

A Neurobiological Approach to Foreign Policy Analysis: Identifying Individual Differences  
in Political Violence

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**Abstract:** A great deal of foreign policy analyses relies on social and environmental factors, or anecdotal evidence. In seeking to address this problem in a more systematic manner, we move from an investigation centered around state actors to one focused on variation in individual behavior accounting for the combination of social, cultural, environmental, psychological and biological differences. Our proposed approach to the study of political violence requires the integration of methods and skills from geneticists and neuroscientists with those in the behavioral and social sciences. Specifically we seek to introduce an approach to study political violence which : 1) quantifies the effects of genes, environments, and their interaction on behavior; 2) identifies specific genetic and environmental contexts that lead to such behavior; 3) develops a comprehensive model of the biological and social pathways to political violence; and 4) identifies populations under specific circumstances which pose a higher or lower prevalence for any specific genes, neurobiological or environmental mechanisms which pose an increased liability for political violence; 5) develops mechanisms to identify individuals within given populations who are most at risk for committing violence, as well as those most resistant to such action; and 6) creates environmental responses which can mitigate risk among those individuals .

Various scholars have approached the study of foreign policy analysis from a variety of different frameworks. Some stress the cross cutting cleavages imposed by different levels of analysis (Starr, 2006), while others emphasize the importance of examining different topic areas (Hill, 2003) or countries (Beasley et al., 2001). Some authors acknowledge the critical impact of culture on outcome (Hudson, 2006), while others point to the decisive influence provided by leaders (Breuning, 2007). Yet, the vast majority of models in security studies have traditionally stressed the importance of states and institutions to the relative neglect, if not outright dismissal, of the individual level of analysis. Indeed, the vast majority of these models essentially ignore the variance in individuals' personal attributes, including that of leaders, arguing that structural incentives provide sufficient explanation for state behavior (e.g. Waltz, 1979). From these higher order levels of analysis, the source of security threats rest on assessments of structural and objective indicators, such as an adversary's military prowess, the nature of its political and economic institutions, or assumptions regarding the opponent's intentions. Each of these perspectives has added important contributions to our understanding of the various factors which determine foreign policy design, implementation and analysis.

However, while the focus of study in political violence has historically concentrated on the state, such state centered approaches have become increasingly obsolete since the end of the cold war. Events in the last few decades repeatedly demonstrate that this emphasis must shift to concentrate on individual actors. And if we direct our attention to an investigation of particular individuals, we must begin to account

for individual differences among and between populations if we are to explain and predict differences in individual predilections to engage in political violence.

The events of 9/11 provided a tragic illustration of the new challenges confronting policy makers from non-state actors. Influential actors operate outside the system of sovereign states, and present threats that are impossible to predict from within the rigid confines of traditional state-centered perspectives. Indeed, the greatest limitation of such traditional models lies in their myopic focus on the environment in which states operate, or a narrowed focus on aspects of the state itself, thereby ignoring the powerful and independent roles that individuals play in shaping the nature of international politics (Byman & Pollack, 2001).

One of the increasingly critical features in all aspects of foreign policy analysis is the focus on the individual actor, whether it is Osama bin Laden, Hosni Mubarak, or Muammar Quaddafi, and the importance of personal agency in explaining important variations in international outcomes. As Hudson (2005: 1): writes, “(f)oreign policy analysis is characterized by an actor-specific focus, based upon the argument that all that occurs between nations and across nations is grounded in human decision makers acting singly or in groups.” And traditional state centered theories often provide little help in seeking to understand the actions or motivation of many aspects of foreign policy which are clearly driven by individual goals and incentives.

The terrorist attacks on the United States on September 11, 2001 precipitated a renewed focus on the importance of individual non-state actors in changing world events. Rather than the previously expected clash between superpowers, a few determined individual actors crippled the world’s hegemon over the course of a few hours, with

effects arguably more lasting and damaging than any other attack upon the US in the last half century. Indeed, this very event precipitated a series of high level decisions in American foreign policy which set the path for the United States to engage in two major wars which have lasted the better part of a decade, precipitated severe and enduring economic consequences and which show little signs of either victory or withdrawal. Prior to 9/11, the notion that the next war would be one fought against individual actors would have seemed ludicrous, as would the prospect that the United States would face the kind of economic ruin as a result of extreme defense spending which we precipitated among the Soviets in the 1980s and 1990s. Yet today, testimony from the Department of Defense before the House of Representatives declares that the US is “waging a war against individuals” (Feldman 2006). One of the problems with this challenge, of course, lies in the fact that without a state, victory becomes ephemeral because there is no one to surrender for all actors, and as long as a single person with the intention and means of causing damage survives, the conflict will continue.

Strategic and tactical operations have changed to meet threats from the “individual”, yet the methods for understanding and properly characterizing individuals who engage in political violence in current scholarship have not yet caught up, despite urgent need: “...the same U.S. federal interagency report that documents the significant increase in funding for combating terrorism and reviews plans and activities by dozens of civil and military agencies reveals scant evidence of serious effort or funding to understand why individuals become, or to prevent individuals from becoming, terrorists in the first place” (Atran 2004).

We argue this is true because the vast majority of foreign policy analyses which acknowledge the importance of individuals in determining significant outcomes rely on purely social and environmental factors, or anecdotal evidence. In seeking to address this problem in a more systematic manner, we move from an investigation centered around state actors to one focused on variations in individual behavior which account for the combination of social, cultural, environmental, psychological and biological differences.

Technological and scientific advances in the last century have given researchers the ability to explore endogenous influences on behavior. The use of genetic, neurological, and neurochemical analyses have led to numerous psychological, medical and behavioral breakthroughs. Despite the valuable information gained however, none of the current methods in foreign policy carefully interrogate the endogenous underlying bases for individual differences in behavior. This is unfortunate because there is little question that complexities of human individual behavior cannot be fully understood without exploration into all forms of individual variance, including biological, neurological and genetic mechanisms (Kendler and Eaves 2005). This is true for political actors as well (Fowler and Schreiber 2008). Recent advances in the study of political and social behaviors allow for the opportunity to begin rectifying this lacuna in the foreign policy literature (for a review see Hatemi et al 2011).

Our proposed approach to the study of political violence requires the integration of methods and skills from geneticists and neuroscientists with those in the behavioral and social sciences. Specifically we seek to introduce an approach to study political violence which : 1) quantifies the effects of genes, environments, and their interaction on behavior that is a threat to national security; 2) identifies specific genetic and

environmental contexts that lead to such behavior; 3) develops a comprehensive model of the biological and social pathways to political violence; 4) identifies populations under special circumstances which pose a higher or lower prevalence for genetic, neurobiological or environmental mechanisms which pose an increased liability for political violence; 5) develops mechanisms to identify individuals within given populations who are most at risk for committing violence, as well as those most resistant to such action; and 6) creates environmental responses which can mitigate risk among predisposed individuals. Below we provide the details of supporting studies to warrant such an approach as the basis of developing a more comprehensive model for understanding the biological and environmental pathways that precipitate political violence.

