Addiction professionals are frequently called upon to assess and treat individuals with histories of excessive alcohol and other drug use and interpersonal violence. The formula $P + D + E = V$ depicts the multiple factors that can interact to influence the risk of violence, but there is a high degree of variability in how these factors interact.

In the person-drug-environment interaction, many factors determine whether the threshold between an impulse to commit violence and the act of violence is crossed. Within the P component are such influences as developmental history, prior history of aggression, personality, current mental status, type and degree of social support, and values. The D factors vary by particular drug or drug combination, drug purity and dosage, method of ingestion, and the drug state (e.g., intoxication versus withdrawal). The E factors include the degree of social density, social expectations of drug effects, accessibility of potential victim(s), weapon accessibility, and violence-related social norms. As the unique elements within these three factors combine, they create one of seven potential effects on the risk of interpersonal violence. This article describes these seven potential relationships between substance use and violence and explores the clinical implications of each.

**Independent Effect**

In the first possible $P + D + E = V$ interaction, we have an act of violence by an individual who has consumed a drug, but the drug plays neither a promoting nor inhibiting role in the violent act. The failure to recognize the potential for such an independent effect is captured in comments like, "More than 80 percent of persons who commit acts of violence do so under the influence of alcohol or another drug." This statement implies that 80% of these acts were caused by drug impairment. Such pronouncements, by representing correlation as causation, make great propaganda but poor science and social policy.

There are also clinical implications that grow out of the failure to recognize these independent effects. If the addictions
professional assumes that a client’s battering of his or her intimate partner or a mother’s abuse of her child is a function of drug toxicity or radical personality change resulting from drug use, then the clinical corollary is that the violence will spontaneously remit with abstinence. But if the violence was unrelated to substance use, then the partner or the child could be at an even greater risk of victimization by a now sober and more physically competent perpetrator.

Clinical signs of violence as an independent behavior include violent acts that predated the onset of substance use, and a pattern of violence in which the timing, frequency and intensity of violent episodes are unrelated to drug choice, level of intoxication or stage of withdrawal. Where this is the case, violence needs to be understood as an independent, primary problem that requires concurrent treatment.

Rationalizing Effect

In this interaction, alcohol or other drug use plays no role in an act of violence but its real or feigned presence is used retrospectively to excuse aggressive behavior (“The devil/drug made me do it!”) Substances may even be consciously used to provide a post-assault escape from personal responsibility. Substance use does contribute to violence in cultures that permit this escape from accountability, but its contribution must be understood in symbolic and social terms, not in terms of psychopharmacology.

Two clinical implications emerge from an understanding of this potential interaction between substance use and violence. The first is an awareness by the addictions professional that he or she may have clients feigning a substance disorder to escape culpability for violent criminal conduct that had little or nothing to do with substance impairment. This pattern of “pseudoaddiction” is indicated by the marked absence of denial and minimization of substance use (in fact, quite the opposite), and a history of conduct disorder predating drug experimentation. Such clients initially comply but later escalate boundary testing and aggression within the treatment milieu.

Causative Effect

A third potential interaction between a person, drug and environment involves situations in which violence is elicited by the effects of a drug with little contribution from the person or the environment. This raises the question: Can a drug cause an individual to commit an act of violence where there is no pre-existing risk for such aggression? The answer is a qualified yes, with a recognition that such instances are rare. This category of substance-influenced violence includes toxic organic psychoses resulting from acute or chronic drug use that are accompanied by delusions of persecution and auditory or visual hallucinations. In these states, drug-induced errors in perception and cognition can dramatically increase the risk of violence even in persons without a history of, or innate propensity for, aggression. Conditions that are traditionally included within this category include toxic organic psychoses produced by alcohol (e.g., idiosyncratic intoxication, alcoholic hallucinosis), stimulants such as cocaine and methamphetamine, and excessive doses of hallucinogens. The addictions professional, particularly those working in emergency services, intake or detoxification units, should be aware of the symptom configurations of the major toxic organic psychoses associated with violence. Most of these conditions require careful clinical management to simultaneously protect the safety of the client, the service professional, and the public.

