

THE INFECTIOUSNESS OF TERRORIST IDEOLOGY

Insights from Ecology and Epidemiology

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Terrorism in the twenty-first century is unconventional, unpredictable, and potentially unavoidable. In part, this is because contemporary terrorists are increasingly transnational, industrious, unorthodox in their methods, and decentralized (e.g., Ariza 2006; Ehrlich and Levin 2005). Some have proposed that we view terrorism through the lens of epidemiology, where terrorist ideology is analogous to an infectious agent of threat to global public health (in particular, Stares and Yacoubian 2005). While the terrorist ideology–infectious agent analogy has obvious utility, we recognize that it is both young and imperfect. Here, we take the next step and investigate the value of this analogy.

Similarities between terrorism and pathogen outbreaks have resulted in parallel, but independent, tracking, prevention, and control efforts. The National Strategy for Homeland Security, for instance, “recognizes that the capabilities and laws we rely upon to defend the U.S.A. against terrorism are closely linked to those which we rely upon to deal with non-terrorist phenomena such as disease” (Office of Homeland Security 2002, 4). If sufficient parallels exist between infectious agents and terrorism, counterterrorist efforts may be able to draw on the already substantial body of theory developed for public health (see the box “Making the Metaphor Practical”). In this chapter, we explore if and how epidemiological theory can increase our understanding of the dynamics and spread of terrorist ideology—the belief structures on which acts of terror are supported, justified, and carried out. We use the terms infectious agent, disease, parasite, and pathogen interchangeably throughout this chapter and as analogs to terrorist ideology. We use our findings to identify key unanswered questions and avenues for future research. In considering this analogy, we do not promote any particular counterterrorist strategy or recommend policy.

Making the Metaphor Practical

In their informative article “Unconventional Approaches to an Unconventional Threat: A Counter-Epidemic Strategy,” Stares and Yacoubian (2005) point out what they perceive to be the three most practical applications of epidemiology and public health to combating terrorism. (They specify Islamist militancy, while we consider terrorism generically.)

1. Epidemiologists observe rigorous standards of inquiry and analysis to understand the derivation, dynamics, and propagation of infectious agents. They seek clarity on the origins, geographical, and social contours of an outbreak: where is the pathogen concentrated, how it is transmitted, who is most susceptible to infection, and why are some immune? Applying the same methodological approach to mapping and understanding terrorism can yield immediately useful guidance on where and how to counter it.
2. Epidemiologists recognize that infectious agents emerge and evolve as a result of complex interactive processes between hosts, pathogens, and the environment in which they live. To make sense of this complexity, epidemiologists deconstruct the key constituent elements of an infectious agent. This model helps to understand the phenomenon in its entirety and anticipate how it might evolve in the future. The same systemic conception of infectious agents can be adapted to understand the constituent elements of terrorism and their evolution (Fig. 12.1).
3. Epidemiologists view infectious agents as complex, multifaceted phenomena. Public health officials have thus recognized that success in controlling and rolling back an epidemic requires a carefully orchestrated, systematic, prioritized, multipronged effort to address each of its constituent elements. However, it is also recognized that significant progress or major advances can sometimes be precipitated by relatively minor interventions. Again, there are lessons and insights to be learned here for orchestrating a global counterterrorism campaign.

Ideology can be considered a type of meme or suite of memes. Memetics, the study of the contagiousness of thought and the dynamics that govern the spread of ideas, has captivated a broad audience for many years (e.g., Dawkins 1976; Lynch 1996; Gladwell 2000; Distin 2005). Dawkins (1976) defines memes as any cultural entity (e.g., a fashion fad, song, idea, religion, language) that is replicated through exposure to humans and has evolved as

an efficient (though not necessarily perfect) copier of information and/or behavior. Much of the inquiry into memetics has centered on why some memes spread in an “epidemic” fashion. Like genes, memes spread if they provide a clear benefit to an individual, for example, through group identity, enhanced sexual attractiveness, or increased resource acquisition (Sober and Wilson 1998). Moreover, memes with no apparent benefit may arise and become fixed in a population simply because they possess characteristics that render them more likely to be adopted than another meme (e.g., incorrectly transmitted verbal phrases that contain more audible syllables than their grammatically correct counterparts [Dawkins 1989]). Even memes with a seemingly negative impact may spread through their provision of a net indirect benefit. For example, individuals with a “handicap” may be attractive to others due to the perception that the handicapped individual tolerates adverse conditions (Zahavi 1975) or actions that lead to the perception of a future benefit (rewards in the afterlife) can spread even if they do not increase fitness (see also Sosis and Alcorta, this volume). Here, we focus on terrorist ideology, but the analogy could apply to ideology in general. Society has often attempted to suppress memes such as political ideology, religion, and language, and the infectious disease analogy could serve to better understand these actions. However, while the infectious disease analogy could apply to ideology in general, our interest is specifically focused on the present broad-scale efforts to suppress terrorism ideology.