#### Advocating for an Individual Approach to the Study of Political Violence

We introduce a powerful approach to the study of individual action in foreign policy analysis, which has a proven record in other behavioral domains. The approach combines genetic and biological analyses of individuals in the context of environmental triggers. Doing so provides a more complete picture of the causes and consequences of political violence in a world increasingly affected by individual actions and initiatives. This focus on individual behavioral variation is built upon well-developed models in psychiatric genetics developed to uncover those forces in the social environment that trigger predisposed individuals to act in distinct and predictable ways. Focusing on individual differences interacting with certain environments allows us to develop new tests to evaluate, assess, and screen threats that nation-states are likely to face in the future, and offer new suggestions for how best to prevent or mediate them. Certain individuals may

prove more prone to successful recruitment into extremist fundamentalist groups, more likely to resort to political violence, and less able to resist the appeal of violent fundamentalist organizations than others given particular exacerbating or ameliorating conditions. Seeking to identify those most susceptible to commit political violence, when triggered, offers the possibility for more effective, targeted, programs to help mediate those very environmental triggers which prove most threatening for those at higher risk of committing violence. In this way, we provide a unique approach for understanding individual variation in motivation for engaging in, and responding to, political violence.

In order to explicate the nature of these processes more systematically, we proceed by describing some of the existing literature designed to explain the sources of political violence, highlighting some of its remaining limitations. Next we introduce techniques drawn from behavioral genetics and describe how these models might apply to behaviors and traits relevant to the study of political violence. We then apply findings derived from earlier work on precursors to violence to suggest how interacting genes with particular environmental triggers might help scholars better identify the propensity for violence and distribution of risk within and across various populations. We hope that this approach might prove useful in helping to generate more effective prevention and protection strategies.

### *Benefits of a Biological Approach*

Individual differences do not exist in a social, political or cultural vacuum. Rather, culture infuses and imbues meaning and purpose into the dispositions inherent within given individuals, helping to precipitate different behavior among similar individuals who develop in different environments. But biology also contributes to such

variance as well, and such an understanding needs to be incorporated into any comprehensive model designed to explicate the sources of individual variance in proclivities for engaging in political violence. After all, why do only a small fraction of individuals residing in repressive political contexts engage in campaigns of terror to bring about their desired political changes?

A great deal is known about the environmental conditions that determine various aspects of foreign policy. Still, no matter how comprehensive, explanatory models which fail to incorporate endogenous motivations for individual action inevitably account for only a fraction of the total variance of human behavior, suggesting the strong possibility that environmental determinants of political violence do not tell the entire story (e.g., Post, 2005). At a broader societal level, within a given population faced with the same environmental stressors, how is it that only a handful of individuals can jump on a grenade to save their comrades? Join an underground army to fight for their beliefs? Strap a bomb to themselves? Or, run a plane into a building? Thus, in spite of the obvious resentment among the 1.3 million inhabitants of the Gaza strip, only a very few actually engage in acts of terrorism. Of approximately 9,000 “Arab Terrorists” detained by Israeli security forces in Judea and Samaria, less than 400 were deemed to be potential suicide bombers. Of the approximately 800,000 Catholic residents of Northern Ireland, Sinn Fein commanded as much as 100,000 votes among Nationalists, but perhaps as few as 750 were active IRA soldiers. If the focus remains on the environment, then what is it that differentiates those who take violent action from those who do not among individuals who reside within the same environment?

A primary advantage of a behavior genetic approach to the study of important topics in foreign policy analysis, such as terrorist motivation and action, lies in its complementarity to other extant approaches. Rather than competing with existing approaches, a neurobiological perspective adds an additional dimension to the explanatory capacity of existing environment-only models. Such a neurobiological approach can help leaders and states develop novel ways to limit the development or recruitment of terrorists, or other violent political activists by helping to more accurately identify those individuals who pose the greatest risk or uncover the environmental conditions which offer the greatest protection against such action.

Three critical features would help define such a behavioral genetic model as applied to political violence. First, it takes a large number of genetic, neurobiological and environmental factors to create behavior. Second, specific high risk environments have a stronger effect on individuals more genetically sensitive to specific triggers. And last, specific individuals at high genetic and social risk tend to self-select into environments that reinforce their specific vulnerability. No one person is genetically predisposed to be violent, nor is any one environment going to elicit violence 100% of the time. Rather, the behavioral genetic approach locates causality at precisely the intersection of individuals' unique genetic predispositions and their specific social and environmental contexts.

Through this approach, we offer a novel perspective on the role of individual differences in precipitating political violence. We suggest that scholars who wish to entertain a more comprehensive and accurate approach to understanding the precipitants of political violence would be well served by incorporating endogenous factors into their

models of political leadership, behavior and action. Our main argument incorporates three major elements. First, political violence cannot be fully understood if we ignore individual differences between people embedded within cultures. Second, locating the sources of individual variance in willingness to harm others is especially important because the roots of political violence are multifactorial; they result from interactions between a large number of biological (genetic) and social (environmental) factors. Third, these forces might interact in ways which may differ profoundly within and between populations.

### *Past Approaches to Political Violence*

In the traditional political science and policymaking literature surrounding the origins of political violence, three sets of arguments have been most commonly put forth. The first typically relate to divisions associated with the allocation of financial and economic resources and their distribution across society (Barber, 1996; Friedman, 2000). These arguments often mix with, overlap, and engage in rich dialogue with those which speak about the clash of cultures and civilizations which can also highlight and further societal and political fractures between rich and poor, Christian and Muslim, democrat and autocrat (Huntington, 1996; Fukuyama, 1996). While such arguments certainly help locate the social stage upon which environmental triggers make their play, they do little to help explain the reasons why individual actors differ in the way they play their scenes within the confines of the set onto which they are born.

The third set of arguments focus on the role of the individual in precipitating political violence. This scholarship sought to find the source of destructive outcomes in dysfunction origins and suggested that political violence remains rooted in individual

psychopathology. While this approach considers individual differences, it has remained restricted by a vision refracted through the prism of abnormal psychology and psychopathology, rather than expanded by a conception of the desire or willingness to resort to political violence as part of a continuum of normal human aggressive action and behavior in response to environmental triggers. For example Post (1998) argues that terrorists suffer from particular personality disorders, and their particular psychology drives them to commit terrorist acts. Post argued that individuals with particular personality characteristics were more likely to be drawn to terrorist groups as well. However, this view has been challenged by findings that most terrorists are in fact normal and that there is no such thing as a particular personality type which characterizes terrorists. Crenshaw (1981) concluded that “the outstanding common characteristic of terrorists is their normality.” Separate studies involving Palestinian suicide bombers, and members of the Irish Republican Army, the Algerian National Liberation Front (FLN), West German terrorists, and Italian terrorists, found that those who engage in terrorist acts are not mentally ill, emotionally disturbed, or depressed, but rather exist in the normal range for most typical DSM conditions (Rasch, 1979; Jager, Schmidtchen, and Sullwold, 1981; Segal 1987; Sullwold, 1981; Jager, 1981; Ferracuti and Bruno, 1981; Heskin, 1980 see Heskin 1984).

