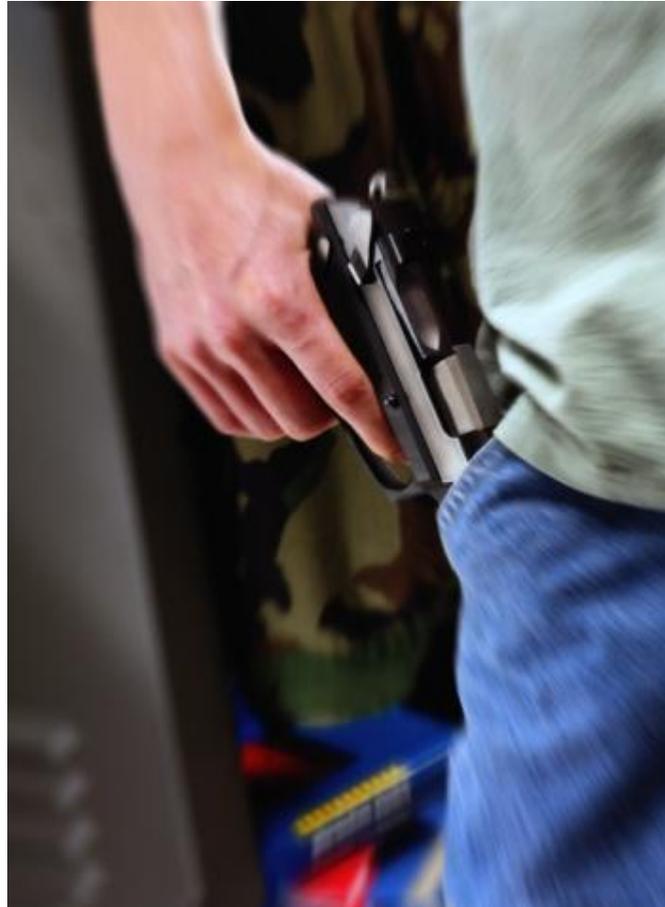


Forecasting Aggression: Toward a New Interdisciplinary Understanding of What Makes Some Troubled Youth Turn Violent

By Daniel S. Schechter, M.D.



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Editor's note: It takes a series of unfortunate circumstances for an adolescent to turn violent. While early exposure to familial violence can play a role, so too can biological influences such as hormone levels and genetic predispositions. The combination of these factors can be deadly. Although genes and other biological causes are difficult to identify and may be impossible to overcome through known therapeutic methods, medical professionals' intervention techniques can help minimize aggressive behavior related to environmental factors.

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“Scientists have already given us a good idea of the general conditions in which tornadoes form. It is the details that still need to be understood—in particular, the mixture of air pressure, temperature, and air circulation in which tornadic thunderstorms evolve... Why do some severe thunderstorms produce tornadoes while others do not? If scientists could find out the conditions in which [tornadic thunderstorms otherwise known as] supercells arise, there would be fewer instances of tornadoes spinning up without warning...”¹

It is every bit as complicated for a baby to develop into a violent adolescent as it is for a tornado to form on a beautiful spring day in Kansas. In each case, a complex, dynamic system needs to develop from multiple convergent elements, and researchers are only beginning to understand how these elements interact with one another.²

A 25-year longitudinal study of 926 babies in Christchurch, New Zealand, supports the idea that certain variables are good predictors that a child will develop aggressive behavior by adolescence. These predictors include poverty, changes in caregivers (as in foster care situations), childhood exposure to abuse and violence, parental substance abuse, maternal smoking during pregnancy, being male, having a lower IQ, and gravitating to conduct-disordered peers. These predictors might better be considered as associated risk factors that often occur together. But all of them together account for only about 40 percent of the probability that a child will develop a conduct disorder in adolescence.³ What is missing? Researchers conducting epidemiologic studies have recently begun examining the genetic, developmental, and deeper psychological factors—and interactions among them—to see if they can account for the other 60 percent.

Parent-Child Attachment

The study of human attachment, especially between a young child and parent, has led to consistent findings across many populations and cultures. Central to these has been the identification of risk factors for the perpetuation of violence and maltreatment across generations. John Bowlby, the father of attachment theory, began his clinical observations of teenagers with conduct disorders in his paper “Forty-four Juvenile Thieves: Their Characters and their Home-Life.”⁴ The mental representation of these 44 youths’ attachment relationships yielded a concept that Bowlby called “internal working models of attachment.”^{5,6} Some children who experience parental abuse go on to become violent; they may retain a working model of what it is like to be in a relationship with an abusive parent who dominates aggressively and a co-parent who is victimized.⁷ Among people who are maltreated or exposed to family violence as children, about one-third will become violent;

another third will remain at risk to become violent toward their children or their romantic partner.^{8,9} In a prospective study, harsh, excessively punitive and abusive parenting during preschool predicted aggressive behavior by a child toward his or her peers in elementary school more strongly than other child- and family-related variables such as child hyperactivity, family poverty, or teenaged parents.¹⁰