The Infectiousness of Terrorist Ideology: Constructing the Analogy

The analogy takes shape at the system level, in the complex where infectious agents and terrorist ideologies exist (Fig. 12.1). Both entities require a unique set of conditions to remain established in a given system. Infectious agents depend on one or multiple hosts to support and complete their lifecycle (development, maturation, and reproduction). Host-pathogen dynamics can be viewed at the level of a host individual, host population, or community of species. Analogous to this, a given ideology can be “hosted” by a terrorist, terrorist cell, or terrorist organization. It is within these individuals and groups that ideology is conceived, formed, developed, and honed. Infectious agents need hosts just as ideology cannot exist without the minds that harbor it. In addition, pathogens and ideologies can persist in alternate forms outside of a host; many infectious agents have free-living resting stages and ideologies can be preserved outside the mind by various media.

Neither an infectious agent nor its host can exist in a system where external conditions are unsuitable. Indeed, abiotic (i.e., climatic) and biotic (i.e., competition, predation) factors help to shape the boundaries of an infectious organism’s geographic range. The analog to this is the political,

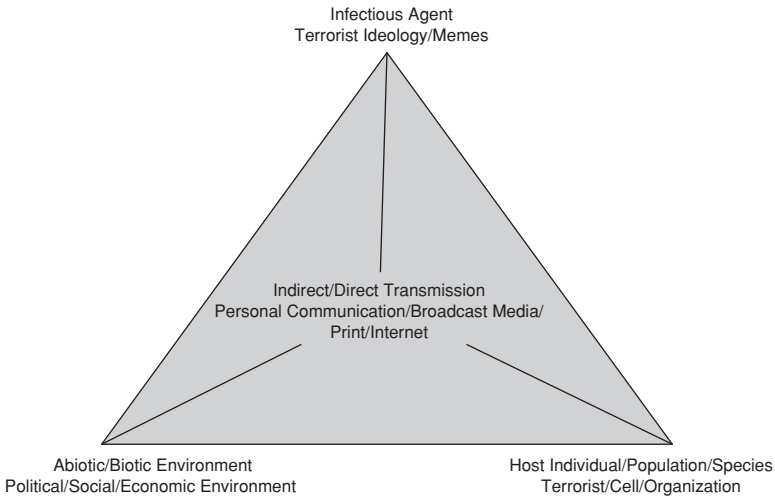


FIGURE 12.1 The analogy between the spread and dynamics of infectious agents and terrorist ideology is best depicted through a conceptual model of the systems where each entity exists. Adapted from Stares and Yacoubian (2005).

social, and economic environment that shapes the region where terrorist ideology evolves and thrives. Changes to the environment might foster the evolution of ideology away from that which promotes terrorism. For instance, the transition of a terrorist group to a recognized political power may lead to ideological or methodological changes better suited for formal governing (Prusher 2006).

Finally, it is the transmission of infectious agents and terrorist ideology between hosts/terrorists that maintains the entity's existence within the system. Infectious agents can be transmitted between susceptible hosts directly, as in the case of sexually transmitted pathogens, which require person-to-person contact. Transmission may also occur indirectly, by way of a vector that harbors the infectious agent (e.g., mosquitoes carrying malaria or ticks carrying Lyme disease), or through a contaminated vehicle such as food (e.g., salmonella) or liquid (e.g., giardiasis). The spread of terrorist ideology among individuals occurs via similar routes. Ideology may be transmitted directly, through the oral exchange of ideas from a terrorist to an individual who does not yet harbor the ideology. It may also spread indirectly, via a vehicle/vector such as broadcast media, print media, or the Internet.

Conceptual models, such as the one depicted in Fig. 12.1, are a common first step in understanding the framework of a system where an infectious agent exists. As we have shown here, the model is also well suited for

deconstructing the complex where terrorist ideology thrives. Following the path of an investigative epidemiologist, our next task is to consider the quantitative models that best describe the spread of infectious agents/terrorist ideology.

Ecology and Epidemiology

Ecology and epidemiology have a highly developed set of theoretical tools and mathematical modeling approaches for understanding the basic properties of infectious agents. In many cases, such techniques have greatly informed control practices (Smith et al. 2005). Here we compile the theoretical framework from ecology and epidemiology that is commonly used as the basis for studying the spread of infectious agents. We extend these concepts to account for the establishment and spread of terrorist ideology and consider their utility in counterterrorism strategies. Johnson and J. Madin (this volume) similarly explore the relevance and applicability of models developed for fisheries stock assessment to the control of insurgent forces in unstable areas.