Moreover, insofar as more recent Middle Eastern terrorism is concerned, despite public sentiment that suicide terrorists are “crazed cowards bent on senseless destruction” no recognizable psychopathology is present (Atran’s 2003). On average, they are similar in education level and economic class to the general population. Ruby (2003) concludes that “...terrorists are not dysfunctional or pathological; rather, terrorism is basically

another form of politically motivated violence that is perpetrated by rational, lucid people who have valid motives.”

Indeed, one of the best arguments for the relative normality of terrorists lies in the efficacy of their action; severely mentally ill people are not sufficiently well organized, disciplined and controlled to plan and conduct large scale actions such as devastating terrorist acts require. Organized actors typically cannot accomplish these things by relying on psychotics. As Atran argues, “Recruiters for groups sponsoring terrorism acts tell researchers that volunteers are beating down the doors to join. This allows terrorist agents to choose recruits who are intelligent, psychologically balanced, and socially poised. Candidates who want mostly virgins in paradise ... are weeded out. Those selected show patience and the ability to plan in subtle, quiet ways that don't draw attention. Al Qaeda, especially, is rarely in a hurry. It can wait years and then strike when least expected.”

From an objective view, it makes sense that most terrorists should appear normal along personality dimensions which are consistent across cultures, and such individuals should remain fairly normally distributed within populations. After all, in order to become an effective terrorist, a person must retain at least a semblance of an ability to engage in efficacious, organized action, or his or her terrorist activities would only meet with failure. Moreover, psychopathologies, such as anti-social disorders, schizophrenia, and other personality *disorders* affect only a small portion of the population, and are present in all societies. Although such individuals may engage in maladaptive behaviors or behaviors inconsistent with societal norms, such as extreme violence, such behaviors

are rarely predictable or strategic in nature among such individuals, thus defining their behavior as abnormal in any context.

Thus, although some scholars have attempted to locate vulnerability for terrorist action in psychological disorders, and others in normality, neither model adequately interrogates the source of individual variance in such action. After all, if terrorists emerge unduly from particular segments of the population, it would be worthwhile to understand further the ways in which they diverge from the normal population. On the other hand, if they in fact appear psychologically inseparable from the broader population as defined by traditional normal and abnormal measures, it remains critical to determine which forces precipitate such action, since most people do not engage in terrorist activity. Yet despite these divergent approaches to understanding individual variance in terrorist proclivity, to date there has not been any empirical investigation of the genetic, biological or neurological differences that may exist among and between individuals prone to engage in terrorist acts and those who refrain from such activity even under identical environmental pressures. Models drawn from behavior genetics offer an ideal avenue of inquiry to pursue this investigation.

#### *The Behavior-Genetic Approach*

The field of behavior genetics has been perhaps most involved in the exploration of endogenous sources of individual differences and human behavior. For readers unfamiliar with the behavior-genetic paradigm, we outline the elements briefly here. The basic issues are discussed in far greater detail, with examples, by Eaves et al. (2005), Kendler (2005), Carey (2003), and Neale and Cardon (1992).

The underlying foundation of the research program engages both endogenous and exogenous factors and explicitly acknowledges that genes operate in conjunction with environmental factors during human development (i.e., from infancy to maturity). It is critical to be explicit that genes do not “determine” any behavior or trait. Rather, in simple terms, they provide the platform for the synthesis of proteins which then trigger a series of chemical processes which have neurological, cognitive and emotive implications among others effects, dependent on environmental cues. These neurobiological changes then inform and influence behavior in interaction with environmental stimuli. While genes are static, gene expression is not, and depends on the behavior of the individual, the environments the individual is exposed to, and the interaction with others (Johnston and Edwards 2002). For example, dopamine appears critically involved in such behaviors as aggression, novelty seeking and reward dependence. Certain dopamine genotypes lead to higher or lower levels of dopamine uptake and regulation. Yet children with the exact same variant on a dopamine gene, can also manifest different levels of dopamine influenced behavioral aggression in a given modern context depending on whether or not they received adequately sensitive parenting and external social support during critical formative years . In this way, their gene expression or regulation of dopamine can be altered during childhood by sensitive parenting, but interacts with modern circumstance to guide behavior. Individuals who did not receive such care display more problem behaviors, including higher rates of attention deficit disorder, while those who are fortunate enough to receive attentive care show lower levels of such behavior than children without that particular variant of the gene (Belsky et al., 2007).

To reiterate, the behavior-genetic paradigm does *not* find if “something is genetic”. Rather, the individual is a responsible agent for his/her behavior, yet this approach also remains cognizant of the reality that ignoring *a priori* one critical source of individual differences, genetic variation, may have grave consequences for our ability to model variance in individual behavior once certain processes are triggered. If, for example, political violence bears any similarity to other kinds of extreme behavior, including violence (Volavka, 1999), anti-social behavior (Lyons et al, 1995) , aggression (Chen et al, 2005; Filley, 2001), deviance (Booth and Asgood 1993), conduct disorder (Eaves et al, 1997; Foley et al, 2004), and substance use disorders, understanding the role of genetic differences may be critical to understanding why one individual is more likely to become a suicide bomber in the face of foreign occupation while another is not.

#### *Potential Genetic Precursors to Political Violence*

The vast range of behavior-genetic studies demonstrates that to understand fully human behaviors we cannot ignore genes any more than we can ignore a person’s environment. As noted above, this certainly holds true for violence. Treating genes and environment in isolation from one another overlooks essential characteristics of human behavior. Human development results from a conversation between genes and the environment that modifies the expression of genes and shapes the environment in which development occurs. Humans have a remarkable facility to create, evaluate, and modify their environment through extended parental care and familial cohabitation and other complex social groups. Such reciprocal effects generate relationships between genetic and environmental influences that are frequently referred to as “gene-environment interplay.”

Susceptibility to political violence can be conceived in terms using models and techniques from the well established study of behavior genetics, just like any other psychological trait. However for political violence, the “line” of what exactly constitutes problematic behavior changes with the times and power structures within the international community. For example, throwing rocks at American soldiers in the United States would be considered extremely deviant behavior. However, Palestinians doing the same thing to Israeli soldiers in the West Bank is far more common. Therefore, careful operationalization of the operative concept remains crucial in undertaking any such study of “political violence.

Individuals clearly differ in their vulnerability or propensity to engage in political violence. The greater a person’s liability, the more likely they are to engage in violent behavior.

