of evolutionary biology considerations.[55,56] Together, these developments provide a new foundation for thinking about the variety of factors that may lead ultimately to persistent childhood aggression, and for evaluating the scientific merits of claims that they reflect an underlying disorder within the child.

A Developmental Psychopathology Perspective

One of the most significant contributions of the developmental psychopathology perspective has been the heuristic power of its emphasis on the principle of equifinality drawn from systems theory.[57] Equifinality refers to the fundamental capacity of all open systems to achieve similar outcomes or behavior patterns through a variety of different causal pathways. Thus, not only may there be multiple contributors to the aggressive behavior of a given child, but the relevant causal processes may vary qualitatively between similar-appearing aggressive children. For one child the relevant causal influences may include a diathesis for impulsivity (inherited or acquired), poor self-esteem, a violent home environment and/or community, and minimal support or nurturance from caregivers.[58] For another child, however, the relevant factors may be positive characteristics, such as high intelligence and resourcefulness, in conjunction with conditions of poverty, poor parent monitoring, and/or a criminogenic neighborhood. Yet other children, for whom none of these factors is relevant, may develop persistent patterns of aggression and antisocial behavior primarily because they were socialized into a deviant value system, or as a strategy for coping in a hostile, dangerous environment. From this perspective, a major limitation of many efforts to identify and/or classify subtypes of aggressive children (including the conduct disorder classifica-

tion) has been the reliance on behavioral similarities, *not* on the presumed or postulated underlying causal processes of ultimate interest to developmentalists.

Disorder as Harmful Dysfunction

The developmental psychopathology perspective is of tremendous heuristic value in conceptualizing, isolating, and understanding complex causal processes and their interactions. Judgments concerning whether and under what conditions a particular matrix of causal influences warrants the attribution of an underlying mental disorder, however, require the additional guidance of a coherent definition of disorder. The search for such a definition has led to a variety of proposals over the decades, the most notable of which have differentially emphasized suboptimal functioning, statistical deviance, unexpectable distress/disability, and/or biological disadvantage. Each of these, however, has been shown to possess severe limitations in its ability to accommodate noncontroversial disorders while excluding conditions that are widely considered *not* to be disorders. On the basis of a review and critique of these limitations, Wakefield recently proposed an overarching, hybrid, "harmful dysfunction" concept of disorder, with an associated set of criteria for distinguishing between disorder and nondisorder in the domains of both physical and mental functioning:

> A condition is a disorder if and only if (a) the condition causes some harm or deprivation of benefit to the person as judged by the standards of the person's culture . . . , and (b) the condition results from the inability of some internal mechanism to perform its natural function, wherein a natural function is an effect that is part of the evolutionary explanation of the existence and structure of the rnechanism.[56]

By preserving the strengths and discarding the weaknesses of earlier proposals, the harmful dysfunction concept holds several advantages over its predecessors. First, it focuses on a broadly defined concept of internal mechanisms; it adopts an evolutionary biology approach that acknowledges the brain and its functions as legitimate manifestations of biological mechanisms developed through natural selection.[59] Although it assumes axiomatically that mental processes (*e.g.*, cognition, emotion, and perception) are ultimately traceable to underlying biological processes, it imposes no requirement to assess those processes at an anatomical or physiological level. Therefore, the harmful dysfunction concept is not yoked to the criterion of an identifiable physical lesion or any other single referent. Instead, those constructs may be postulated in psychological or biological terms, and may be indexed through biological and/or behavioral indicators at differing levels of abstraction. Consequently, the model is capable of accommodating both medical and mental disorders with equal ease.

Second, the harmful dysfunction concept emphasizes natural, evolved mechanisms within the individual that have gone awry. It therefore forces attention onto a specification of or reasoned speculation about those processes, and on the need to rule out alternative explanations in the form of normal functioning causal processes operating within, and environmental factors outside, the individual. It also minimizes the likelihood that conditions that are merely undesirable by social or political standards will be classified as disorders, as in the case of notable psychodiagnostic errors of the past such as "drapetomania" (the mental condition attributed to runaway slaves) and "childhood masturbation disorder" (see [56]). Finally, the harmful dysfunction criterion places on a more solid scientific platform

the basis for distinctions between disorder and other forms of human misery, unhappiness, and troubles.