Additive Effect

The fourth potential relationship between substance use and violence is a common one. Here we have a person who has some innate risk for violence (that includes most of us) but who under most circumstances is able to suppress violent impulses. A drug could play an inciting role in moving this individual across the threshold
of a violent impulse to a violent behavior. When we speak of additive effect, it means that substance use contributes to rather than causes violence. It makes this contribution by lowering the point of reactivity through disinhibition and impairment of judgment. In this case, substance intoxication doesn’t create aggression; it simply lowers the threshold at which existing aggressive impulses are transformed into aggressive behavior.

The additive effect constitutes a significant portion of alcohol-related violence. This effect is particularly prominent when the following factors are combined: socioeconomic and psychological distress, high population density, high density of alcohol outlets, illicit and competing drug markets, and high density of handguns. Substances can generate a priming effect when an individual with high risk for aggression gets purposely “pumped” or “wired” to induce a chemical courage that intensifies the violent act.

One of the clinical implications of the additive effect involves the growing number of people being mandated to addiction treatment who do not meet traditional diagnostic criteria for addiction, but who will spend most of their lives in systems of punishment and control if they do not find a way to management this substance-violence interaction. Such individuals may be appropriately treated in addiction treatment programs if the program includes the following elements: special clinical tools to assess and treat individuals with a history of violent victimization and perpetration, personally relevant rationales for abstinence, and a wide menu of metaphors for the reconstruction of personal identity and interpersonal relationships. In an individual lacking the classic symptoms of alcoholism, the insight, “when I drink, I go to jail,” may be more transformative than the mantra “I have the disease of alcoholism.” Similarly, linking sobriety to the metaphors of “freedom” and “self-control” may be more meaningful for these clients than the metaphors of “powerlessness” and “surrender.”

Synergistic Effect

In our fifth possible interaction between substance use and violence, persons who combine exceptionally high risks for and past histories of violent perpetration are combined with high risk drug choices, dosages and methods of ingestion. This usually creates a risk and level of violence far exceeding the additive effect. What is produced instead is a multiplication effect that increases the risk and intensity (potential lethality) of violence.

Studies of persons who have committed multiple acts of violence, committed acts of violence against multiple targets, and committed acts of violence in patterns of self-accelerating frequency and intensity reveal a profile of this worst possible drug-person-environment interaction. This risk profile, which has emerged from the pioneering research of Dr. Lonnie Athens (see his The Creation of Dangerous Violent Criminals, 1992, and Violent Criminal Acts and Actors Revisited, 1997) and others, often includes the following developmental sequelae:

- Neurological trauma (e.g., prenatal drug exposure, birth complications, blunt trauma, malnutrition, infection/fever) as evidenced by a history of seizure disorders or cognitive/sensory impairments.
- Neurological adaptations (changes in epinephrine [adrenalin], noradrenaline, and serotonin levels) resulting from sustained stress/trauma and as evidenced by atypical physical/emotional reactivity to environmental stimuli, atypical pain tolerance, and use of drugs to achieve state regulation.
- Abandonment (the failure to bond/attach; absence of safety, consistency and continuity of care in a primary relationship; early loss of primary relationships; isolation and self-containment; emotional numbing; inability to trust) (See Ken Magid and Carole McKelvey’s High Risk: Children without Conscience, 1989).
• Brutalization (physical, sexual, emotional victimization) accompanied by multiple traumagenic factors (early onset of abuse, long duration of abuse, multiple perpetrators, perpetrators drawn from the family/kinship network, boundary invasive forms of abuse; failure to protect following disclosure).
• Horrification (exposure to the violent victimization of others).
• Violent coaching (transmission of the technology of violence via violent mentors, paramilitary subcultures or mass media).
• Violent style (onset and increased frequency and intensity of violence).
• Virulence (rigid self-encapsulation, predatory view of all interactions, preoccupation with power and control, aggression a core element of identity, high risk of lethality).