The majority of studies of child-parent attachment are performed when a child is 12 to 24 months old using a simple laboratory procedure called the Strange Situation Paradigm.¹¹ This observational measure helps determine a child's level and type of attachment to a parent based on the reaction when a parent enters, leaves, and re-enters a room. The child's reactions are categorized as either secure or insecure, based on the child's ability to approach and to communicate having missed the returning parent, as well as the ability to accept comfort and to resume play and exploration of toys with the parent. The disorganized/disoriented pattern of insecure attachment is characterized by the child's paradoxical approach and avoidance of the parent when the parent re-enters the room after separation during this paradigm. This pattern is further characterized by freezing and inconsistent strategy for relating to an attachment figure. A child might, for example, approach his returning mother but with his eyes closed, or he might stay at the door when his mother re-enters the room and then glance fleetingly toward her from a distance. Children who display this disorganized, disoriented pattern of insecure attachment behave more aggressively over time.¹² By ages three to five, many of these children try to act like they are the ones in charge as a way of taking control of relationships with others rather than being at the mercy of others' unpredictable behavior—they may boss others around or take care of others like a parent would.¹³ What leads some of these children to develop and maintain prominent aggressive, even violent, behavior into adolescence and adulthood?

Children with abusive parents have to maintain the illusion of being safe and protected by separating positive memories of their caretakers from frightening memories and suppressing the latter.¹⁴ An adaptive way for children to do this is to split or dissociate experiences, compartmentalizing memories in order to continue to depend on someone they fear.¹⁵ If they carry this pattern of dissociation to nonthreatening relationships, though, what had been adaptive becomes pathological, rigid, and overly simplified. Over time, people who have had to hide their violent experiences from themselves and others may develop a tendency to be both suddenly explosive and menacing, as well as to choose blindly to be with others who are violent toward them.¹⁶ They cannot form a realistic picture of their disturbed caregivers without the help of an outside perspective. They begin to assume that others with

whom they have an important relationship are dangerous and thus either must be attacked or avoided at all cost.

From Generation to Generation

What happens when violence-exposed individuals have children? Is there an environmental risk for the development of aggression in their offspring? In three studies, parents who suffered from violence-related post-traumatic stress disorder, or PTSD, were found to have difficulty detecting and responding to their young child's attempts to connect with them. They especially had trouble putting up with and setting limits on the typical distress, helplessness, and aggression that parents of young children encounter—they had trouble saying no and dealing with tantrums and day-to-day separation.^{17,18}

My laboratory's research has an additional emphasis: the effects of typical day-to-day toddler distress (i.e., separation anxiety and temper tantrums) on parent-child interactions over time, and therefore on the child's subsequent social and emotional development.^{19,20} When we followed up on families who had been in our study, we found that we could predict the severity of poorly regulated child aggression based on the severity of maternal, violence-related PTSD we had seen earlier.²¹ In addition, a pattern of four symptoms in the child—recklessness, hypervigilance, separation anxiety, and reversal of parent-child roles—cluster into a constellation called secure base distortion, in which the child can appear bossy and aggressive at one moment and then fearful or hyper-compliant at another.¹³ Violence in the child's life as well as in the parents' history may contribute to risk in terms of modeling a specific behavior (or “identifying with the aggressor,” as Anna Freud suggested)²² and via a parent's neglect of the child's need for checks on his own aggression, as parents often approach such a child's behavior from the point of view of a helpless victim rather than an empowered caretaker.

Other Biological and External Factors

Biology also can play a role in the development of violence in adolescents. Minor congenital anomalies and obstetrical complications have been associated with violence in adolescence,²³ although obstetrical complications may be a marker of family adversity more than a cause. The neurological consequences of complications, such as mild oxygen deprivation at birth, may interact with family adversity to increase the risk that the child will develop aggressive behavior. What initially appear to be constitutional factors may be secondary effects of social risk factors such as poverty and other family adversity.²⁴

Another popular theory used to explain the development of aggressive behavior in recent decades, particularly in the wake of the killings at Columbine High School, is that violent media adversely affect the developing brain.²⁵ In and of itself, violence in video games and other visual media is not likely to lead to the development of aggressive behavior; rather, it may exacerbate pre-existing difficulties.²⁶ A recent study has shown that mothers exposed to real-life violence, suffering from PTSD, and living in high-risk, inner-city households tend to gravitate toward such media and expose their young children to it.²⁷