Weaknesses in Existing Aggression-Disorder Claims

From the standpoint of the harmful dysfunction analysis, the correlates of persistent childhood aggression described earlier fall far short of meeting a reasonable standard for the disorder attribution. It is certainly true that most forms of persistent antisocial behavior in childhood are objectionable by conventional social and moral standards, and that parental concern over such behavior is often the basis for clinical referral. Moreover, there is a wealth of data demonstrating that aggressive children, particularly those whose antisocial behavior begins in early childhood, are at significant risk for a wide variety of harmful conditions and deprivations of benefit.[5] For a variety of methodological and conceptual reasons, however, the existing data do *not* provide adequate support for the claim that these behavior patterns are caused by dysfunctions of natural mechanisms within the child.

First, it is seldom clear whether identified differences between very aggressive and nonaggressive children on various indices of nervous system functioning reflect causes, spurious correlates, or consequences of the aggressive behavior. There is a regrettable bias in the literature toward strong causal interpretations of weak data patterns based on overly permissive scientific standards, frequently in the absence of any serious consideration of alternative, equally plausible explanations.This is particularly troubling in light of the impressive evidence from the basic neurosciences showing the extent to which nervous system functioning at all levels can be influenced by environmental experiences. Second, most of the identified differences in nervous system functioning between groups of aggressive and nonaggressive children have been relatively modest, typically reflecting considerable overlap between groups. Third, even if a rigorous case *could* be made for the causal influence on aggressive behavior of some basic characteristic of nervous system functioning, this alone would not constitute evidence for an underlying dysfunction in the implicated mechanism. Given that all behaviors, harmful and beneficial alike, are reflected ultimately in underlying physiological processes, it is only a matter of time before we will be able to identify the physiological underpinnings of all manner of behavior. This is not to say that it is necessary, desirable, or even possible to adopt a reductionist approach to understanding human functioning. Rather, it is intended to underscore the obvious fact that equating internal causes of harmful behavior with dysfunctions in natural mechanisms is logically equivalent to attributing all harmful behaviors to underlying disorders. It is precisely for this reason that the harmful dysfunction concept directs attention to *dysfunctions* of *natural* mechanisms: the failure of natural mechanisms to function in the capacities for which they evolved, resulting in harm to the individual.

FUTURE CHALLENGES

The concept of harmful dysfunction only circumscribes the conceptual decisions that must be made in discriminating disorders from nondisorders. It does not specify how those decisions are to be made. These decisions necessarily

require an admixture of complex scientific and value judgments. The identification of dysfunction, for example, requires scientific knowledge or theory about the natual mechanisms in question and their functional significance, as well as criteria for determining the dysfunctional status of those mechanisms or processes. Similarly, the harm requirement leaves open important questions concerning how to assess harmful effects, and how to discriminate between the *inability* of a mechanism to perform its natural function and a mere production deficit of an intact mechanism.

Although these decisions tend to be straightforward in the case of well-understood conditions, they become more controversial and subject to scientific dispute when the underlying mechanisms and processes are not yet well understood.[56] Thus, in the domain of mental disorders, tasks such as defining the domain of natural mechanisms, understanding the difference between normal variability and dysfunction, linking dysfunctions causally with harmful conditions, and even defining those harmful conditions are likely to be matters of considerable scientific dispute. These are not weaknesses, however, in the harmful dysfunction concept itself. Rather, they are burdens that must be shouldered within the substantive domains of developmental psychopathology, evolutionary biology, and related disciplines.[62] In the case of persistent childhood aggression, this burden is rendered especially difficult by limitations in our understanding of the relevant underlying mechanisms; the tentative, speculative nature of our knowledge about the causal status of variables that have been identified as correlates and/or risk factors; and our limited ability to operationalize and measure many of the constructs we believe to be most relevant.