There are many early symptom clusters that predict this risk profile (enuresis, fire-setting, cruelty to animals) as well as protective factors that can interrupt this developmental trajectory (e.g., healthy surrogate families, non-violent coaching, early professional treatment). When this developmental trajectory is not interrupted, we have an individual “wired” for violence via:

• bias for action (as opposed to cognitive or emotional processing),
• atypical tolerance for pain,
• propensity to attribute hostile intent to others and misinterpret harmless cues as threatening,
• limited impulse control and problem solving abilities,
• impaired abilities to bring emotion under control once released,
• inability to initiate or sustain intimate relationships, and
• diminished capacity for empathy and remorse.

Collectively, such characteristics create a high risk for violence apart from any drug-related influences. A synergistic effect occurs when drugs are consumed whose dose and frequency of use, method and duration of use, and pharmacological effect inflate this already high risk (See Illustration).

Most addiction professionals, unless cross-trained via advanced studies in psychiatry or psychology, are not qualified to treat such individuals by themselves, but may be appropriately included in multidisciplinary teams assembled for this purpose.

Neutralizing Effect

The fact that certain drugs might work to lower the risk of violent assault has long been known in medicine. In persons whose aggressive and violent behavior is related to a primary psychiatric disturbance, a broad spectrum of psychotropic medications are available to bring these symptoms under control, increase the quality of their lives, and reduce their threat to others. Pharmacological interventions are similarly utilized to chemically inhibit those with histories of repeated sexual assaults. A question remains about the extent to which illicit drug use can reduce violence within particular individuals. Drugs that lower one’s threshold of sensory overload, lower anxiety, blunt emotional experience and lower psychomotor activity could produce such an effect.

In an individual who brings a high risk for violent assault, drugs could be ingested that could alter this degree of risk. Alcohol intoxication, for example, might elevate that risk (via disinhibition) while opiates and low-potency cannabis might inhibit violence. Someone with high risk for violence could exhibit no such behavior during periods of heroin intoxication, but might pose a great threat of violence during withdrawal or when drug free.

One clinical implication of this neutralizing effect can be seen in a client maintained on methadone who might pose little threat of violence while being
maintained, but whose threat could dramatically increase if the client were “administratively detoxed” due to rule infractions. The increased risk of violence should be a consideration in the clinical protocol governing such decisions. This does not imply that someone regularly using narcotics is incapable of committing an act of violence. It merely means that to do so, he or she must break through the narcotic shield that diminishes the likelihood of such action. In contrast, alcohol, at all but the highest doses, provides no such shield while lowering the reactivity threshold at which aggressive impulse turns into violent behavior. In a person at personal risk for violence, alcohol acts like gasoline on a fire. In the same person, narcotics are like taking fuel or oxygen from the fire.

Signs of this neutralizing effect include a pattern of violence that predates the onset of substance use, declines or disappears during periods of use, but reappears during periods of voluntary or imposed abstinence. Treatment alternatives under such circumstances include substituting a medically monitored licit substance for the less reliable illicit drug or, where possible, replacing the chemical shield against violence with a psychological shield.

**Contextual Effect**

To illustrate how the environment can influence the risk for violence, we will close this discussion with a brief review of how the E element of our formula can tip the scales toward violence. Contextual violence occurs when an act of violence emerges not from personal risk, pharmacology, or their interaction, but from a social environment in which violence is a learned way to achieve status and power and to resolve problems.

This effect is most intense in the marriage between drug and criminal cultures. The 1980s witnessed a significant increase in the number of people entering treatment from such cultures who had been socialized to view violence as an appropriate strategy to maintain, protect, or expand one’s territory within illegal drug markets. As that value spread, violence as both a strategic behavior and as a core dimension of personal character increased. The treatment of this pattern of violence focuses not on the drug but on removal of the individual from anti-social subcultures, the development of non-violent skills and values, and enmeshment of the individual in a pro-social culture of recovery.

The addictions professional faces many challenges in addressing the issue of violence. These challenges, which range from the clinical to the ethical and legal, all hinge on understanding the complex relationship between substance use and violence. This article has underscored the complexity of this relationship by describing seven potential interactions between individuals, psychoactive drugs, and the physical and social environments in which they are consumed.

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* This illustration is based on the work of Dr. L. Athens and incorporates additional work by K. Magid & C. McKelvey & W. White.