Models

The most basic models of pathogen spread divide a population into two categories that reflect infection status: individuals susceptible (S) to or infected (I) with the pathogen (Fig. 12.2). SI models apply specifically to a group of infectious agents called microparasites, which include many viruses, bacteria, and protozoa. The rate of change in the abundance of individuals in each category (S or I) is described by a differential equation constructed from variables such as contact rate, susceptibility, birth rate, mortality, and the abundance of individuals in the host population. The frequency of a particular category can increase, decrease, or stabilize over time. A key to such models is that the differential equations are coupled. For simple formulations, it is possible to solve these equations analytically and gain general insight. In most cases, however, finding analytical solutions for coupled differential equations with more than two categories is mathematically intractable. In these cases, epidemiologists have turned to a very effective shortcut called the basic reproductive ratio, or R_0 .

R_0 is the estimated number of secondary cases directly arising from one primary case (Anderson and May 1979). In deterministic models, if $R_0 < 1$, the epidemic will fizzle, while for $R_0 > 1$, an epidemic will occur. Once an epidemic begins, it grows at a rate known as the effective reproductive ratio, R , which tends to decline from R_0 . R can stabilize around 1 (become endemic in the population), or it can tend toward 0, at which point the pathogen is extirpated from the population (cycling, chaos, and other

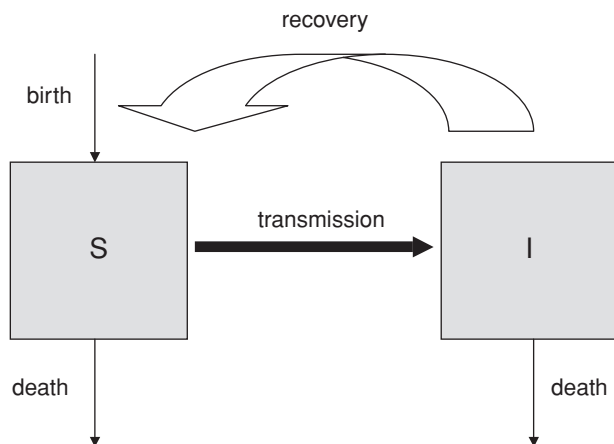


FIGURE 12.2 Flow diagram representing the basic SI (susceptible, infected) model used to study the spread of infectious agents. The model may be applied to the spread of terrorist ideology. Hosts/individuals are classified into one of the two categories. Young born into the susceptible category may come into contact with an infected individual/terrorist, transfer into this category, and either die from the infection/terrorist ideology or recover and return to the susceptible category.

complex outcomes are also possible). Determining the structure of the R_0 equation helps identify the variables (and their interactions) that are most likely to determine if infection will spread through an uninfected population.

Although an SI model tracks two classes of individuals over time, only the rate of change of the infected individuals is needed to calculate R_0 . A simple component of an SI model that tracks the per capita rate of change of infected individuals (I) in an SI population can be written as

$$dI/dt = S\beta - (\mu + \alpha + r),$$

where S is the number of susceptible individuals, β is the transmission coefficient (how contagious the infectious agent is to susceptibles), μ is the background mortality rate, α is the additional mortality rate suffered by infected individuals, and r is the recovery rate. This model assumes that contacts occur at random and are density dependent (e.g., an individual living in a large city will come in contact with more people per day than an individual living in a rural community). R_0 is simply a ratio comprising the expected rate of transmission through contact with susceptible individuals

divided by the sum of the death and recovery rates. The basic reproductive ratio is, therefore,

$$R_0 = S\beta/(\mu + \alpha + r).$$

By analogy, individuals that are exposed to and “infected” with terrorist ideology become terrorists. Some part of the remaining individuals in the population, the nonterrorists, are susceptible to the ideology. Understanding the components of R_0 for the spread of terrorist ideology may provide greater insight into the types of counterterror strategies that could be used to reduce R_0 below 1 and thus diminish the likelihood that terrorist ideology will spread. Efforts to reduce R_0 below 1 may concentrate on increasing the death rate of “infecteds,” decreasing the abundance (or susceptibility) of susceptibles, or decreasing contact rate between the two. This seems, at first glance, to be a very clean analogy with obvious application. There is, however, an important caveat: the type of pathogen that terrorist ideology is compared to may fundamentally alter the predictions of the analogy.

Terrorist Ideology: A Virus or a Worm?

Parasitologists realized that parasitic worms were not well modeled by the SI equations developed for viruses. This is because hosts exposed to many worms suffer higher pathology than hosts exposed to few worms. Hosts with many worms also contribute more to the growth of the total worm population. In addition, few infected hosts have the average level of infection: most have light, nonpathogenic infections, and the majority of the worms are in a few highly infected individuals (such distribution of parasites in a host population is referred to as aggregation). For this reason, the SI categorization of hosts as infected and uninfected fails to capture important aspects of the population dynamics of parasitic worms. New “macroparasite” (worms) models were developed to account for this level of biological detail (Crofton 1971; Anderson and May 1979; May and Anderson 1979). These models provided considerable insight into how to target control efforts toward the few, most heavily infected individuals.