Nondisordered Variants of Persistent Aggression

Just as the harmful dysfunction framework focuses attention on dysfunctions in natural mechanisms that might account for antisocial behavior, it also highlights the importance of considering how children might develop antisocial behavior patterns in the absence of internal dysfunctions. Their conduct problems instead may be caused by interactions between intact, normally functioning mechanisms and a variety of environmental influences. One obvious example might be children raised in criminogenic neighborhoods and/or families who engage in antisocial, even criminal actions because those are the behaviors modeled, expected, and/or rewarded by the major influences in their environments.[63] This is a particularly salient model to consider in the late twentieth century America, where in many major cities the allure of drug-related crime is ever present, and where gangs virtually control the social commerce and economic life of many neighborhoods. It is undeniably true that gang involvement places children at risk for an unimaginable array of negative outcomes, but from the perspective of many children living in those neighborhoods there are considerable physical and social risks as well in *not* joining a gang. Even beyond the issue of gangs, the short-term payoffs for participating in criminal and/or gang activity can be exceedingly attractive, especially to those who are disenfranchised, surrounded by violence and death, and who see no realistic chance of access to the opportunity/payoff matrix available to those in mainstream society. A reasonable case can be made that there may be many such children and youth for whom chronic antisocial behavior is, in our everyday sense of the word, a *choice* among alternatives; these children perform *willful* acts that are the products of deviant (from the mainstream) environments

and/or value systems and are therefore *psychopathologically exculpable* in the sense that there is no underlying dysfunction in their natural mechanisms.

The potential seductiveness of antisocial and criminal lifestyles was illustrated recently in the biography of Henry Hill, a New York gangster now residing in the federal witness protection program, whose life was depicted in the recent American film *GoodFellas.* As a young child, Hill's family lived across the street from a local mob hangout, providing him with a vantage point afforded to few in the neighborhood:

> I was the luckiest kid in the world . . . I was fascinated by the place. I used to watch them from my window, and I dreamed of being like them. At the age of twelve my ambition was to be a gangster. To be a wiseguy. To me being a wiseguy was better than being president of the United States. It meant power among people who had no power. It meant perks in a working-class neighborhood that had no privileges. To be a wiseguy was to own the world. I dreamed about being a wiseguy the way other kids dreamed about being doctors or movie stars or firemen or ballplayers[64] (p. 13).

Hill's lifelong pattern of antisocial and criminal behavior was sufficiently pervasive to warrant the diagnosis of conduct disorder in childhood and antisocial personality disorder as an adult. The question raised by his circumstances and account, however, is whether it is necessary, useful, or justified to attribute his behavior to an underlying mental disorder. There is considerable room here for reasonable speculation about possible functioning deficits within Hill *(e.g.,* deficient learning mechanisms, underactive inhibition system) that may have accounted for why he was so attracted to the criminal element. There also is ample reason to be wary of Hill's own account of his motivations.[65] It is also easy to imagine, however, how a normally functioning child exposed to those temptations, especially in conjunction with poor parent monitoring, supervision, and discipline, might be drawn into an antisocial and/or criminal lifestyle with mental processes intact—that is, in the absence of underlying dysfunction in the sense defined by Wakefield. We cannot know, of course, and we need not know in Hill's case, but the questions raised by his account are provocative and warrant careful consideration in the case of tens of thousands of children living in equally seductive environments, often in conjunction with poor parenting and other social risk factors, whose antisocial and criminal behavior patterns are attributed by the DSM-IV to an underlying mental disorder.

