



Violence Is a Contagious Disease

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**Gary Slutkin, M.D.
The University of Illinois at Chicago**

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VIOLENCE IS A CONTAGIOUS DISEASE⁶

Gary Slutkin, M.D.

*The University of Illinois at Chicago
Cure Violence (formerly CeaseFire)*

Violence is a contagious disease. It meets the definitions of a disease and of being contagious—that is, violence is spread from one person to another. This paper will clarify (1) how violence is like infectious diseases historically by its natural history and by its behavior; (2) how violence specifically fits the basic infectious disease framework—and how we can use this framework to better understand what is known of the pathogenic processes of violence; and (3) how we can provide better guidance to future strategies for reducing violence, in order to get more predictable results, and develop a clearer path to putting violence into the past. This paper intends to clarify to the scientific and policy community, as well as the general public, how violence is acquired and biologically processed, and begins to outline how the spread of violence can be interrupted in short-term emergencies and longer term situations.

The Great Plagues and Violence

We begin by reminding ourselves that the great infectious diseases and violence have each killed tens to hundreds of millions of persons throughout history. Nothing else has caused this level of human fatalities. Yet, before we understood the causes of the great infectious diseases, that is, before discovering what was causing epidemics of leprosy, plague, tuberculosis, cholera, and other infectious diseases, we frequently treated the people affected as “bad people”; we blamed them for the problem, and in particular lamented their moral character. People with leprosy, plague, typhus, cholera, tuberculosis, and other maladies were frequently considered morally “bad,” suffering stigma at a minimum, and in many cases worse treatment, including being put in dungeons, burnt at the stake, or thrown down wells.

Why did we do this?

We did this because we did not know—did not *yet* know—what was really happening. *Why* we did not know was because the causes and

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underlying processes were *invisible*. Plague, for example, is due to an invisible microorganism, carried by a flea, otherwise living inside a rat.

Who knew?

It was not until very recently in human history, the 17th century, that Anton Leuwenhoek, a tradesman and scientist-to-be, invented the microscope and discovered these previously invisible microorganisms (De Kruif, 1926). Another 200 years passed before Louis Pasteur, a chemist working as a consultant for the beer, wine, and milk industries who wanted to know why these products spoil, discovered that Leuwenhoek's organism *did something*. It was then up to Robert Koch to definitively prove that these invisible creatures caused animal and then human disease, first anthrax and then tuberculosis, the latter the most highly feared killer of the time (Green et al., 1982). These massively important discoveries built on each other and led over the course of the next few decades to the identification of most of the infectious organisms that cause epidemic diseases. This then led, over just a few short decades of human history that followed, to *entirely new and rational strategies* for reducing the amount and impact of these historical major killers—strategies as varied as case finding and therapy for tuberculosis; immunization for polio; and environmental sanitation, better food handling, the use of toilets, and hand washing for diarrheal disease (Dowling, 1977; Nelson and Williams, 2007; Heymann, 2008). One historic killer, smallpox, has been totally eliminated by a global immunization strategy. Some of these strategies, for example, using impregnated bed nets for malaria, are still evolving and improving.

But before these discoveries, and a new understanding of the problem, humankind was *stuck*.

Misdiagnosis and Mistreatment

It now seems as if the problem of violence, like the great infectious diseases of the past, has been stuck—not because we do not care enough, nor because we do not have enough money devoted to it, but because *we have made the wrong diagnosis*. Wrong diagnoses, in particular moralistic diagnoses, usually lead to ineffective and even *counterproductive* treatments and control strategies. Problems of mankind frequently do get stuck, sometimes for decades or even for the history of man, commonly because we do not correctly understand the problem *scientifically*, a step that is *required* to design and implement *rational and effective control measures*. It also seems that, historically, moralistic views and solutions usually fill that gap in understanding.

Moralistic ideas actually have a very poor record of solving problems, in part because people differ in their interpretations of moralistic ideas, and in part because they lack an understanding of the actual biology of

the problem. Sometimes this is because of the fundamental attribution bias where we humans replace incomplete understanding with blame of others. As a result, people who have learned violence, as for those affected or infected with the great infectious diseases, have been misdiagnosed and mistreated. However, in 2012 we have more pieces of the puzzle. Violence can now be better understood scientifically, and as a result, there *must be a new strategy* to reduce and eliminate violence.

Scientific Understanding

Violence, for starters, is a phenomenon driven by the brain, as the brain regulates and controls behaviors. Like our previous lack of knowledge of infectious organisms, our knowledge of the invisible workings of the brain has also been a field in the dark (or dark ages). Recent discoveries, if brought together into a coherent framework, allow us to see that brain processes are in fact contagious too. If we can begin to draw on the fairly new research findings of social psychology (40 to 50 years old) and functional magnetic resonance imaging of the brain (15 to 20 years old), connect these findings with what is known from infectious disease epidemiology, and add the first studies of new therapeutic approaches—we can now define a new set of causations and strategies to *reduce violence more predictably*. Understanding epidemiology and invisible brain mechanisms will carry us farther out of the middle ages to new possibilities immediately available.