As with parasitic worms, the spread of terrorist ideology may not be well explained by SI models. In particular, if ideological fervor can be considered a continuous variable, then some segments of a population may possess none of the ideology, some may possess a little, and a few may subscribe with high intensity. Individuals that subscribe most to the ideology may be more likely to spread the ideology than do those that are merely sympathetic or supportive of the ideology. This opens the question as to whether variation in the intensity of terrorist ideology among individuals is sufficiently important to justify models that are more complicated. If so, it is important to note that macroparasite models only indirectly account for

the intensity of infection. Such models do not distinguish infected from uninfected hosts. Instead of tracking infected hosts, they track the total worm population. In applying macroparasite models to terrorism, the currency tracked would be the amount of terrorist ideology in the population, not the number of individuals subscribing to the terrorist ideology. The population level of terrorist ideology would have to be measured by various proxies (e.g., attack rate or internet chatter). The differential equation representing the growth of the worm population has a number of new terms. In a very simple formulation (ignoring here the change in the host population), the per capita rate of change of the adult worm population is

$$dW/Wdt = IS\beta/W - (\mu + \alpha + d + \alpha W(k + 1)/Sk),$$

while the per capita rate of change in infectious stages is

$$dI/I dt = \gamma W/I - \phi - \beta S,$$

where the variables are the same as above except that W is the total number of worms in the worm population, I is the total number of infectious stages, β represents contact between infective stages and hosts, d is the death rate of adult worms within the host, γ is the rate that worms produce infective stages, ϕ is the death rate of infective stages in the environment, and k is an inverse measure of the degree of aggregation of worms in the host population. Solving for the basic reproductive ratio of a macroparasite is much more difficult than for a microparasite, and alternative formulations for R_0 in macroparasites can be considerably different, depending on the biological details of the model (Roberts et al. 2002). One approach is that R_0 is the product of the mean number of new infections produced by a single adult parasite and the average life expectancy of adult and larval stages, or

$$R_0 = S\beta\gamma/[(\mu + \alpha + d)(\phi + S\beta)].$$

Clearly, R_0 increases with the lifespan of adult worms. Therefore, one can use chemotherapy to reduce the spread of disease. The spread of ideology could similarly be controlled by changing the ideology of terrorists, or, as seen in the microparasite models, reducing terrorist life span. An additional insight from this formulation not found in microparasite models is that R_0 is sensitive to the lifespan and production of infective stages. This is not often possible to control in parasite control programs, but the ideological analogue of infective stages (writings, media, and direct communication) may be possible to target.

An alternative to a macroparasite model is an SI model where contact rate varies among individuals. This has been of particular interest in understanding

the spread of the HIV virus because the number of sexual partners varies greatly among individuals, with most of the HIV transmission stemming from a small number of highly sexually active people (Johnson et al. 1989). Similarly, some individuals who subscribe to terrorist ideology may actively make contact with others to promote its spread, while others are more private in their beliefs and thus contribute less to transmission. The derivation of models applied to this scenario is beyond the scope of this chapter, but the important result is that variation in contact rate among individuals can greatly increase R_0 (May and Anderson 1988). This suggests that targeting individuals who are disproportionately active will reduce the spread of terrorist ideology, a conclusion that seems intuitive. However, these models describe differences in contact rate that are distinctive to an individual whether or not he or she is infected (i.e., the link between infection and the propensity to spread an infection is indirect), which is different from the macroparasite models, which stipulate a direct link between infection intensity and spread of infection. It is not necessarily clear which approach provides a better analogy for terrorism. We attempt to tackle this conundrum by deconstructing and examining, in detail, the major contributing variables in the models: susceptibility, contact rate, recovery, and mortality.

Extending Epidemiological Variables to the Spread of Terrorist Ideology

Susceptibility

Without susceptibles, an infectious agent cannot spread. Although some infectious agents are able to evolve adaptations to get past host defenses, they are less likely to adapt to two host species that differ greatly in physiology and/or evolutionary history (Combes 2001). As a result, not all host species are susceptible to all pathogens, and host specificity, or the restriction of the number of host species that a pathogen can infect, is widespread.

Ideological specificity might arise for the same reasons as host specificity. The specific cultural identity of individuals (religious, national, historical) may predispose them to some, but not other ideologies. If an ideology is recognized as clearly foreign, an individual may more likely reject it as “nonself.” Xenophobia is a common aspect of human cultures that probably acts to maintain cultural identity and prevent the spread of foreign ideologies (Boyd and Richerson 2005). For this reason, an ideology that is successful in spreading through a particular population may do so because it contains attributes compatible with that population’s culture. In contrast, a population may be less susceptible to a terrorist ideology if that ideology differs greatly from the culture’s modal ideology. This suggests that the cross-cultural spread of terrorist ideology may be unusual, and it is probably not reasonable to assume that all cultures are equally susceptible to a

particular ideology—just as host species are differentially susceptible to infectious agents. While some terrorist ideologies span cultures, others take vastly different forms between cultures, or the same goals are rationalized in different ways salient to each culture.