CONCLUSION

The example of Henry Hill and others like him highlights an important question that invariably arises in discussions of chronically antisocial children: Isn't a prolonged pattern of inherently dangerous, self-destructive behavior ipso facto evidence that there is something fundamentally wrong with a child? The answer depends very much on what we mean by "wrong." If wrong means that we find the behavior misguided, regrettable, morally repugnant, and a source of our concern about the child's values and welfare, then the term wrong certainly applies. Harmful behavior, however, by itself is too broad a criterion for the attribution of an underlying mental disorder. There are numerous legal occupations that also place individuals at much higher than average risk for high levels of personal distress, physical harm, and even early death. Police officers, firefighters, rescue workers, soldiers, and missionaries are just a few notable examples of those who

deliberately place themselves in harm's way on a regular basis. Yet we do not consider their behavior to be evidence of an underlying mental disorder. The reason is that we understand how the prevailing culture both values and rewards what they do; but this is no less true of the Henry Hills of the world. Mainstream society resists the notion that they can be fundamentally normally functioning human beings seduced by circumstance into deviant, antisocial lifestyles. As Nicholas Pileggi understood, however, the deviant (from mainstream) values and reward structures of subcultures can be every bit as coherent and powerful in their effects as those of the mainstream culture:

> For Henry and his wiseguy friends the world was golden. They lived in an environment awash in crime, and those who did not partake were simply viewed as prey. To live otherwise was foolish. Anyone who stood waiting his turn on the American pay line was beneath contempt. Those who did — who followed the rules, were stuck in low paying jobs, worried about their bills, put tiny amounts away for rainy days, kept their place, and crossed off workdays on their calendars like prisoners waiting their release — could only be considered fools . . . Henry and his pals had long ago dismissed the idea of security and the relative tranquility that went with obeying the law. They exulted in the pleasures that came from breaking it. Life was lived without a safety net. They wanted money, they wanted power, and they were willing to do anything necessary to achieve their ends[64] (p. 37).

We need not accept Hill's outlook and behavior to allow that these may be the characteristics of a normally functioning individual who has adopted and adapted to a different world view and set of values than those endorsed by the main culture.

It would be equally wrong, however, to assume that all antisocial children living in such high-risk environments arrive at their behavioral dispositions through normal processes. Indeed, much of what we know about the predictors and correlates of antisocial behavior suggests that there are probably numerous pathological pathways as well. The difficulty, of course, is that these very environments — those that are most likely to give rise to such psychopathologically exculpable behavior- are also among those with the most potential for producing dysfunctions in the Wakefieldian sense described earlier. The challenge for psychiatry and the developmental sciences is therefore to develop strategies and criteria for discriminating between what may be phenotypically similar though etiologically different forms of antisocial behavior. To assume instead that it always reflects an underlying mental disorder confuses different universes of discourse and frames of reference.[63] It blurs important distinctions between moral/social deviance and psychopathology, evaluative judgments of behavior and its underlying causes, and issues of public health and science. Moreover, a failure to maintain these distinctions jeopardizes any opportunity for constructing a meaningful concept of mental disorder and stands as an obstacle to achieving the level of scientific understanding necessary for guiding intervention efforts. This is particularly salient in the case of chronically antisocial and delinquent children and youth, for whom the collective efforts of science, psychology, psychiatry, criminology, juvenile justice, social work, and education have thus far failed to produce effective interventions.

REFERENCES

1. RICHTERS, J. E. 1993. Community violence and children's development: Toward a research agenda for the 1990's. Psychiatry **56**: 3-6.
2. RICHTERS, J. E. & P. E. MARTINEZ. 1993. Violent communities, family choices, and