Infectious Diseases and Violence in Populations

There are three main characteristics of infectious diseases in *populations*: clustering, spread, and transmission. *Clustering* in space, or spatial grouping, is simple in concept and is characteristic of epidemic diseases. Clustering is shown in Figure II-1 for the infectious disease cholera in Bangladesh, and in Figure II-2 for violence in Chicago. *Spread* in epidemics is characteristically nonlinear. This may be one of the reasons why many researchers have difficulty attributing rises and falls to simple causative factors such as the economy or jobs. Nonlinear spread may occur as waves, frequently appearing as waves on top of waves. (This is characteristic of plague, smallpox, and many other infectious diseases; see Anderson and May, 1991, and as shown in Figure II-3 for the homicides in the United States over the past several decades.) This pattern of waves upon waves occurs because epidemics frequently consist of many epidemics, as spread itself diffuses and as contagious populations meet with new susceptible populations in new locations, and to be met with new provoking factors.

Another characteristic of spread in some circumstances is that seen from point source epidemics, sometimes exhibiting very rapid spread, as



FIGURE II-1 Clustering in cholera epidemic, Bangladesh.
SOURCE: Ruiz-Moreno et al., 2010.

shown in Figures II-4 (cholera) and II-5 (violence). In these cases, one initial infectious event may cause many subsequent cases (for cholera, precipitated by an infected water source in Somalia; for the Rwanda genocide, the killing of the Rwandan president). Secondary epidemic waves are seen in each of these figures. With cholera, the secondary wave occurred when a new group of “susceptibles,” in this case, refugees new to camp, became infected later. In Figure II-5, in this case, a violence/killing curve from Rwanda, the secondary wave similarly represents a new group of “*susceptibles*,” in this case, persons who were previously hiding and then were found and killed (Verwimp, 2004). The similarities of these patterns reflect similar contagious dynamics.

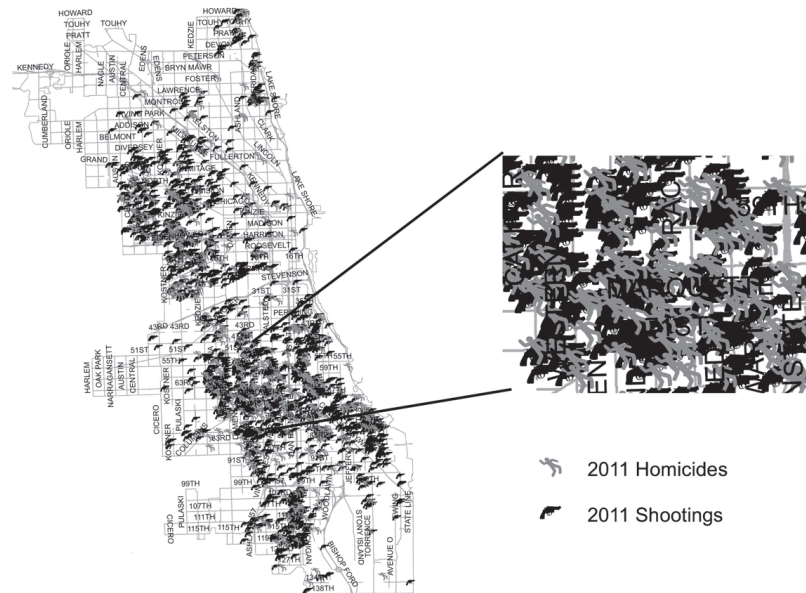


FIGURE II-2 Clustering in violence epidemic, Chicago.
 NOTE: This data was provided by and belongs to the Chicago Police Department. Any further use of this data must be approved by the Chicago Police Department. Points of view or opinions contained within this document are those of the author and do not necessarily represent the official position or policies of the Chicago Police Department.
 SOURCES: City of Chicago Data Portal.

Spread may be dramatic and rapid, or slow, depending on many factors. Rapid spread, well known for infectious diseases, is seen, for example, in foodborne outbreaks, flu, or severe acute respiratory syndrome (SARS). Rapid spread is seen in violence outbreaks such as gang wars, soccer riots, or the Rwanda genocide. Dramatically rapid recent outbreaks include the London and UK riots and even the “Arab Spring.” Slower spread may be seen in infectious disease outbreaks with longer incubation periods, such as tuberculosis or AIDS—showing spread over decades—analogueous to the spread of violence in U.S. cities that showed increases over decades.

Some acute-phase outbreaks are from *common* or *point source transmission*, as described above; while longer term outbreaks are more commonly a result of *person-to-person transmission*. The speed of transmission varies not only according to incubation periods of the infection, but also according to the number of persons susceptible and infected from a given source, as well as other factors. World War I was a violence outbreak with multiple features including multiple “point sources” as new countries “joined in.” The result: 15 to 20 million persons died in less than 4.5 years.