A second pattern in susceptibility to infectious agents is that for a single species (or closely related species), populations that have had an evolutionary history often evolve adaptations against an infectious agent, while naïve populations can be more susceptible and less tolerant. The pressures that result in the evolution of resistance derive from the negative fitness consequences of an infectious agent on its host. This suggests that nations with a history of battling terrorism might be more likely to resist the future spread of terrorist ideology within their population.

The environment can also alter a host's susceptibility to an infectious agent. Thermal stress, for example, appears to increase the susceptibility of some marine organisms to different infectious agents (Lafferty et al. 2004). Other physiological stressors, such as lack of food, may force a host to redistribute resources away from defense against infectious agents (Rigby and Moret 2000). While stress at the individual level should tend to increase susceptibility to infectious agents, it may, unexpectedly, reduce the spread of a pathogen through a population. This occurs for the following reasons: stress may reduce host abundance (and, therefore, contact rates), and stress can increase pathogen mortality rates either through killing pathogens directly or by leading to differentially high mortality of infected hosts (Lafferty and Holt 2003). The environment could similarly affect the number of individuals susceptible to terrorist ideology if personal stress is a result (unemployment, reduced mating opportunities, frustration, humiliation, revenge). In environments that are unfavorable (poor in justice, resources, or freedom), new terrorist ideologies might hold appeal, particularly if they offer the promise of change, and current or alternative ideologies do not appear effective.

Contact Rate

Pathogens and ideologies spread primarily through contact. SI models assume that in dense populations, individuals are more frequently in physical contact with one another. Another aspect of contact is the extent of movement among populations. This is difficult to capture in simple epidemiological models, but the logic is straightforward: contact between individuals of different populations is likely to lead to a wider spatial spread of an infectious agent. This is why increased contact through diffuse networks related to increases in transportation and modern trade has led to increasing concern for the spread of pandemics (Hufnagel et al. 2004). Current concern about avian influenza is one such example, and rightly so, as the

1918 avian flu pandemic may have been associated with large-scale troop movements during World War I (Barry 2004).

How similar is the spread of an ideology to the spread of an infectious agent? Whereas sick people do not consciously try to contact and infect other individuals (with the exception of some infectious diseases such as rabies), it is human nature to share ideas and convince others to agree with opinions. This is not to suggest that pathogen transmission is passive in comparison to ideological transmission. Infectious agents that are most likely to persist are those with traits enabling them to spread from one individual to another. Symptoms such as coughing, sneezing, and diarrhea are examples of behaviors induced by infectious agents to facilitate spread (Ewald 1993). It is also likely that ideologies are under analogous forms of selection for characteristics that favor spread (Dennett 2006). Successful religions often have doctrines favoring spread, such as active conversion of others, increased reproduction, early indoctrination, and retention (e.g., symbiotic theories of the spread of religious ideologies). It has been suggested that such “symbiotic” relationships between religion memes (i.e., ideology) and their “hosts” may take the form of mutualism, in which both the meme itself and the host benefit, or they may be parasitic, in which the host is in some way oppressed by the religious ideology while the meme itself benefits (Dennett 2006). We may expect successful terrorist ideologies to possess adaptive traits for spread or to be aligned with existing religious ideologies. For example, terrorist ideologues have been successful at recruiting members through religious schools (Gunaratna 2005).

There are different opportunities for the spread of an ideology than for the spread of an infectious disease. Electronic communication greatly facilitates the spread of information at speeds and scales far exceeding physical contact. Information contact is increasing due to fewer language barriers, landline and cellular phones, television, radio, and the Internet. This decouples the spread of an ideology from local population density (Ariza 2005) while simultaneously favoring decentralization and spatial spread. Terrorist cells, in particular, tend to communicate and exchange ideology and information primarily through the Internet. This is not to downplay the importance of direct communication. A study of enlistees into a terror network found that communication of ideologies primarily occurs horizontally, through immediate and secondary friends (~80%). The remaining 20% occurs vertically, through kinship ties (Sageman 2004). Following enlistment, individuals often self-organize into isolated cells, the preferred size of which is eight members.

Recovery

Infected hosts frequently recover, often with the assistance of an immune system. Recovered individuals may be permanently immune to subsequent

infection. Anyone old enough to reflect on his or her childhood appreciates that ideologies change over time, and it seems safe to assume that “terrorist” is not necessarily a permanent ideological state. In borrowing from the SI model framework, we might divide individuals into distinct ideological classes of terrorist (infected) and nonterrorist (susceptible). While conversion from nonterrorist to terrorist seems to be analogous to an infectious process, recovery from terrorist to nonterrorist is not analogous to an immune defense. In addition, while an individual’s ideological intensity may wane or increase over time, a person does not necessarily adopt an ideology then lose it over time to a nonideological state. Ideologies can persist over time and may coexist with or interact with other ideologies. In other words, there may be a potential series of “infected” states. This is particularly important for the concept of recovery because a change in ideological states would depend on the extent that individuals can be exposed to alternative ideologies. In a sense, this is more like pitting two infectious agents against one another than the effect of an immune system.