Developmental neurocognitive disturbances that impede verbal communication and go untreated also have been shown to increase the risk for development of violent behavior.²⁸ Disturbances in the parent-child relationship that lead to understimulation can aggravate such deficits.²⁹ These difficulties can interact with the attachment-theory-based notion of the development of mentalization, the ability to consider the thoughts and feelings of oneself and others.³⁰ By fostering mentalization and communication skills among the parents who can then both model and stimulate the development of these skills with their children, we might help diminish the likelihood of aggressive behavior when addressing interpersonal conflict.³¹ In other words, by encouraging parents to be conscious of what they think and feel about their child's expressions, gestures, play, and words, and then to consider what their child might be thinking and feeling at a given moment at their particular age, parents can improve important features of their relationships with their children.¹⁹

Sex and Genes

Researchers have found that both our sex and our genes are associated with the development of violence. Males commit the vast majority of violent crimes. A recent study of 90 nine-year-old children showed that the next strongest correlation, after male sex, is high androgen levels, which constitutes a biological marker to consider in relation to individual differences in aggressive behavior. Generally, the more testosterone, the more aggression.^{32,33} Testosterone is thought both to alter neuronal activity in particular regions of the brain that produce the neurotransmitters dopamine and serotonin and to affect the reuptake of serotonin. Excessive dopamine and low levels of serotonin in the central nervous system have been associated with increased aggression in primates.³⁴

We are also born with certain intrinsic risks for developing serious psychiatric disorders, researchers believe. Adults with psychotic disorders such as bipolar disorder and schizophrenia, who may also be dealing with substance abuse and severe personality disorders, are often thought to be at greater risk for violence—and, often, to have been so

since childhood or adolescence. If someone has difficulty telling what is real from what is imagined and has an additional condition such as substance abuse that reduces inhibition, the likelihood of carrying out violence is obviously greater.^{35,36} Even so, having multiple forms of severe psychopathology alone does not guarantee that a person is violent. Nearly all forms of psychopathology, including post-traumatic stress disorder,³⁷ involve risks related both to genes and to gene-environment interaction during development.

Can genetic variations, or polymorphisms, in key regions of the brain predict the development of aggressive behavior? Polymorphisms reflect differences in DNA sequences among individuals, groups, or populations. Genetic polymorphisms might account for an individual having blue or green eyes, straight or curly hair, or being behaviorally inhibited or impulsive. When a particular environmental condition is thought to exert an influence on a person significantly more often or exclusively when a specific polymorphism is present, a gene-environment interaction is thought to take place. In psychiatry, two of the fundamental gene-environment interactions involve early-childhood stress interacting with a variation of the serotonin transporter gene that is known as the short allele of the 5-HTTLPR. The interaction of this variation with stress from an adverse environment can lead to anxiety and depression in parents and children. It also can increase the risk for child maltreatment. For example, post-traumatic stress as a specific form of anxiety in the parent is associated with increased parental difficulty in reading child social cues. Another gene variation, called the MAO-A low activity allele, is associated with aggressive behavior.^{38,39}

Both of these polymorphisms center on the metabolism of two monoamine-type neurotransmitters: serotonin and dopamine. And both polymorphisms lead to increased levels of these two neurotransmitters in the brain. The short-allele of the serotonin transporter 5-HTTLPR gene is associated with increased levels of serotonin in the brain as compared to the long allele. The low-activity MAO-A fails to break down serotonin and norepinephrine—a dopamine-derived neurotransmitter that is associated with fear conditioning—and leads to elevated levels of serotonin and dopamine in the brain. A buildup of these monoamine-type neurotransmitters together can theoretically contribute to impulsivity and specifically to violent behavior. But, incongruously, *insufficient* serotonin alone in the nervous system is also thought to be a marker of violent behavior. And the short allele of the serotonin transporter 5-HTTLPR gene is thought to be associated with increased levels of serotonin in the central nervous system more than the long allele. This incongruity—whether it is too little or too much serotonin available in the nervous system—plus the fact that serotonergic pathways originating in the brain stem involve multiple brain regions and change across

human development—make it very hard to forecast the role of the serotonergic system in the development of violent tendencies in a given person across the lifespan.⁴⁰ In addition, people suffering from a variety of head injuries and temporal lobe epilepsy have displayed aggressive behavior, but never in a manner that would allow scientists to define a physical pathway of aggression. Little specific anatomical intervention, therefore, can absolutely and specifically stop aggressive behavior.