children's chances; An algorithm for improving the odds. Dev. Psychopathol. **5:** 609-623.
3. RICHTERS, J. E. & P. MARTINEZ. 1993. The NIMH Community Violence Project: Children as victims and witnesses to violence. Psychiatry **56:** 7–21.
4. NATIONAL RESEARCH COUNCIL. 1988. The behavioral and social sciences: Achievements and opportunities. National Academy Press. Washington, DC.
5. MOFFITT, T. E. 1993. Adolescent-limited versus life-course persistent delinquency. Psychol. Rev. **100:** 674–701.
6. RICHTERS, J. E. & D. CICCHETTI. 1993. Mark Twain meets DSM-III-R: Conduct disorder, development, and the concept of harmful dysfunction. Dev. Psychopathol. **5:** 5–29.
7. WRIGHT, R. 1995. The biology of violence. The New Yorker (March 13).
8. AMERICAN PSYCHIATRIC ASSOCIATION. 1994. Diagnostic and Statistical Manual of Mental Disorders (4th ed.). American Psychiatric Association. Washington, D.C.
9. HUTSCHNECKER, A. 1969. A plan for prevention of crime. Memo to the President of the United States on the Eisenhower Commission on Crime.
10. GOODWIN, F. & K. JAMISON. 1990. Manic-depressive Illness. Oxford University Press. New York.
11. SULLIVAN, L. W. 1992. Remarks by the Secretary of Health and Human Services to the American Academy of Child and Adolescent Psychiatry. October 22. Washington, D.C.
12. NATIONAL INSTITUTES OF HEALTH. 1994. Report of the Panel on NIH Research on Antisocial, Aggressive, and Violence-related Behaviors and Their Consequences. National Institutes of Health. Rockville, Maryland.
13. LEWIS, D. O. 1996. Conduct disorder. In Child and Adolescent Psychiatry: A comprehensive textbook (2nd ed.). M. Lewis, Ed.: 564-577. Williams and Wilkins. New York.
14. AMERICAN PSYCHIATRIC ASSOCIATION. 1980. Diagnostic and Statistical Manual of Mental Disorders (3rd ed.). American Psychiatric Association. Washington, D.C.
15. NATIONAL ADVISORY MENTAL HEALTH COUNCIL. 1990. National Plan for Research on Child and Adolescent Mental Disorders. National Institute of Mental Health. Rockville, Maryland.
16. GARMEZY, N. 1978. Never mind the psychologists: Is it good for the children? Clin. Psychol. 31: 1-6.
17. CAMPBELL, S. B., A. M. BREAUX, L. J. EWING & E. K. SZUMOWSKI. 1986. Correlates and prediction of hyperactivity and aggression: A longitudinal study of parent-referred problem preschoolers. J. Abnorm. Child Psychol. **14:** 217–234.
18. CAMPBELL, S. B. 1991. Longitudinal studies of active and aggressive preschoolers: Individual differences in early behavior and in outcome. In Rochester Symposium on Developmental Psychopathology, Volume 2: Internalizing and Externalizing Expressions of Dysfunction. D. Cicchetti & S. L. Toth, Eds.: 57–89. Lawrence Erlbaum Associates. Hillsdale, NJ.
19. PATTERSON, G. R., D. CAPALDI & L. BANK. 1991. An early starter model for predicting delinquency. In The Development and Treatment of Childhood Aggression. D. Pepler & K. H. Rubin, Eds.: 139-168. Lawrence Erlbaum Associates. Hillsdale, NJ.
20. LADD, G. S., J. M. PRICE & C. H. HART. 1990. Preschooler's behavioral orientations and patterns of peer control: Predictive of peer status? In Peer Rejection in Childhood. S. R. Asher & J. D. Coie, Eds.: 90–115. Cambridge University Press. Cambridge.
21. MELTZER, L. J., M. D. LEVINE, W. KARNISKI, J. S. PALFREG & S. CLAREK. 1984. An analysis of the learning style of adolescent delinquents. J. Learn. Disabil. **17**: 600–608.
22. DISHION T. J., G. R. PATTERSON & M. S. SKINNER. 1989. April. A process model for the role of peers in adolescent social adjustment. Paper presented at the biennial meeting of the Society for Research in Child Development. Kansas City, MO.
23. DISHION, T. J. & R. LOEBER. 1985. Adolescent marijuana and alcohol use: The role of parents and peers revisited. Am. J. Drug Alcohol Abuse **11:** 11–15.
24. CAIRNS, R. B., B. D. CAIRNS & H. J. NECKERMAN. 1989. Early school dropout: Configurations and determinants. Child Dev. **60:** 1437–1452.