Although replacement is not the rule for infectious agents, we can turn to flukes (parasitic trematode worms) for insight. Trematodes have complex life cycles that typically involve a snail as a first-intermediate host. Once infected, snails rarely revert to an uninfected state. In many cases, several species of trematode infect the same species of snail, but there is generally only room for one trematode species at a time within an individual snail (few trematode species pairs can coexist). For this reason, trematodes have developed special adaptations for battling with other trematodes inside the snail, and, in many cases, one can predict which trematode species will win the internal battle for host occupation (Kuris and Lafferty 1994). If ideologies, like trematodes, have dominance hierarchies, facilitation of dominant ideologies could lead to permanent “recovery.”

Mortality

All else being equal, highly pathogenic infectious agents have a smaller R_0 because infected individuals with short lifespans have fewer opportunities to transmit an infection. A terrorist’s actions may create an analogous situation. Even in the absence of counterterrorist efforts, terrorist mortality rate should increase because militant terrorist activities are inherently dangerous (e.g., weapons training, handling explosives, suicide missions, primitive living). These dangers may therefore make it more difficult for terrorist ideology to spread. However, risky behavior and high mortality rates may also increase ideological spread. This would be particularly true if suicide attacks lead to martyrdom, benefits for the attacker’s family, or a desirable afterlife, or if mortality achieves a terrorist goal and inspires others to join.

Ecological and epidemiological models also suggest that removing infected individuals from the population can reduce the impact of the infectious agent. Although society does not permit the culling of infected patients, culling infected animals has been shown to be effective in preventing the spread of veterinary and zoonotic pathogens (recent examples include foot and mouth, avian influenza, and Nipah virus) (Barlow 1996). Analogously, counterterrorism efforts often attempt to remove terrorists. This may be motivated by a sense of justice, to directly reduce threat, and/or to help reduce the spread of terrorist ideology. However, as implied previously, the mode of removing terrorists from the population might ironically enhance the spread of ideology through martyrdom and other factors. For instance, the killing of a terrorist might inspire susceptibles to adopt the very ideology counterterrorist operations aim to defuse. An SI equation that links death and transmission through martyrdom is

$$dI/I dt = S\beta(1 + \alpha m) - (\mu + \alpha + r),$$

and

$$R_0 = S\beta(1 + \alpha m)/(\mu + \alpha + r),$$

where the variables are as before, and m (martyrdom) is the extent that the death of a terrorist (resulting from terrorist activity) increases transmission to susceptibles.

We might then wish to explore how lethal removal of terrorists affects the spread of terrorism. The partial derivative of R_0 , with respect to α , indicates the slope of the relationship between removal and spread. This will be positive for

$$m > 1/(r + \mu).$$

This means that where m (martyrdom) is near zero, increasing terrorist death rate (such as through military intervention or suicide attacks) will always slow the spread of terrorist ideology. Nevertheless, with increasing m , r , or μ , an increase in terrorist death rate can increase the spread of terrorism. This makes obvious sense for m . Less intuitive is how a high background mortality or recovery rate increases the value of martyrdom. This occurs because when the lifespan of a terrorist (physical or ideological) is sufficiently short, that person's premature loss from the terrorist population has little cost to the spread of the ideology. Therefore, understanding the strength of martyrdom relative to recovery rates and background survivorship might allow better evaluation of counterterrorist strategies.

Control and Prevention

We have taken the first steps to determine the utility of basic ecological and epidemiological theory in understanding the spread of terrorist ideology. If terrorist ideology is analogous to an infectious agent, what insight can public health offer counterterrorism?

Preventative Control

Outbreaks of infectious agents are oftentimes apparent in the early stages, when very few individuals are infected. This is a direct result of the incentive that an infected individual receives for reporting their condition and thus receiving treatment. This, in turn, increases the likelihood that public health officials will learn of enough independent infectious cases to implement control initiatives before the rate of spread increases. Not surprisingly, this scenario is in stark contrast to the spread of terrorist ideology. Indeed, the very practice of terrorism and the ideologies upon which it is based demands a high level of covertness outside of the immediate cell. Relative to pathogen outbreaks, such behavior makes it exceedingly difficult to forecast and control emerging terrorist ideologies, though this is not to suggest a complete lack of indicators. Symptoms of an emerging terrorist ideology may include an increase in attacks, the death or capture of militants, or the dissemination of information (Stares and Yacoubian 2005). It is important to recognize, however, that the inception of infectiousness is not necessarily correlated with the appearance of symptoms. This level of unpredictability is why public health favors prevention, particularly vaccination, as the primary control strategy.