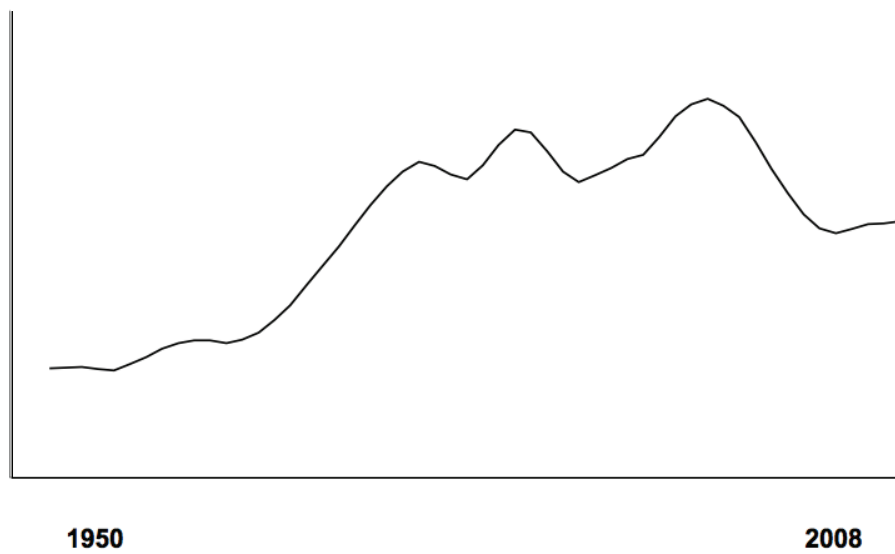


FIGURE II-3 Epidemic of killings in the United States, showing waves on top of waves.
SOURCES: BJS, 2005; FBI, 2008.

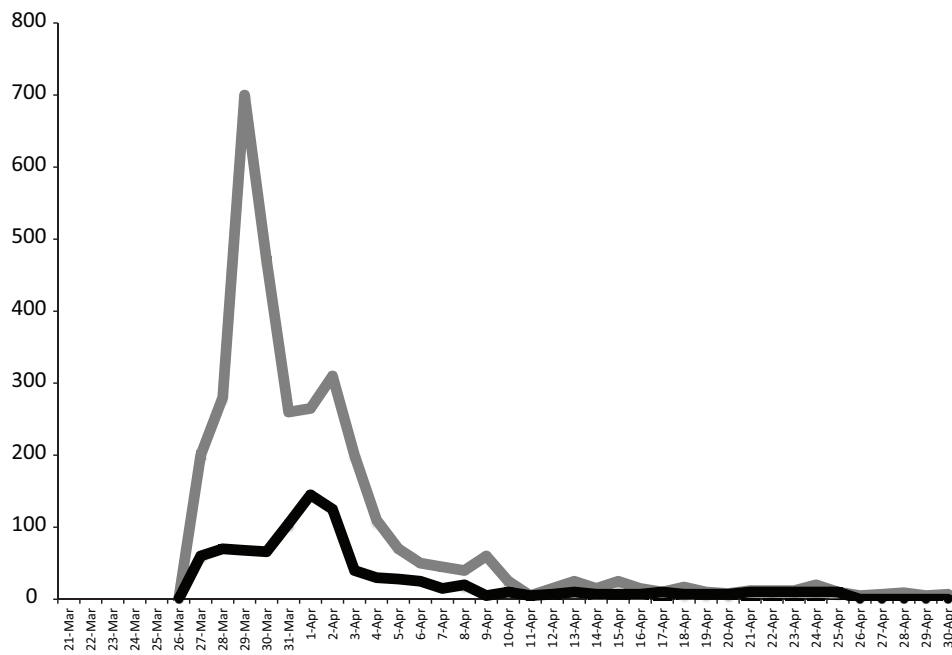


FIGURE II-4 Cholera—Gannet, Somalia.
SOURCE: Data from Farah, 1985, Figure 1.

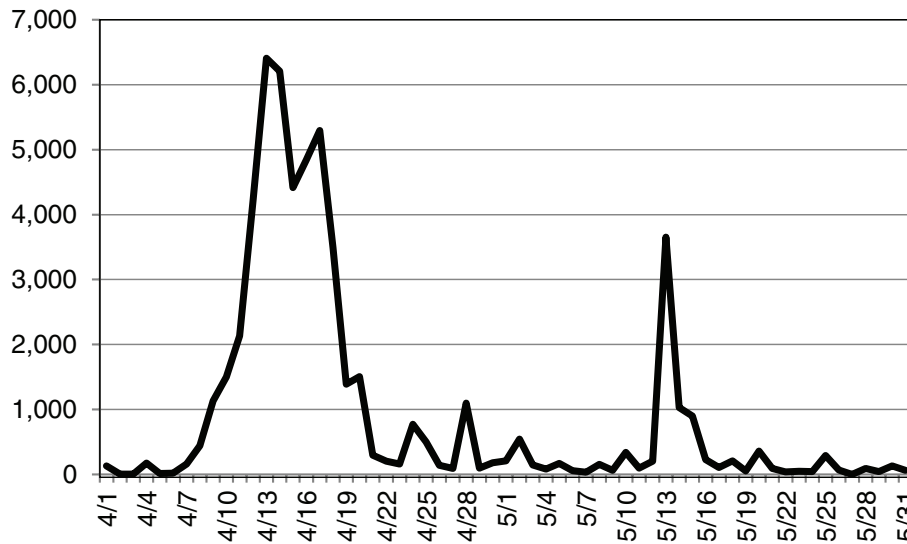


FIGURE II-5 Killings—Kibuya, Rwanda.
SOURCE: Data from Verwimp, 2004, Table 8.