**Figure 1: Interaction of Genes and Environment in Risk to Political Violence  $P\{V\}$ .**

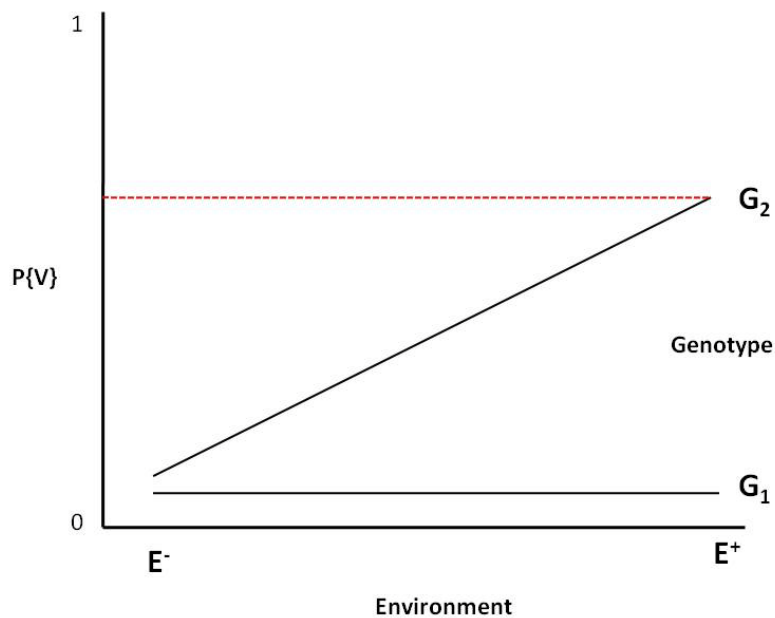


Figure 1 represents two hypothetical individuals with differing genetic constitutions. In the absence of exposure to a specific environmental “trigger” (such as growing up in the Gaza strip) ( $E^-$ ) both individuals ( $G_1$  and  $G_2$ ) have a low probability of performing an act of terrorism ( $P\{V\}$ ). However, when the environment is changed ( $E^+$ ), on average one of the two “genotypes” ( $G_2$ ) responds with a higher probability of violence.

The general notion that there are observable threshold effects in the interactive relationship between an individual’s genetic makeup and their social environment enjoys strong support in existing genetic models of violence, aggression, and other precursors to political violence . Three decades of research investigating individual risk for complex psychological and psychiatric traits of body and mind suggests that no one “gene” or single “environmental” factor holds the key to understanding behavior. Rather, unique configurations of many genes and environmental factors are necessary to understand behavioral differences.

Space constraints prevent us from reviewing every relevant study which might provide novel approaches or allow us to gain a better of understanding of the genetic and neurobiological influences which might be involved in precipitating political violence (see Appendix 1 for a summary table). However, we highlight the relevance and significance of a few important studies below to provide some illustrations of how work in this area might proceed . Hormones in the body help regular cognition and emotion. Overall, it appears that genes in the glutamate family and in the adrenergic systems, including some which influence the dopaminergic and serotonergic systems, are implicated in the expression of behavioral offensive and impulsive aggression (Brodkin et al. 2002; Brown et al. 1982; Chen et al 2005; Placidi et al 2001).

In addition, serotonin, while more commonly known for the treatment of depression, is centrally involved in an enormous number of important processes, especially related to mood, metabolism and memory (Davidge et al. 2004; Jensen et al. 2009). In particular, serotonin has long been believed to be involved in aggressive forms of violence and aggression; for example, variance in serotonin activity accounted for 5% of the divergence in violent outcomes in criminality (Retz et al., 2004). However, it is important to note that for any gene, hormone, or neurobiological mechanism, the relationship is not a simple linear casual pathway. That is, decreased serotonin does not equal increased violence (Olivier & van Oorschot, 2005). Rather, complex genetic *and* myriad environmental factors influence the relationship between serotonin, environmental triggers, and the extent, duration and lethality of aggressive violence.

De Boer et al. (2009, 52) presents one of the most compelling examples of the relationship between genes and environment in explaining the emergence of violence, and how violence and aggression are regulated through genetic systems. These processes differ between individuals, and are also affected in different ways by specific environmental stressors, which can leads to different behavior in those with the same genotype, or even within the same person over time acting under different circumstances. This study showed that mice with certain kinds of serotonin receptors appear much more likely to react aggressively after being exposed to violence in their environment. Thus, based on genotype, mice differed in their behavior; mice with a particular genotypic variant on their serotonin receptors responded aggressively more often and more quickly, and were more persistent, indiscriminating and more likely to injure others. Simply put, these mice were more prepared to learn aggressive behavior once they witnessed it,

versus others mice who were not so genetically predisposed. Like those who themselves have been the victim of child sexual assault, or witnessed domestic violence in their own home are more likely to become abusers themselves, some victims go on to become perpetrators while others do not; some of this difference in response may be attributable to genotypic variance among individuals, just like in the mice. Such a model could easily be juxtaposed to larger human environments as well. In conditions of relative peace, individuals more susceptible to decreased serotonin and dopamine activity may appear similar to everyone else. However, place those individuals under stress in an environment prone to violence such as Iraq, the West Bank or Gaza strip and they are most at risk to engage in violence themselves, due to the combination of the overwhelming stressful and violent world they are living in and their own genetic liability.

Human studies have also found similar relationships between aggression and serotonin regulation (Brown et al., 1982). Overall, individuals with a low activity form of serotonin manifest increased impulsivity, explosive violence, and higher levels of testosterone (Virkkunen et al, 1994). For example, Placidi et al. (2001) found that those with a lifetime history of higher rates of aggression had lower levels of serotonin. Interestingly, they noted a complex relationship between the lethality of aggressive and suicide attempts and serotonin levels, such that high lethality attempters showed significantly lower levels of serotonin than those who had exhibited lower lethality attempts, who did not differ in their levels from normal controls.

Reif et al. (2007) further elucidated the relationships between specific genetic variants and aggressive behavior by finding a relationship between adverse childhood events and other parts of the serotonergic pathway. First, polymorphisms in monoamine

oxidase (MAOA) and serotonin (5-HTT) transmitters were found to correlate with violent behavior. However, adverse childhood events and the presence of MAOA exerted independent effects on its emergence. People with MAOA who had nurturing childhoods and relatively stable lives can be peaceful, as can those without MAOA who have stressful lives. However, each alone could precipitate violence as well under varying levels of provocation, and the combination proved particularly likely to precipitate violence. The influence of the MAOA variant only erupted in later life violence, but required the prior contribution of adverse childhood events in order to manifest. In addition to MAOA also precipitating behavioral aggression under conditions of provocation (McDermott et al., 2009), higher levels of testosterone (Booth and Mazur, 1998; Johnson et al., 2006) also appear highly predictive of the propensity for aggression in both individuals and across populations.

Other work has suggested a role for dopamine pathways in precipitating violence (Chen et al 2005), as well as other aspects of cognition, including planning, as well as reward and novelty seeking (Backman et al. 2000; Cropley et al. 2006; Noble et al. 1998; Reeves et al. 2005), two behaviors related to impulsive violence under certain conditions (Hess et al 2009). Dopamine is particularly noteworthy not only because of its relationship to aggression levels, but also because genetic variants of critical dopamine receptors differ markedly in systematic ways across population groups by region (Harpending and Cochran 2002).