Vaccination works by exposing uninfected individuals to dead or attenuated infectious agents, after which the immune system goes through the process of building specific antibodies that then lead to immunity. Even vaccinating only a fraction of susceptibles in the population can reduce R_0 below 1 (a phenomenon called herd immunity). Vaccination is most viable as a broad-scale control strategy when used against well-known pathogens. An analogy to vaccination for terrorist ideology is exposing a population to a discredited version of the ideology to make it less likely that newly exposed individuals will find it attractive (Stares and Yacoubian 2005). An in-depth knowledge of a particular terrorist ideology would seem necessary for this approach to succeed.

Although vaccination is available for many of the pathogens that plague mankind, the evolutionary novelty and unpredictability of emerging infectious agents dampens its viability as a broad-scale control strategy. In the absence of vaccines for pathogens on the verge of an outbreak, public health officials generally favor two basic control practices: (1) isolation of

symptomatic individuals and (2) tracing and quarantining their contacts (Fraser et al. 2004). Both the implementation and success of these measures rely on a number of factors, ranging from the epidemiological characteristics of the infectious agent to the communication infrastructure of the public health agencies charged with control. Mathematical modeling of contemporary outbreaks (in particular SARS, HIV, smallpox, and influenza) suggests that the success of these control measures is equally dependent on the proportion of transmission that occurs prior to the onset of clinical symptoms (θ) and the inherent transmissibility of the infectious agent (R_0 as described in the previous section of this chapter) (see Fraser et al. 2004 for model details). Model simulations suggest that isolation, contact tracing, and quarantine are indeed sufficient to control outbreaks of infections when the values of θ and R_0 fall below a critical threshold. Control through isolation alone is possible when $\theta < 1/R_0$, but when $\theta > 1/R_0$, contact tracing is also required. When θ is very high, neither control measure (alone or in combination) is able to prevent the outbreak from progressing.

Similar to emerging infectious diseases, the early detection of new terrorist ideologies may aid control. Due to the covert nature of terrorism, early detection is also extremely difficult. In the first three years following 9/11, the U.S. counterterrorism strategy was to target operational terrorist cells, as opposed to their ideological motivations. Coined “operational counterterrorism,” the campaign monitored suspected terrorists, collaborators, supporters, and sympathizers and used the acquired intelligence to facilitate arrests (Gunaratna 2005). By targeting terrorists in the early stages of planning, operational counterterrorism reduced what may have resulted in longer-term terrorist activity (Gunaratna 2005). However, models of infectious pathogen outbreaks suggest that the success of counterterrorism will increase if detection occurs before the onset of the obvious indicators (attacks, media dissemination, etc.). As suggested by the pathogen outbreak model described above, if θ gets too high, even the most robust public health control measures become ineffective. There is reason to believe that the same would hold true for emerging terrorist ideologies.

Strategic counterterrorism seeks to reduce the political and operational space for terrorism to develop, spread, and sustain (Gunaratna 2005). The targets of strategic counterterrorism include the foundations on which a culture’s education, religion, media, legislation, and ideology are set. The mission is to counter terrorism in these very institutions by promoting an ethic against terrorist ideology that extends to the greater community. Because strategic counterterrorism is in many ways preemptive, susceptible individuals are likely to become infected with an antiterrorism agenda before they are ever exposed to a terrorist ideology.

As discussed earlier, unfavorable environments (in terms of justice, resources, or freedom) may promote the adoption of new terrorist ideologies by those suffering personal stress. Therefore, efforts that reduce the desire for change or provide alternative pathways for change might decrease susceptibility. Still, environmental conditions that decrease the susceptibility of individuals to a particular ideology will not necessarily prevent the spread of that ideology. For example, improved economic conditions might decrease desire for change in individuals, but also increase opportunities for communication or implementation of a terrorist ideology, thus making it difficult to predict the net effect.

The study of pathogen outbreaks and emerging infectious agents has grown in recent years. In the midst of fear factors such as avian influenza H₅N₁, we can expect the trend to continue. To date, however, scientists have found it extremely difficult to identify the forces that would allow public health officials to forecast the size or timing of pathogen outbreaks. Not surprisingly, this is because the ultimate dynamics of pathogen outbreaks are highly influenced by numerous factors that are in no way static: the social structure and immunology of the population, the epidemiological characteristics of the infectious agent, the environment, and contact rates between infecteds and susceptibles. Despite these difficulties, scientists are getting better at accounting for these factors and thus reducing the forecast envelope for the size and timing of pathogen outbreaks (Fraser et al. 2004; Hufnagel et al. 2004; Drake 2006). As progression in this field continues, we should be able to add even more dimensionality and depth to the infectious agent-terrorist ideology analogy.