Transmission is the passage of an infection (or other condition) from one organism to another. The classic infectious diseases are transmitted by invisible infectious agents (e.g., viruses or bacteria), while violence is transmitted from human to human by equally invisible and now newly discovered pathways. Essentially transmission means that the disease or condition causes *something of itself* to be communicated, causing another person (or animal) to take on *some of the same characteristics*. In infectious disease language it means simply that being exposed to the disease makes it more likely that you will also develop the symptom complex characteristic of the same disease. This phenomenon has been shown for violence through many studies: people who are exposed to violence—either by observing, witnessing, or being subjected to violence themselves—are more likely to become what is called a perpetrator of violence (Widom, 1989; Stith et al., 2000; Reitzel-Jaffe and Wolfe, 2001; Ehrensaft et al., 2003; Guerra et al., 2003; Crooks et al., 2007; Huesmann and Kirwil, 2007; Kokko et al., 2009; Roberts et al., 2010). This is true for multiple forms of violence, as will be summarized and interpreted later in this paper.

Infectious Diseases and Violence in Individuals

Violence not only shows the characteristics of infectious diseases in populations, but also the characteristics and key concepts of an infectious disease in an individual. These characteristics are listed in Table II-1 and

TABLE II-1 Concepts in Infectious Diseases in *Individuals*

Susceptibility (versus immunity, resistance)
Exposure, infectivity, transmission
Incubation, latency
Pathogenesis
Inapparent/subclinical
Carriers
Clinical spectrum (mild, severe, acute, intermittent, chronic)
Cure, relapse

shown schematically in Figure II-6. Space does not permit an in-depth review of these concepts, but the reader is referred to infectious disease textbooks (Anderson and May, 1991; Nelson and Williams, 2007). In brief, all of these concepts apply to violence, including susceptibility, exposure, transmission, incubation, and latency periods, as well as possibilities for different clinical courses and clinical outcomes, from minimal infection to death.

An infectious disease begins with *exposure* to the infection by a susceptible person. *Susceptibility* refers to the level (or lack) of *resistance* to infection for an individual; this could be due to the immune system (or other factors). For the usual infectious diseases, there are several mechanisms of immunity or resistance (e.g., mucosal cell integrity, or prior antibody or cell-mediated responses). Susceptibility and resistance are relative terms that can be overridden by dosage, types of exposure, or other circumstances. Drops in immunity can occur with time or context or due to changes in other biological or environmental circumstances, such as extreme temperatures or immune suppression. Immunity or resistance to exposure to violence may be a result of a family or peer environment in which views, behaviors, and norms against violence are very well established and maintained, and

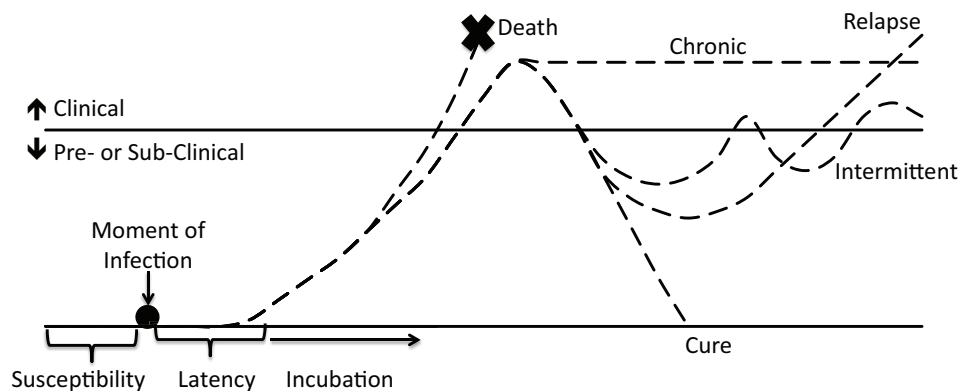


FIGURE II-6 Natural history of an infectious disease.

where alternative responses to exposure to violence are well supported, in particular among close peers (Berman et al., 1996; Osofsky, 1999; Garbino et al., 2002). In infectious disease language this is sometimes referred to as “herd immunity.”

Incubation periods, defined as the time from infection to evidence of clinical disease, is variable in both infectious diseases and violence. In other words, influenza has an incubation period of days, while leprosy has incubation periods of years. The incubation period between HIV infection and AIDS can vary from months to decades. Some infectious diseases have extremely variable periods that can be weeks or years, for example, malaria or tuberculosis. Violence can also have quite varied incubation periods—rapid like cholera, such as for soccer riots, or gang wars, or the genocide in Rwanda (Verwimp, 2004), or longer incubation periods like tuberculosis, where the period between being subjected to child abuse and becoming a perpetrator of community or family violence may be years or decades later (Ehrensaft et al., 2003; Huesmann et al., 2003).

Even prolonged latencies of decades can be seen for both, where conditions for *reactivation* may be important (e.g., Huesmann et al., 2003). Interestingly both tuberculosis and violence show this ability for a person to be infected very young and then show active disease decades later. For example, a child younger than age 5 exposed to tuberculosis may show active disease in his late teens or early 20s; likewise, an abused child age 5 or less may exhibit violent behavior (community violence or be a child abuser himself) in the late teens, 20s, or later. The intervening years would be called the *incubation period* for an infectious disease, and could also be called an incubation period for violence.