#### *Applying a Behavioral Genetic Model to Political Violence*

The literature discussed above suggests three critical ways in which behavioral genetic approaches may inform our understanding of variation in individual levels of

political violence. The first is to help observers better ascertain those most likely to engage in political violence under specific environmental conditions within a given population. Second, if specific genetic markers, such as dopamine for example, are found to relate to certain types of violence under certain conditions, behavioral genetics might help identify the relative propensity per region for violence by population given a certain stimulus, such as a foreign occupation. That is, certain genetic and hormonal systems vary by population in systematic ways and given suitable environments, these differences may result in different percentages of a population engaging in violent activity. We recognize that accurately predicting these differences may not yet be possible, but it is worth considering at least at a theoretical level that defense and relief efforts may be best predicted by inclusion of population differences at both environmental and biological levels. Third, by being able to identify genetic variants relative to violent behavior under a given condition, we might also be able to develop strategies which might reduce the effects of genetic liability to engage in violence, and thereby reduce the overall potential for individuals to engage in violence.

#### *Identifying Individuals and Population Risk to Engage in Political Violence*

Different environments pose different levels of risk for the potentiating violence. We are under no pretense that particular individuals are genetically predisposed to violence. Rather, the global conditions create the stage to precipitate violent activity under particular circumstances. Rather, we suggest that certain environmental conditions, including but not exclusive to economic pressure, lack of opportunity, political strife, oppression, restrictive borders, the death of one's children, maddrassas, and other such propellants create the possibility to instigate any human reaction, including violence. Yet,

individuals differ remarkably in how they respond to those conditions. Overall, violence may increase, under such conditions, but not everyone engages in it, even though the most extreme circumstances would appear to invite such behavior. Genetic factors constitute unlikely proximate causes for political violence. However, they may contribute to placing individuals at increased risk for engaging in political violence when exposed to stressful and threatening environments. Huntington (1996), Friedman (2000) and others who focus on cultural clashes and economic stresses as potentiators of the probability of conflict at a population level are certainly correct. However, we can also investigate those social *and* biological factors which create the greatest likelihood for producing a conflagration of violence at the individual level when individual risks stew in a soup of rage and humiliation to generate an environmental powder keg.

One of the best ways to explore these dynamics is to examine previous hot spots in which the local environment remained synonymous with violence for decades, but individual family members are divergent in engaging in violent action. Once a place becomes safe in the wake of peace agreements, as for example has largely occurred in Northern Ireland, it becomes possible to examine different related individuals in the context of the same negative environment to see whether and how certain individuals are more prone to violence in response to the same precipitant, such as witnessing the violent death of a loved one at the hands of a political opponent.

This kind of investigation allows a novel form of methodological approach to provide prophylactic treatment of high risk populations, rather than merely wait to pay the costly price of a search for perpetrators after occasions of violence. Such an approach can thus allow scholars and policy makers to learn more from previous

experiences and engagements, and to use extended family designs, to help identify, and more effectively intervene, between experiences of oppression, brutalization, humiliation, or other triggers, and the incidence of political violence. This can be done, for instance, through a systematic examination of siblings who may have experienced the same precipitating event, such as watching a parent brutally murdered, whereby one brother took up arms against the offender, and another decided to work through peaceful channels for a resolution to overall levels of violence. A behavior genetics approach can leverage precisely the intersection of experience and biology to highlight the fluids which both inflame and extinguish the fires of foment. In this way, we propose using the same kind of methodological model used to explore complex social behavior such as the study of impulsive aggression in mice described above to further explore the context of political violence in humans among those at greatest environmental risk.

Through this process, we can determine whether certain genetic markers correlate more strongly with engaging in violence, just as dopamine levels impulsivity appear to correlate with risk and reward seeking. And, as noted with dopamine, the prevalence of some of these genetic markers can be shown to differ geographically across populations. Thus, if we find important markers which raise the risk for certain populations to react with higher frequency to environmental assaults with violence, this information can aid in national security policy expectations for social disobedience, upheaval and unrest, as well as prospects for non-violent versus violent proclivities to potentiate social change. These propensities can hold tremendous implications for national security and future foreign policy decision making.

*Can We Use Behavioral Genetic Designs to Identify Prevention Strategies?*

If political violence bears any similarity to other forms of aggression, then the potential to mitigate genetic vulnerability with environmental support is possible. Genetic influence is not destiny, any more than environmental influences are. Social treatment is quite effective for both genetic and environmental vulnerabilities. One such example of this is presented by Boomsma et al. (2000). Figure 2, reproduced from their study, shows that genetic influences for deviant behavior can be modified by the social environment. Specifically, childhood deviant behavior, including violence, has been found to be highly heritable in numerous studies. However, in Calvinist homes, no such finding exists. That is, there is no difference between a population of identical twin pairs (MZ), who are genetically similar, and a population of fraternal twin pair (DZ), who are as genetically similar as any non twin sibling pair, in deviant behavior. However in non-religious homes, a marked difference does exist between identical and fraternal twins in their susceptibility to deviant behavior. In other words, in homes with a strict Calvinist upbringing, genetic disposition does not affect the likelihood that the person will engage in deviant behavior. This suggests that environmental support can constrain or induce biological tendencies for violent behavior in general. Strong environmental socialization thus can shape, control and constrain the effect of genetic predisposition in ways that do not exist when such environmental forces are absent or diminished.

**Figure 2. Interaction between genes and environment in liability to complex behavior: the Effect of Strict Religious Upbringing on Expression of Genetic Differences in Behavioral Disinhibition**

Notes: MZ refers to monozygotic, or identical twins, and DZ, refers to dizygotic, or fraternal twins.

The analogy with the etiology of phenomenon such as political violence is clear. In an otherwise “benign” or “nurturing” environment, genetic differences in liability to political violence may be present but not expressed. However, in malign or non supportive environments, perhaps resulting from exposure to abuse or injustice, genetic differences are expressed. Indeed, such responses may even be adaptive from an evolutionary perspective because they motivate action which attempts to recalibrate the balance of power between actors. Once again, awareness of relative susceptibility to political violence according to both genetic *and* environmental factors can help identify and support those most at risk for developing violent tendencies before they manifest physical violence.

## Conclusions

Previous work has primarily examined only the social and environmental precursors to violence and ignored the source of what makes each individual unique. While the existing environmental work is critical, it leaves unexplained a large portion of the variance in determining the difference between those who will work peacefully through the political process, and those willing to take violent action to bring about the outcomes they desire when faced with the same environmental stressors. By focusing on individual differences in biological structures, both brain and genetic, in interaction with specific environmental triggers, we can obtain more accurate and detailed additional measures to evaluate both the relative risk for political violence within particular cultures as well as provide insight into the specific individuals most likely to actively participate in such violence.

We have provided a gene-environment (behavioral-genetic) approach to understanding the human factors underlying individual variation in motivation for engaging in adversarial and violent behavior such as terrorist action. Here we discuss terrorist action and political violence as an important aspect of human agency in foreign policy analysis, with important potential outcomes for international relations, but by no means imply that this factor is the only one that matters in foreign policy decision making or that it represents the only area of foreign policy analysis to which behavior genetic theories and methods can be applied. Rather, we argue that any exploration into individual differences, including leadership, elite behavior, bureaucratic, legislative or other arena could benefit from the method introduced here. The proposed model will allow us to examine the influence of endogenous human factors on individuals' behavior

and to predict which individuals are most likely to become adversaries under particular environmental circumstances.