25. LOEBER, R. & T. J. DISHION 1983. Early predictors of male delinquency: A review. Psychol. Bull. **74**: 68–99.
26. LOEBER, R. & M. STOUTHAMER-LOEBER. 1987. Prediction. *In* Handbook of Juvenile Delinquency. H. C. Quay, Ed.: 325-382. Wiley. New York.
27. FARAONE, S. V., J. BIEDERMAN, K. KEENAN & M. T. TSUANG. 1991. Separation of DSM-III attention deficit disorder and conduct disorder: Evidence from a family genetic study of American child psychiatry patients. Psychol. Med. **21:** 109–121.
28. LOEBER, R. 1988. Natural histories of conduct problems, delinquency, and associated substance use: Evidence for developmental progressions. *In* Advances in Clinical Child Psychology. B. B. Lahey & A. E. Kazdin, Eds.: **11:** 73–124. Plenum Press. New York.
29. WALKER, J. L., B. B. LAHEY, M. F. RUSSO, M. A. G. CHRIST, K. MCBURNETT, R. LOEBER, M. STOUTHAMER-LOEBER & S. M. GREEN. 1991. Anxiety, inhibition, and conduct disorder in children: Relations to social impairment. J. Am. Acad. Child Adolesc. Psychiatry **30:** 187–191.
30. ZOCCOLILLO, M. 1992. Co-occurrence of conduct disorder and its adult outcomes with depressive and anxiety disorders: A review. J. Am. Acad. Child Adolesc. Psychiatry **31:** 547–556.
31. ZOCCOLILLO, M., A. PICKLES, D. QUINTON & M. RUTTER. 1992. The outcome of childhood conduct disorder: Implications for defining adult personality disorder. Psychol. Med. **22:** 971-986.
32. ROBINS, RL. N. 1966. Deviant Children Grown Up. Williams & Wilkins. Baltimore.
33. DODGE, K. A., J. BATES & G. S. PETTIT. 1990. Mechanisms in the cycle of violence. Science **250:** 1678–1683.
34. DODGE, K. A., G. S. PETTIT, C. L. MCCLASKEY & M. BROWN. 1986. Social competence in children. Monogr. Soc. Res. Child Dev. (Serial No. 213), Vol. 51, No. 2.
35. DODGE, K. A., R. R. MURPHY & K. BUCHSBAUM. 1984. The assessment of intention-cue detection skills in children: Implications for developmental psychopathology. Child Dev. **55:** 163-173.
36. LOCHMAN, J. E. 1987. Self and peer perceptions and attributional biases of aggressive and nonaggressive boys in dyadic interactions. J. Consult. Clin. Psychol. **55:** 404-410.
37. DODGE, K. A., J. D. COIE, G. S. PETTIT & J. M. PRICE. 1990. Peer status and aggression in boys' groups: Developmental and contextual analyses. Child Dev. **61:** 1289-1309.
38. ASARNOW, J. R. & J. W. CALLAN. 1985. Boys and with peer adjustment problems: Social cognitive processes. J. Consult. Clin. Psychol. **53:** 80-87.
39. NEWMAN, J. P. 1987. Reaction to punishment in extroverts and psychopaths: Implications for the impulsive behavior of disinhibited individuals. J. Res. Pers. **21:** 464-480.
40. NEWMAN, J. P. & E. HOWLAND. 1989. The effect of incentives on Wisconsin card sorting task performance in psychopaths. Unpublished manuscript. University of Wisconsin at Madison.
41. WHITE., J.., T. E. MOFFITT. A. CASPI. D. J. NEEDLES & M. STOUTHAMER-LOEBER. Measuring impulsivity and examining its relationship to delinquency. *In* Conduct Disorders in Children and Adolescents: Assessments on Intervention. G. P. Sholeva, Ed. American Psychiatric Press. Washington, D.C.
42. MOFFITT, T. E. & P. A. SILVA. 1988. IQ and delinquency: A direct test of the differential detection hypothesis. J. Abnorm. Psychol. **97:** 330-333.
43. KRUESI, M. J. P., J. L. RAPOPORT, S. D. HAMBURGER, E. D. HIBBS, W. Z. POTTER, M. LENARE & G. L. BROWN. 1990. Cerebrospinal fluid monoamine metabolites, aggression, and impulsivity in disruptive behavior disorders of children and adolescents. Arch. Gen. Psychiatry **47:** 419–426.
44. BROWN, G. L., F. K. GOODWIN, J. C. BALLENGER, P. F. GOYER & L. F. MAJOR. 1979. Aggression in humans correlates with cerebrospinal fluid amine metabolites. Psychiatry Res. **1:** 131-139.
45. COCARRO, E. F., L. J. SIEVER, H. M. KLAR, G. MAURER, K. COCHRANE, T. B. COOPER, R. C. MOHS & K. L. DAVIS. 1989. Serotonergic studies in patients with affective and personality disorders: Correlates with suicidal and impulsive aggressive behavior. Arch. Gen. Psychiatry **46:** 587-599.