Genetic and environmental conditions may interact to produce an aggressive, “tornadic” individual, but the interactions can be surprising. Gene-environment interactions predicting aggression appear to be genetically more complex than first thought in the early 2000s. For example, the short allele of the serotonin transporter gene 5-HTTLPR is most commonly associated with anxiety and depression in the presence of an adverse environment.³⁹ However, an environmental factor may not always be needed. Two studies suggest that aggressive behavior is associated with distinct variations in two different genes: the serotonin transporter gene and the 7-repeat allele of the dopaminergic receptor gene DRDR.^{41,42} What underlies this association is not well understood and requires further study. This example of clinically significant interactions between genes and environmental conditions leading to aggression in people with particular genetic features and environmental influences is but one of many types of gene-gene and gene-gene-environmental interactions that researchers still must identify.

Other polymorphisms are implicated in the development of aggressive behavior. Among these are catechol-o-methyltransferase (COMT), which at low activity fails to degrade noradrenaline, leading to a more likely fight response; and brain-derived neurotrophic factor (BDNF), which at higher levels in the hippocampus may also be associated with aggressive behavior.^{43,44} We do not yet understand how genes interact, nor do we understand how different environmental exposures might interact. It remains unknown how abrupt environmental shifts, such as those experienced in immigration, transfer into foster care, or adoption, might interact in the context of development to lead to the expression of certain genetic traits and proneness to certain states associated with violent behavior.⁴⁵

Working to Control the Weather

What forms the tornado in the end? And what makes it mild versus super destructive? Violent behavior emerges in various forms and degrees. That which is reactive to the misreading of others’ intentions differs from that which is self-serving and malevolent, for example. We are desperate to find the causes and antidotes for all forms and degrees of

aggressive behavior: We want to identify the bad allele(s) and the negative environmental conditions that interact and contribute to violence. Even labeling this allele or that environment as a prerequisite for violent behavior is not so simple. We have come to understand that certain alleles may promote plasticity, a potentially positive characteristic that enables adaptation to more stressful or less stressful environments. Such an allele can create a beneficial adaptation in good conditions and a detrimental adaptation in adverse conditions. Such adaptations have been compared to a rare and beautiful orchid that blooms under optimal environmental conditions and dies in compromised environments or a common and ordinary dandelion that thrives anywhere.^{46,47} In a positive caregiving environment, for example, a person with a short allele of the serotonin transporter gene 5-HTTLPR (found to be associated with depression and anxiety in a context of adversity) can actually adapt better than someone with the long allele, who is missing the same degree of plasticity and sensitivity to the environment.

In our current studies in Geneva, we are trying to understand what causes children of abused mothers to be more likely to show aggressive or conflict-avoidant behaviors over time. We may be promoting a new discipline of “psychiatric meteorology” in the process. While we cannot yet effectively change genes or congenital givens, we can improve evidence-based interventions that result in a change in the caregiving environment at the level of parental mental representations of child and self, the interactive behavior between mother and child, or the child’s individual development. The goal is to create better warning systems in the event that the vulnerable child early in development blows up into a severe storm.

In the end, our tornado analogy goes only so far. We may be in a much better position to effect change in the psychobiological atmosphere than in the Earth’s atmosphere. Multiple parent-infant psychotherapeutic interventions have been shown empirically to change the parent-child relationship at the level of mental representation, behavior, and /or child development so as to decrease risk for violence and increase healthy adaptation, growth, and learning.⁴⁸

Recent research shows that relationship-based interventions, while not altering DNA, might be associated with epigenetic brain changes, such as the addition of methyl groups to the proteins that surround DNA and turn on and off the possibility of generating proteins with important brain functions.^{49,50} In Geneva, we are currently studying how a simple, three-session, relationship-based parent intervention called the Clinician Assisted Videofeedback Exposure Sessions, or CAVES, is significantly changing the quality of mothers’ mental representations of their toddlers, such that the representations become more positive and

consistent with the child's actual age.¹⁹ We are working toward understanding how changes in caregiving following this intervention might affect the methylation of several genes that are linked to stress reactivity, fear conditioning, and fight-or-flight behavior in child and mother.

The videofeedback exposure sessions involve asking violence-exposed, traumatized mothers how they think both they and their child were feeling during videotaped play observations. We ask both before and after showing them very short video excerpts of their young child's "storms," or distress during separation, frustration, and introduction to novel, sometimes frightening, play stimuli. This intervention models mentalization such that in the calm after the storm, parents can access their own capacity to think about their children's experience from the children's point of view as much as possible. In so doing, they can more sensitively read their children's emotional communication—they might read it as "scared" rather than "angry and controlling," for example. We are now testing our hypothesis that with an adequate dose and reinforcement of new memories generated by such interventions, future storms will not form such destructive tornadoes.

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