A neurobiological approach should not lead observers to be any more pessimistic about the prospects for positive intervention than more traditional approaches which focus equally challenging environmental risk factors. In fact, these strategies overlap in their implications, suggesting various ways to influence environmental precipitants to reduce the risk of violence. However, a neurobiological approach can provide more effective targeting by identifying both those individuals at most risk for engaging in violence, but also those circumstances which are most likely to trigger such action in those who are most biologically vulnerable to it. In addition it may identify those environments which offer protective influences on those same genetic liabilities. A neurobiological approach places importance on developmental factors, such as nutrition and parenting, which can permanently affect the lifetime propensity for engaging in violent behavior. Children born during times of drought or famine, for example, or those who grow up in war zones or were exposed to radiation, may be permanently altered in ways not captured by approaches which focus on income, resources, or education. Some of these changes, such as anxiety, or those precipitated by radiation, can be genetically transmitted into the next generation. In addition, such individuals may then act to permanently change their environment, through migration or violence, in ways that might decisively influence the next generation's capacity to flourish. States which recognize the influence of such factors can work to mitigate these risks, not under the guise of minor actions of humanitarian intervention, but as major forces which can precipitate generational rises in violent action among those affected.

The political relevance of such an approach appears obvious and ubiquitous. Small differences in some basic biological features, such as baseline levels in certain hormones or genetic tendencies, might be used to inform our understanding of cross-cultural differences in basic behaviors, such as risk-taking, aggression or cooperation. We remain only the beginning of such work, but if we take these arguments seriously, the deeper foreign policy implications remain profound as well. If orphans, or children of women who have suffered from famine, can produce inter-generational effects in risk-propensity or the tendency to engage in political violence, based on in utero deprivations, or post natal caring, for example, then the political gains from imposing sanctions which might, for example, deny food aid can produce very long term deficits and exert potentially very long term political effects which run counter to the interests we are trying to pursue by imposing those restrictions. By using deprivation strategies to put pressure on leaders today, we may directly be reducing prospects for peace with future generations if they become biologically more prone to respond to risk at lower levels of provocation as a result. In such a way, states can destroy their chances for long term rational negotiation by pursuing short-term, and short-sighted, strategies which privilege immediate material incentives while ignoring the deeply enduring biological ramifications of various foreign policy strategies.

A neurobiological approach can help identify those individuals within civil society whose exposure to violence and innate sense of vengeance places them at higher risk for engaging in political violence themselves. And, by assessing individuals' innate propensity to engage in political violence, this approach also offers the prospect of developing a strategy to intervene in the sequence of individuals' exposure to extremist

violence and subsequent recruitment into fundamentalist extremist organizations. This includes the potential to identify processes which may be beneficial in preventing those individuals primed by pro-fundamentalist activities from actually taking violent action. The recruitment strategies of terrorist organizations are predicated on exploiting individual's vulnerabilities to engage in violent action against particular adversaries. Being able to identify individual differences that place certain people at greater risk for committing acts of violence constitutes a first step in designing effective intervention programs to counter extremist violence.

Introducing genetic differences into an understanding of the sources of foreign policy action in no way eliminates or undermines the importance of cultural or environmental context in explaining outcomes of importance. Indeed, only through extreme environmental conditions are genetic liabilities present. In addition, comparing the findings across divergent populations offers an opportunity for culture specific perspectives as well. Our approach places the analysis of individual genetic and biological attributes in the context of specific environmental triggers. Doing so provides a more complete picture of the causes for, and consequences of, individual willingness to engage in political violence in a world increasingly affected by non-state actors.

By focusing on individual differences in biological structures, both brain and genetic, in interaction with specific environmental triggers, we can obtain more accurate and detailed measures to evaluate both the relative risk for political violence within particular cultures as well as providing insight into the specific individuals most likely to be the purveyors of such violence. Developing tests to evaluate, assess and screen threats will facilitate identifying those individuals most prone to successful recruitment into extremist

fundamentalist groups, those most likely to deploy political violence if provoked, and importantly, those individuals most likely to resist, and work against, the appeal of violent fundamentalist organizations.

The implications of this approach for purposes of national defense remain striking and staggering. Conducting behavioral genetic study of political violence provides a unique approach to understanding the root causes of political violence propagated by individuals. This approach allows us to examine not only the causes of political violence, but also their consequences on a larger societal level by exploring the effects of violence on both the perpetrators and their victims. In addition, this perspective allows us to identify those individuals whose exposure to violent and traumatic events would make them more susceptible to post traumatic stress disorder, suicide, substance abuse, and violence against others. Such assessments can provide a filter between exposure and recruitment into fundamentalist extremist organizations whose purpose is designed to exploit and deploy individual vulnerabilities in service of violent actions. We view such fundamentalist actions as one, but not the only example of the larger class of phenomenon of political violence. Identifying individual differences which place certain people at risk for committing acts of violence, and thus inevitable places others at risk for sustaining injury, constitutes the first step in designing effective intervention programs to combat such action and effects.

Both policymakers and academics already have a pretty good idea of the pressures and environments that create the most political violence. A glance around the globe informs any thoughtful observer of the areas which have traditionally been most prone to violence, either because of resource pressures, governmental failures, or other factors.

We know how to identify these areas and regions. What we are not so good at knowing is which individuals within those regions are most likely to react to those environmental pressures and stresses with violence, as opposed to peaceful action. We are also not so good at identifying the environmental buffers that we might employ to reduce the liability of individuals engaging in such behavior. Being able to identify such individuals and environments can help target more effective and efficient interventions which can lower the risk for triggers to spark into violent flame.

## References

- Atran, Scott. 2003. Genesis of Suicide Terrorism. *Science* 299: 1534-1539.
- Atran, Scott. 2004. Mishandling Suicide Terrorism. *The Washington Quarterly* 27(3):67-90.
- Backman, L., N. Ginovart, R. Dixon, et al. 2000. Age-Related Cognitive Deficits Mediated by Changes in the Dopaminergic System. *American Journal of Psychiatry* 157: 635-37.
- Barber, Benjamin R. 1995. *Jihad vs. McWorld*. Hardcover: Crown.
- Beasley, Rayn, Kaarbo, Juliet, Lantis, Jeffrey & Snarr, Michael. 2001. *Foreign Policy in Comparative Perspective: Domestic and International Influences on State Behavior*. Washington, DC: CQ Press.
- Belsky, Jay, Bakermans-Kranenburg, Marian & van IJzendoorn, Marinus. 2007. For Better and For Worse: Differential Susceptibility to Environmental Influences. *Current Directions in Psychological Science* 16(6): 300-304.
- Boomsma, D.I. et al. 2000. Netherlands twin family study of anxious depression (NETSAD). *Twin Research* 3:323-334.
- Booth, Alan, and D. Wayne Osgood. 1993. The Influence of Testosterone on Deviance in Adulthood: Assessing and Explaining the Relationship. *Criminology* 31:93-117.
- Breuning, Marijke. 2007. *Foreign Policy Analysis: A Comparative Introduction*. New York: Palgrave Macmillan.
- Brown, G., Ebert, M., Goyer, P., Jimerson, D., Klein, W., Bunney, W., & Goodwin, F., 1982. Aggression, Suicide and Serotonin. *American Journal of Psychiatry* 139: 741-746.
- Bueno de Mesquita, Ethan. Conciliation, Counterterrorism, and Patterns of Terrorist Violence. International Organization, 2005, Winter, 145-176.
- Bueno de Mesquita, Ethan. 2005. The Quality of Terror. *American Journal of Political Science* 49: 515-530.
- Daniel L. Byman and Kenneth M. Pollack. 2001. Let Us Now Praise Great Men: Bringing the Statesman Back In. *International Security* 25(4):107-146
- Carey, Gregory. 2003. *Human Genetics for the Social Sciences*. Thousand Oaks, Calif.: Sage.