Technically, whereas incubation period refers to the time to clinical disease, *latency* refers to time until infectivity to others. This infectivity or contagion can occur from among asymptomatic or presymptomatic persons, including carriers (see below), but also from persons who have not yet completed their incubation period, but who will become symptomatic later. Latency (or infectivity to others) can therefore come before or at the same time as the end of the incubation period; for example someone may spread a diarrheal infection before they are symptomatic. The violence analogy may be that persons may be provoking others to do violence, but do not (or yet) show the characteristic symptoms themselves (definition issues here will need to be worked out, such as whether persons who train others to do violence are showing a clinical syndrome or are just contagious to others).

Persons exposed to violence, as for infectious disease, can develop a *wide spectrum of possible clinical courses or outcomes* as a result of exposure, including no disease at all, a chronic or relapsing syndrome, disability, or death. *Carrier states* for infectious diseases include the classic example

of “Typhoid Mary,” a cook at the turn of the 20th century who was a carrier of *Salmonella typhi* (the bacterium causing typhoid fever), who although having no clinical disease herself was responsible for transmitting typhoid to more than 50 persons, with 3 deaths. The analogous situation for violence disease would be the person who causes others to become violent (e.g., through provocation) without manifesting overt violence disease themselves (all of these outcomes require treatment, in individual care and public health terms, once detected).

For each infectious agent, there are many different *clinical syndromes*. For example, with plague there are bubonic (lymphatic) and pneumonic (lung) syndromes. For tuberculosis the clinical picture may be that of respiratory disease, bone disease, or even meningitis. These may appear as different disease states, but they are in fact caused by the same microorganism or infection for each of these diseases mentioned.

Likewise there are different *violence syndromes* that are currently viewed as different “types of violence” to the general public, such as community violence, intimate partner violence, child abuse, and suicide. I suggest that these now be classified as *different syndromes of the same disease* because they derive from the same cause, but manifest under different circumstances. Differences in susceptibilities, contexts, and ages may play a part, just as polio may have different manifestations in very early ages than in childhood, or how influenza differentially affects older and very young persons.

Transmission: Including Transmission Across Syndromes

Exposure to violence increases the likelihood that the exposed person will commit violence, that is, to become a perpetrator (Kaufman and Zigler, 1987; Widom, 1989; Stith et al., 2000; Reitzel-Jaffe and Wolfe, 2001; Ehrensaft et al., 2003; Guerra et al., 2003; Crooks et al., 2007; Huesmann and Kirwil, 2007; Kokko et al., 2009; Roberts et al., 2010). In some cases the likelihood of being a victim may increase as well (Coid et al., 2001; Heyman and Slep, 2002; Ehrensaft et al., 2003). If we define *violence disease* as performing acts of physical harm to others or having acts performed against you, we can see through examining these different “categories” of violence that there is a *chain of transmission that occurs across syndromes*. By comparison, someone febrile and coughing with tuberculosis as well as someone with the disease in their lymph nodes or even brain (meningeal) tuberculosis are *all* infected with *M. tuberculosis*. We know that exposure to community violence can lead to perpetrating community violence (DuRant et al., 1994, 1996; Barkin et al., 2001; Kelly, 2010). In its most obvious example, the most likely predictor of a subsequent case of

a shooting in street or gang violence is a previous shooting (Decker, 1996). Likewise, the greatest predictor of subsequent cases of colds, flu, SARS, Legionnaire's disease, and other infectious diseases is a prior case—and specifically exposure to a prior case—of that infection.

It has been said for a long time that violence begets violence, but it is *just as tuberculosis begets tuberculosis, or flu begets flu, that violence begets violence.*

We see violence causing violence in its most acute setting in cases of retaliations in gang violence (Decker, 1996) and even in war. For example, this was seen in what was called civil, or intrastate, wars, such as following the 2005 bombing of the Samarra Mosque in Iraq, or even what we call wars between states, or interstate wars, such as World War II. To an epidemiologist these should be known simply as violence outbreaks.

Furthermore, considerable evidence shows that having been a victim of violence increases the risk of someone perpetrating community violence (DuRant et al., 1994, 1996; Barkin et al., 2001; Morris et al., 2002; Mullins et al., 2004; Kelly, 2010). However, it is also now clear that exposure to *community* violence (outside the family unit) leads to an increased likelihood of *family* violence, both against intimate partners and abuse of (or violence against) children, as well as an increased risk of violence *against self* or suicide (Mullins et al., 2004; Devries et al., 2011). Furthermore, exposure to (observing) violence between parents leads to a greater likelihood of being a perpetrator of intimate partner violence (Stith et al., 2000; Reitzel-Jaffe and Wolfe, 2001; Ehrensaft et al., 2003; Naved and Persson, 2005) or child abuse (Kaufman and Zigler, 1987; Heyman and Slep, 2002; Milner et al., 2010), and to being exposed to community violence (Hanson et al., 2006). Being traumatized as a victim of *child abuse* also leads to *community violence* (Widon, 1989; Crooks et al., 2007), intimate partner violence (Stith et al., 2000; Ehrensaft et al., 2003), and child abuse (Kaufman and Zigler, 1987; Heyman and Slep, 2002; Milner et al., 2010). Exposure to war and political violence, particularly when accompanied by posttraumatic stress disorder, leads to being a perpetrator of intimate partner violence and community violence (Archer and Gartner, 1976; Landau and Pfeffermann, 1988; Sela-Shayovitz, 2005; Catani et al., 2008; Clark et al., 2010; Landau et al., 2010; Teten et al., 2010; Widome et al., 2011). Exposure to violence in the media leads to the perpetration of violence in the community and at home (Huesmann et al., 2003), as does witnessing violence in video games (Huesmann, 2010). Suicide, a type of violence directed at oneself, can also frequently follow exposure to intimate partner violence, community violence, (Cavanaugh et al., 2011; Devries et al., 2011) or other suicides (Gould, 2001; Gould et al., 2003; Jeong et al., 2012).