46. HIGLEY, J. D., P. T. MEHLMAN, D. M. TAUB, S. B. HIGLEY, S. J. SUOMI, M. LINNOILA & J. H. VICKERS. 1992. Cerebrospinal fluid monoamine and adrenal correlates of aggression in free-ranging rhesus monkeys. Arch. Gen. Psychiatry **49**: 436-441.
47. RAINE, A., P. H. VENABLES & M. A. WILLIAMS. 1990. Autonomic orienting responses in 15-year-old male subjects and criminal behavior at age 24. Am. J. Psychiatry **147:** 933-937.
48. DAUGHERTY, T. K. & H. C. QUAY. 1991. Response perseverationanddelayed responding in childhood behavior disorders. J. Child Applied Discip. Psychol. **32:** 453-461.
49. SHAPIRO, S. K., H. C. QUAY, A. E. HOGAN & K. P. SCHWARTZ. 1988. Response perseveration and delayed responding in undersocialized aggressive conduct disorder. J. Abnorm. Psychol. **97**: 371-373
50. NEWMAN, J. P. & D. S. KOSSON. 1986. Passive avoidance learning in psychopathic and nonpsychopathic offenders. J. Abnorm. Psychol. **95:** 257-263.
51. CICCHETTI, D. 1996. Regulatory processes. Special Issue. Dev. Psychopathol. **8**: 1-305
52. CICCHETTI, D. 1984. The emergence of developmental psychopathology. Child Dev. **55:** 1–7.
53. CICCHETTI, D. 1990. Perspectives on the interface between normal and atypical development. Dev. Psychopathol. **2:** 329-333
54. SROUFE, L. A & M. RUTTER. 1984. The domain of developmental psychopathology. Child Dev. **55:** 17–29.
55. WAKEFIELD, J. C. 1992a. Disorder as harmful dysfunction: A conceptual critique of DSM-III-R definition of mental disorder. Psychol. Rev. **99:** 232-247
56. WAKEFIELD, J. C. 1992b. The concept of mental disorder: On the boundary between biological facts and social values. Am. Psychol. **47:** 373-388.
57. BERTALANFFY, L. VON. 1968. General System Theory. Braziller. New York.
58. CICCHETTI, D. & M. LYNCH. 1993. Toward an ecological/transactional model of community violence and child maltreatment: Consequences for children's development. Psychiatry **56:** 96–118.
59. Buss, D. M. 1984. Evolutionary biology and personality psychology: Toward a conception of human nature and individual differences. Am. Psychol. **39:** 1135–1147.
60. MEEHL, P. E. 1978. Theoretical risks and tabular asterisks: Sir Karl, Sir Ronald, and the slow progress of soft psychology. J. Consult. Clin. Psychol. **46:** 806–834.
61. MALTZ, M. D. 1994. Deviating from the mean: The declining significance of significance. J. Res. Crime Delinquency **31:** 434-436.
62. RICHTERS, J. E. & D. CICCHETTI. 1993. Editorial: Toward a developmental perspective on conduct disorder. Dev. Psychopathol. 5: 1–4.
63. MCCORD, J. 1993. Conduct disorder and antisocial behavior: Some thoughts about processes. Dev. Psychopathol. **5:** 321–329.
64. PILEGGI, N. 1985. Wiseguy: Life in a Mafia Family. Pocket Books. New York.
65. FARRINGTON, D. P. 1993. Motivations for conduct disorder and delinquency. Dev. Psychopathol. **5:** 225-241.