- Chen , Thomas, Blum, Kenneth, Matthews, David, Fisher, Larry, Schnautz, Nancy, Braverman, Eric, Schoolfield, John, Downs, Bernard & Comings, David. 2005. Are dopaminergic genes involved in a predisposition to pathological aggression? Hypothesizing the importance of super normal controls in psychiatricgenetic research of complex behavioral disorders. *Medical hypotheses* 65(4):703
- Crenshaw, Martha 1981. The causes of terrorism. *Comparative Politics*, 13(4):379-399.
- Cropley, V., M. Fujita, R. Innis, and P. Nathan. 2006. 'Molecular Imaging of the Dopaminergic System and its Association with Human Cognitive Function.*Biological Psychiatry* 59: 898–907.
- Davidge, K. M., L. Atkinson, L. Douglas, et al. 2004. Association of the Serotonin Transporter and 5HT1D Beta Receptor Genes with Extreme, Persistent and Pervasive Aggressive Behavior in Children. *Psychiatric Genetics* 14 (3): 143–46.
- De Boer, Sietse, Caramaschi, Doretta, Nataragan, Deepa & Koolhaas, Jaap. 2009. The Vicious Cycle Towards Violence: Focus on the Negative Feedback Mechanisms of Brain Serotonin Neurotransmission. *Frontiers in Behavioral Neuroscience* 3: 52.
- Eaves L, Silberg J, Maes H. 2005. Revisiting the children of twins: can they be used to resolve the environment effects of dyadic parental treatment on child behavior? *Twin Res* 8 (4): 283-290
- Eaves LJ, et al. 1997. Genetics and developmental psychopathology: The main effects of genes and environment on behavioral problems in the Virginia Twin Study of Adolescent Behavioral Development. *J Child Psychol Psychiatry* 38:965-980.
- Eaves, LJ et al. 2008. Social and Genetic Influences on Adolescent Religious Attitudes and Practices. *Social Forces* 86(4): 1621-1646.
- Eaves L, Silberg J, and Erkanli A. 2003. Resolving multiple epigenetic pathways to adolescent depression. *J. Child Psychol Psychiatry* 44(7):1006-14.
- Feldman, Lloyd. 2006. Office of the Secretary of Defense Testimony before the Subcommittee on Tactical Air and Land Warfare. Armed Services Committee United States House of Representatives. September 21.
- Filley, C. et al. 2001. Toward an Understanding of Violence: Neurobehavioral Aspects of Unwarranted Physical Aggression. *Neuropsychiatry, Neuropsychology & Behavioral Neurology*. 14(1):1-14.
- Foley, Debra L., Lindon J. Eaves et al 2004. Childhood Adversity, Monoamine Oxidase A Genotype, and Risk for Conduct Disorder. *Arch Gen Psychiatry* 61:738-744.
- Ferracuti, F., and Bruno, F. "Psychiatric Aspects of Terrorism in Italy." In I. L. Barak-Glantz and C. R. Huff (eds.), *The Mad, the Bad and the Different: Essays in Honor of Simon Dinitz*. Lexington, Mass.: Heath, 1981.
- Friedman, Thomas. 2000. *The Lexus and the Olive Tree: Understanding Globalization*. New York: Anchor.
- Fukuyama, Francis. 1992. *The End of History and the Last Man*. Free Press.
- Harpending, Harry & Cochran, Gregory. 2002. In Our Genes. *Proceedings of the National Academy of Sciences* 99(1): 10-12.
- Hatemi, PK, Dawes C, Frost-Keller, A, Settle, J, Verhulst B. 2012. "News from the Political Front: Genetics of Social Attitudes" *Biodemography* (forthcoming)
- Heskin, K. *Northern Ireland: A Psychological Analysis*. New York: Columbia University Press, 1980
- Hill, Christopher. 2003. *The Changing Politics of Foreign Policy*. New York: Palgrave Macmillan.

- Hudson, Valerie, 2005. "Foreign Policy Analysis: Actor-Specific Theory and the Ground of International Relations," *Foreign Policy Analysis* 1 (1): 1-30.
- Hudson, Valerie. 2006. *Foreign Policy Analysis: Classic and Contemporary Theory*. New York: Palgrave Macmillan.
- Huntington, Samuel P.. 1996 *The Clash of Civilizations and the Remaking of World Order*, New York, Simon & Schuster.
- Jager, H. "Die individuelle Dimension terroristischen Handelns: Annäherungen and Einzelfälle" [The individual dimension of terrorist actions: Approaches to individual cases.] In H. Jager, G. Schmidtchen, and L. Sullwood, *Analysen zum Terrorismus*. [Analysis of terrorism.] Vol. 2: *Lebenslauf-Analysen* [Biographical analysis]. Opladen: Westdeutscher Verlag, 1981.
- Jager, H., Schmidtchen, G. and Sullwold, L. *Analysen zum Terrorismus* [Analysis of terrorism]. Vol. 2: *Lebenslauf-Analysen* [Biographical analysis]. Opladen: Westdeutscher Verlag, 1981.
- Jencks, Christopher, et al. 1972. *Inequality: A Reassessment of the Effect of Family and Schooling in America*. New York: Basic Books.
- Jensen, K. P., J. Covault, T. S. Conner, et al. 2009. A Common Polymorphism in Serotonin Receptor 1B Mrna Moderates Regulation by Mir-96 and Associates with Aggressive Human Behaviors. *Molecular Psychiatry* 14: 381–89.
- Johnson, D. McDermott, J. Cowden, E. Barrett, R. Wrangham, and S. Rosen. (July, 2006). Male Overconfidence and War. *Proceedings of the Royal Society of London B (Biological Science)*. 273: 2513-2520.
- Johnston, TD and L Edwards. 2002. Genes, interactions, and the development of behavior. *Psychological Review* 109:26–34.
- Kendler KS. 2005. A gene for...: the nature of gene action in psychiatric disorders. *Am J Psychiatry*.162(7):1243-52.
- Kendler KS and LJ Eaves. 2005. *Psychiatric Genetics*. American Psychiatric Publishing.
- Kendler KS et al 2007. Creating a social world: a developmental twin study of peer-group deviance. *Arch Gen Psychiatry* 64(8):958-65.
- Kruglanski, Arie W., Xiaoyan Chen, Mark Dechesne, Shira Fishman and Edward Orehek. 2009. Fully Committed: Suicide Bombers' Motivation and the Quest for Personal Significance. *Political Psychology*.
- Lyons, WR et al. 1995. Differential heritability of adult and juvenile antisocial traits. *Archives of General Psychiatry* 52:906-915.
- McDermott, R., Tingley, D., Cowden, J., Frazzetto, G. & Johnson, D. (2009). Monoamine Oxidase A gene (MAOA) predicts behavioral aggression following provocation. *Proceedings of the National Academy of Sciences*. 106 no. 7 2118-2123.
- Meyer-Linderberg, AM, Weinberger DR. 2006. Intermediate Phenotypes and genetic mechanisms of psychiatric disorders. *Nat Rev Neurosci* 7(10):818-27
- Mintz, Alex and Brule, David. 2009. "Methodological Issues in Studying Suicide Terrorism," *Political Psychology* 30 (1).
- Neale MC, and LL Cardon. 1992. *Methodology for genetic studies of twins and families*. Kluwer.
- Noble, E. P., T. Z. Ozkaragoz, T. L. Ritchie, et al. 1998. 'D2 and D4 Dopamine Receptor Polymorphisms and Personality. *American Journal of Medical Genetics* 81: 257–67.