Further evidence of this *cross-syndrome connection* has been shown, for example, in studies by Eric Dubow and Rowell Huesmann in war settings. These studies have shown, in the setting of Israeli Jew, Israeli Arab, or Palestinian Arab, that exposure to or involvement in ethnopolitical violence leads to the performance of violence against spouses and peers, removing any pretense of the primacy of “reasons” for violence (Dubow et al., 2009; Landau et al., 2010). Like the example of different forms of tuberculosis, something common has been transmitted—in this case, a tendency toward violence, likely mediated by underlying biological processes. A violence disease or predisease state is present.

Therefore, something is being transmitted across and between various “types” of violence. Because something common is being transmitted, likely involving common intermediate brain pathways, these different “types” of violence should be called syndromes of the same *violence disease*.

Definitions—Violence Is a Contagious Disease— and Is Like an Infectious Disease

Disease

Dorland’s Illustrated Medical Dictionary, 32nd Edition (2010), defines a disease as “any deviation or interruption of structure or function of a part, organ, or system of the body, as manifested by *characteristic symptoms and signs* (causing morbidity and mortality); the etiology, pathology, and prognosis may be known or unknown.” The classic Oxford dictionary defines a disease as a “*pathological condition* of a part, organ, or system of an organism resulting from various causes, such as infection, genetic defect, or environmental stress, and characterized by an *identifiable group of signs or symptoms*.”⁷

I would suggest that the characteristic signs and symptoms of violence are the behavioral actions that cause or attempt to cause physical injury to another person or to one’s self, and that these constitute a disease. I would add that anyone who has suffered physical injury as a result of violence, and in some cases been traumatically threatened, may also be considered infected, or diseased. In other words I am suggesting that both what is called perpetrator and what is called victim in the current literature be considered as *violence infected* or having the violence disease. I also suggest that, until we develop a clear marker for infection, we consider most persons that are exposed as infected, and clinical disease as the presence of symptoms. In

⁷ A second definition, referring to a condition of society, reads “a condition or tendency, as of society, regarded as abnormal and harmful.”

many infectious diseases, there are many more people infected than have clinical disease.

Contagious and Infectious

Dorland's medical dictionary defines contagious as "capable of being transmitted from one individual to another; communicable." This has been shown in the preceding section of this paper, for many clinical syndromes of violence. Violence is a contagious disease.

For infectious disease, some definitions or medical experts may prefer or choose to require a free-living microorganism, or physical agent, and for them violence may not be considered an infectious disease. However, not all microorganisms or microscopically transmissible definable entities are free living, for example, viruses or prions. Some medical textbooks refer to infectious as having a presence of a microorganism, but not always (Dorland, 2010; Stedman, 2012). The characteristic of infectivity itself is frequently synonymous with contagious or communicable, and this sometimes differential in medical textbooks may be simply conforming to the need for practitioners to be able to use antimicrobial agents or conventional medical approaches. However, as a practitioner, I am aware of the existence of many infectious diseases in which we do not have effective antimicrobial agents nor immunization (e.g., Ebola, Marburg, many viral diseases, antibiotic-resistant diseases, and for many years, AIDS), yet we still need to have effective approaches.

Using the term *contagious* remains technically sound, while avoiding possible controversies around the need for a physical agent that the term *infectious* might require for some.

Means of Transmission

Infectious diseases have many routes and means of transmission, from respiratory to fecal-oral to bloodborne to vectorborne. A full listing is available in most infectious disease textbooks. Pathogens can enter via the respiratory tract, gut, skin, or other routes to then cause dysfunction or dysregulation of one or more organs.

In the case of violence, we are looking at a process clearly mediated by the brain, with transmission appearing to come from at least two possible pathways: visual observation (o) and direct victimization (v). A third mechanism may be considered intentional training (t), for example by the military. Following transmission there are mediating factors that help predict the likelihood of a "take," and intervening or mediating mechanisms facilitate whether exposure or infection is likely to result in disease, which in this case is a violent act.

Mechanisms of Contagion or Infectivity, and Pathogenesis of Disease Formation

Biological mechanisms underlie the acquisition of infectious and other diseases. These are not just mechanisms of destruction or tissue damage, but frequently changes in organ *function* such as regulation, or dysregulation (e.g., immune responses in the lung to tuberculosis, flu or cold viruses).

For infectious processes, biological mechanisms must be elucidated for acquisition, and pathogenesis and mediators of progression defined. With respect to violence, where the behavior is being transmitted, Albert Bandura showed that social learning or what we could call imitating or modeling, is a principal mechanism for the acquisition of behaviors (Bandura and Huston, 1961; Bandura et al., 1961; Bandura, 1977, 1986). Several variables cause behaviors to more likely be copied, such as proximity to the learner and dose, effectively the amount, or intensity of exposure. The biological mechanisms here are not well known, but may involve cortical mirror-type of circuits, which are likely more complicated than mirror neurons alone (Iacoboni et al., 2005; Uddin et al., 2007). Besides acquiring simple behaviors, there is evidence for the acquisition of “scripts” or more likely responses to common events (Huesmann and Eron, 1984; Huesmann and Kirwil, 2007). Such behaviors are then maintained in large part by how the brain maintains habits, and by the largely invisible force of social pressure or expectations of peers. It may be that rewards for social approval, or other cues to belonging to social networks (e.g., positive reputation, consensus) may be mediated by dopamine-like reward pathways (Baumeister and Leary, 1995; Izuma et al., 2008; Losin et al., 2012). Perhaps equally importantly, it appears that *not* belonging (or social isolation) engages the same brain regions (shows up on brain scans) with some of the same patterns as physical pain (Panksepp, 1998; Eisenberger and Lieberman, 2004, 2005; Macdonald and Leary, 2005; Eisenberger, 2011, 2012), and is therefore avoided at great cost. Additional research shows that trauma (an outcome of exposure to violence) causes dysregulation in the limbic system and prefrontal cortex leading to hypervigilance (Margolin and Gordis, 2000; Perry, 2001; Fonzo et al., 2010), and hostile attribution (Joshi and O’Donnell, 2003) to perceived insults, resulting in more rapid and less regulated responses to real or perceived insults. These regions are affected by exposure to violence (Wang et al., 2009; Hummer et al., 2010). These mechanisms appear to be some of those that may underlie the infectivity of violence itself, as well as those underlying the capabilities for escalation, and rapid recruitment of individuals and further events.

In other words, both the infectious nature of the violence disease and the intervening brain processes causing the violence disease process can, at least in part, be defined, or at least speculated on, with refinements and

new research certain to continue. These pathways could be considered, for example, parallel to how infection by the cholera bacterium causes the severe diarrhea characteristic of cholera disease, not by *destroying* the intestines, but by causing a *dysregulation* of salt and water transport in the intestine (with *V. cholera*, the dysregulation is manifested by a blocking of the Na-K pump that absorbs water in the small intestine, thereby causing diarrhea and likewise the perpetuation and additional infectivity to others of the clinical syndrome). Similarly, brain processes affected by observation and trauma cause both *alterations and dysregulation* of specific mechanisms and pathways in the brain noted above.

It is important to add that not all people infected with infectious diseases (or violence) will show disease. In fact for many infectious diseases, a minority of persons develop clinical disease following infection. For example, approximately 2 billion people in the world are currently infected with tuberculosis, but only approximately 9 million have cases of the clinical disease, with 1.4 million deaths per year (WHO, 2012). Many factors influence the likelihood of disease, and both infectious diseases and violence are more likely to “take” and progress with larger doses, particular contexts, less immunity, certain types of exposures, and absent or ineffective treatment.

Treating Violence as an Infectious Epidemic Is Effective

Three main strategies are used in reversing infectious epidemic processes. These are (1) detecting and interrupting ongoing and potentially new infectious events; (2) determining who are most likely to cause further infectious events from the infected population and then reducing their likelihood of developing disease and/or subsequently transmitting; and (3) changing the underlying social and behavioral norms, or environmental conditions, that directly relate to the spread of the infection (Nelson and Williams, 2007; Heymann, 2008).

The Cure Violence (previously known as CeaseFire Health) Method uses these same principles that are used to reverse infectious epidemics to prevent and reverse epidemic violence. The Cure Violence Method is therefore, both a science and community/street-based intervention. The method was designed in the late 1990s in Chicago, piloted in 2000 in West Garfield Park, replicated in multiple cities throughout the United States and other countries, independently evaluated, and is now considered a best practice by several national and international organizations and publications (DOJ, 2009; *The Economist*, 2009; Skogan et al., 2009; U.S. Conference of Mayors, 2012; Webster et al., 2012).

The Cure Violence Method begins by analyzing the clusters involved and transmission dynamics, and uses several new categories of disease

control workers—including violence interrupters, outreach behavior change agents, and community coordinators—to interrupt transmission (or the contagion) to stop the spread of the violence disease and to change underlying norms. Workers are trained as disease control workers, similar to tuberculosis or HIV/AIDS workers or those looking for first cases of bird flu or SARS (Slutkin et al., 2006; Ransford, in press).

Tuberculosis workers help find cases and ensure that persons are sufficiently rendered noninfectious, albeit in the case of tuberculosis it is through the use of antimicrobial agents. However, tuberculosis outreach workers also require the use of persuasion (e.g., for taking medications) to ensure that effective change is occurring. Cure Violence disease control workers have training in modern methods of persuasion, behavior change, and changing community norms—all essential for limiting spread of outbreaks of violence. The principles underpinning the approach come from modern knowledge of social psychology and brain research, just as the principles of controlling other infectious disease flow from understanding their underlying mechanisms and patterns of flow.