- Olivier, Berend & van Oorschot, Ruud. 2005. 5-HT1B receptor and aggression: A review. *European Journal of Pharmacology* 526 (1-3): 207-217.
- Pape, R. 2005. *Dying to win: The strategic logic of suicide terrorism*. New York: Random House.
- Placidi, Giovanni, Oguerdo, Maria, Malone, Kevin, Huang, Ying-Yu, Ellis, Steven & Mann, John. 2001. Aggression, Suicide Attempts and Depression. *American Journal of Psychiatry* 50(10) 783-791.
- Post., J. 1998. Terrorist Psycho-Logic: Terrorist Behavior as a Consequence of Psychological Forces. In W. Reich & W. Laqueur (Ed). *Origins of Terrorism*. New York: Woodrow Wilson Center Press.
- Post, J. 2005. Psychological operations and counterterrorism. *Joint Forces Quarterly*, 105-110.
- Powell, Robert. 2007. Defending against Terrorist Attacks with Limited Resources. *American Political Science Review* 101:527-541.
- Rasch, W. "Psychological Dimensions of Political Terrorism in the Federal Republic of Germany." *International Journal of Law and Psychiatry*, 1979, 2, 79-85.
- Reeves, S., P. Grasby, R. Howard, et al. 2005. A Positron Emission Tomography (PET) Investigation of the Role of Striatal Dopamine (D2) Receptor Availability in Spatial Cognition. *NeuroImage* 28: 216-26.
- Reif, Andreas and 11 coauthors. 2007. Nature and nurture predispose to violent behavior: Serotonergic genes and adverse childhood environment. *Neuropharmacology* 32: 2375-2383.
- Retz, Wolfgang, Retz-Junginger, Petra, Supprian, Tillman, Thome, Johannes & Rosler, Michael. 2004. Association of serotonin transporter promoter gene polymorphism with violence. *Behavioral Sciences and the Law* 22(3): 415-425.
- Ruby, Charles L. 2003. Are Terrorists Mentally Deranged?. *Analyses of Social Issues and Public Policy* 2(1):15 - 26
- Silberg, Judy L. and Michael L. Rutter. 2001. Nature-nurture Interplay in the Risks Associated with Parental Mental Disorder. Pp. 13-36. *Children of Depressed Parents: Alternative Pathways to Risk for Psychopathology*. S. Goodman, editor. John Wiley & Sons.
- Starr, Harvey. 2006. *Approaches, Levels and Methods of Analysis in International Politics: Crossing Boundaries*. New York: Palgrave Macmillan.
- Sullwold, L. "Stationen in der Entwicklung von Terroristen: Psychologische Aspekte biographischer Daten" [Stages in the development of terrorists: Psychological aspects of biographical data]. In H. Jager, G. Schmidtchen, and L. Siillwold, *Analysen zum Terrorismus [Analysis of terrorism]*. Vol. 2.: Lebenslauf Analysen [Biographical analysis]. Opladen: Westdeutscher Verlag, 1981.
- Virkkunen, M., and 9 coauthors. 1994. CSF biochemistries, glucose metabolism and diurnal activity rhythms in alcoholic, violent offenders, firestarters and healthy volunteers. *Archives of General Psychiatry* 51(1): 20-27.
- Virkkunen, M., Goldman, D., Nielsen, D. & Lomoiiler, M. 1995. Low brain serotonin turnover (low CSF5-HIAA) and impulsive violence. *Journal of Psychiatry Neuroscience* 20(4): 271-275.
- Volavka. 1999. The Neurobiology of Violence: An Update. *The journal of neuropsychiatry and clinical neurosciences* 11(3):307

Waltz, Kenneth.. 1979. *Theory of International Politics*. New York: McGraw-Hill.

Appendix 1

Aggressive Behavior		diacylglycerol kinase (Dagk1) and the glutamate receptor AMPA3 (Gria3)	glutamate	GWAS	Brodkin et al. 2002. J Neurosci
Aggressive Behavior	propranolol to control aggressive behaviour	MHPG	Adrenergic	Clinical	Brown et al. 1982; Placidi et al. 2001).
Antisocial Behavior	maltreatment as children	MAOA, L	Adrenergic	Can	Caspi et al 2002. Science; Reif et al. 2007
Impulsive Agression		DBH	Adrenergic		Hess et al. (2009)
Impulsive Violence		L version of MAO-A		Can	
Impulsive Violence	Alcohol	CSF 5-HIAA concentrations, SLC6A4	serotonergic	Can	Vrikkunen et al 1995 J Psychiatry Neurosci.
Impulsive Violence	personality disorder	TPH/CSF 5-HIAA/5-HT,SLC6A4	serotonergic	Can	Virkkunen M, et al.: 1994; Nielsen et al. 1994). Manuck et al. (1999)
Offensive Agression	Rodents	5-HT1A and 5-HT1B	serotonergic	Can	Olivier and van Oorschot 2005
Pathological Aggression		D2 and Dat1	dopaminergic	Can	Chen et al 2005, medical hypos
Physical Violence		SLC6A4, 5-HTTLPR	serotonergic	Can	Retz et al 2004. Behavioral Sciences
Trained Violence	Mice	5 HT, SLC6A4	serotonergic	Can	de Boer et al 2009.Front Behav Neurosci.
Violent Behavior	schizoaffective patients	COMT	Adrenergic	Can	Strous RD, et al 1997
Violent Suicide		CSF 5-HIAA, SLC6A4	serotonergic	Can	Lachman et al 1998