Some of these principles include using persons from the same “in-group,” which causes less defiance and more trust, credibility, and access. A number of cognitive processes are sensitive to group membership and for assessing “us” or “them” (Mathur et al., 2010; Bruneau et al., 2012), and determining whether someone is working in your own interest or not. The modern practice of behavior change requires the use of credible messengers, as well as ensuring that the new behaviors are acceptable and feel right socially, including being able to overcome social, physical, and other barriers (for example, the pressure that other groups are doing it). Messages need to be constructed to include new information about the behavior and new skills practiced along with developing opportunities for positive peer reactions and avoiding negative peer reactions. Violence interrupters’ training also includes new and newly anticipated responses so that new brain circuits can be used in the short and longer term, as well as new social pressure and direction for “belonging.”

Changing norms is done most effectively by putting some of these practices in play to scale as well as questioning existing norms and proscribing new norms at population levels. As thoughts, behavioral scripts, and norms are transmissible, new scripts and norms are developed and a new set of behaviors becomes more normal. Interruption is essential; however, brain processes, including preexisting emotional dysregulation as well as continued peer pressures to belong, remain problems if unattended to or untreated.

Changing norms is done most effectively by putting some of these new practices into play to scale—by developing a cascading diffusion through social networks, gradually accumulating the new responses. This

is accelerated by systematically questioning existing norms and proscribing new norms at population levels. As thoughts, behavioral scripts, and norms are transmissible, new scripts and norms are developed and a new set of behaviors becomes more normal. Interruption remains essential; as brain processes, including preexisting emotional dysregulation difficulties—as well as continued peer pressures to belong—remain problems if unattended to or untreated.

These methods have resulted in reductions in shootings and killings of 16 to 28 percent directly attributed to the strategy by time series analysis (see Table II-2); from 41 to 73 percent overall (Skogan et al., 2009); and in its first outside replication, in Baltimore, reduced shootings and killings by 34 to 56 percent (Webster et al., 2012). The initial implementation has been replicated in more than 20 communities in Baltimore, Chicago, New York, and several other cities with large reductions in violence found by independently performed studies commissioned by the U.S. Department of Justice, the Centers for Disease Control and Prevention, and Johns Hopkins University (Skogan et al., 2009; Webster et al., 2012).

This new approach is now being used by more than a dozen U.S. cities and a growing number of countries, including in Kenya to prevent or reduce election violence, South Africa to reduce and prevent community violence, and Iraq to reduce and prevent interpersonal and intertribal violence.

The idea of violence as a contagious or infectious disease is rapidly catching hold. In 2008, the *New York Times Sunday Magazine* cover story by Alex Kotlowitz about the Cure Violence epidemic control method (formerly referred to as CeaseFire) ran with the title “Is Urban Violence a

TABLE II-2 National Institute of Justice External Evaluation of CeaseFire Chicago: Three Approaches to Impact Analysis

	Change in Violence Due to Program		
	Shootings Down	Hot Spots Cooler ^a	Retaliation Homicides Down
Auburn–Gresham	-16%/-21%	-15%	-100%
East Garfield Park	Not evaluated		-100%
Englewood			-100%
Logan Square	-21%		-100%
Rogers Park		-40%	No change
Southwest	-20%/-23%		-100%
West Garfield Park	-22%/-28%	-24%	-46%
West Humboldt Park		-17%	-50%

^a Hot spots are locations where shootings are particularly concentrated. Cooling indicates a reduction in this concentration after implementation.

Virus?” The 2009 *Economist* special “World in 2009 Edition” described the epidemic control approach and predicted that this would be “the approach that would come to prominence.” The recent award-winning documentary *The Interrupters* also highlighted the disease control approach.

The science, and the public understanding that follows this science, are bringing us into a new era. This new era is an era of *discovery*—but more importantly of *transition*. We can now leave the days of a vocabulary of “bad people” and “enemies” and apply a scientific understanding and a scientific approach to this problem. Violence has all of the historical, population, and individual characteristics of an infectious disease. It has routes of transmission, incubation periods, and different clinical syndromes and outcomes. There are definable biological processes underlying the pathogenesis. In addition, treatment as an infectious disease is effective. All of this requires more refinement and research. We are still performing research and refining our approach with tuberculosis, cholera, and malaria as well, but at least we have taken these problems out of the moral, medieval, and superstitious realms of evil and dungeons.

The advantages to this new and scientific understanding and approach to violence are countless. We can more proactively *avoid exposure* and develop new ways of responding to exposure. We can treat and develop *better methods of treating* infected persons and communities. We can further strengthen the Cure Violence and other early epidemic control approaches referred to here. Most of all, we can now move away from counterproductive practices into the *modern era*.

Violence is a contagious disease. This is good news as this knowledge offers new strategies for control. There are massive implications for how to better treat urban violence, as well as for international conflicts. As we have done before—for plague, typhus, leprosy, and so many other diseases—we can now apply *science-based strategies* and, as we did for the great infectious diseases, similarly move violence into the past.