Postestablishment Control

Thus far we have discussed the value of prevention as a tool to control the emergence and spread of infectious agents and, analogously, terrorist ideology. How do public health officials attack infectious agents that are already established, and are there counterterrorism analogues?

Efforts to reduce contact can be effective at slowing the spread of infectious agents when they have become established. Two general approaches are used. The first is to reduce contact rates between individuals irrespective of their infection status. Safe-sex and personal-hygiene campaigns are probably the best examples in modern society. For instance, children are taught to wash their hands, use toilets, and avoid spitting in public. The second approach is to specifically reduce contact between infected and uninfected individuals. In public schools, children with head lice are typically sent home to reduce the spread of lice to their classmates. For more serious infectious diseases, public health officials may quarantine infected individuals (and sometimes those that the infected individuals have

contacted) in an effort to stop spread (Fraser et al. 2004). Such an approach was used to reduce the spread of SARS. Limits on the movement of infected individuals can be difficult because detection is challenging. In the SARS epidemic, body temperature scans in airports (e.g., Hong Kong) were used to identify individuals with fevers, and these individuals were then subjected to medical examination. Recent epidemiological models are making progress in predicting the spread of pathogens such as SARS with evaluation of different control strategies (Hufnagel et al. 2004).

As mentioned earlier, controlling the spread of an infectious disease can occur through reducing contacts in general or isolating infected individuals. Limiting communication within a population might reduce the spread of a terrorist ideology but could also limit the spread of counterterrorist ideology, as well as increase resentment by denying expected freedoms of expression. Identifying and isolating individuals “infected” with terrorist ideology may be a more practical means of reducing spread. While covertness greatly impairs the ability to forecast and control emerging terrorist ideologies, detectable symptoms of an emerging terrorist ideology may include an increase in attacks, the death or capture of militants, or the dissemination of information (Stares and Yacoubian 2005). Active individuals might be identified via their own efforts to communicate their ideology or through identification of existing networks. Strategies presently used to reduce the ability of ideologues to contact others include imprisonment, military isolation, and disruption of communication, among others.

For some infectious agents (e.g., ebola, west Nile, rabies), contact with a reservoir host species drives transmission dynamics. Controlling such pathogens in peripheral hosts, such as humans, is greatly hindered because the pathogen is primarily supported in nonhuman hosts, which may be outside the reach of control efforts. Moreover, when the reservoir host suffers little pathology, there is no selection on the infectious agent to evolve reduced virulence. This is the case for many of the zoonotic infectious agents that spill over from wildlife reservoirs and cause high rates of morbidity and mortality in susceptible human populations.

An analog exists in the spread of terrorist ideology. Individuals that abstain from the practice of terrorism may still sustain the founding ideology, which they actively fund, harbor, and spread to recruit active terrorists. In doing so, they become reservoirs for the ideology. Like peripheral hosts, the individuals they recruit are more likely to exhibit the symptoms of the ideology (terrorist acts) than they are to spread the ideology or participate in recruitment activities. Decoupling terrorist recruitment from terrorist action makes it less likely that operational counterterrorism specifically directed against terrorists and their activities will reduce the spread of terrorism. In this scenario, the reservoir individuals harboring

the ideology would be undertargeted. Terrorist networks with an important reservoir might be better modeled as an infectious pathogen with a reservoir host, with counterterrorism actions targeted specifically against the reservoir.

Discussion

With this chapter, we have presented a framework and set of models as alternative means of conceptualizing the nature of terrorism. The epidemiological approach and tools presented here form the basis for understanding, controlling, and predicting the spread of infectious pathogens in human and wildlife populations. By drawing parallels between the spread of infectious agents and the spread of terrorist ideology, we have sought to highlight both the similarities and contrasts between these two threats. In so doing, we suggest how the vast body of knowledge in ecology and epidemiology may be modified and potentially applied to understanding terrorism.

In some cases, basic strategies for controlling infectious agents may translate directly to controlling terrorism. In particular, ideological analogs to R_0 could be used with coupled differential equations to predict the spread of terrorist ideology. As we have shown, the success of this approach is strongly dependent upon the variables used to construct the equations. Existing generic epidemiological models are not adequately suited for modeling terrorist ideology, just as they are not often adequately suited for modeling specific pathogens. Indeed, a good deal of critical thought will be required to construct adequate definitions for individuals that are susceptible to and infected with terrorist ideology, and for the model parameters that link these stages (e.g., transmission rate, death rate, and aggregation of the ideology, as well as contact rate between individuals). These variable definitions and associated assumptions would ideally be sufficiently analogous to those in the epidemiological models on which they are based without losing the social dimensions that make them inherently unique.

Simple epidemiological models could be customized for terrorist ideology by incorporating a sufficient amount of detail. Such model adaptation might consider the following factors. The types of infected individuals (terrorists) might be broken down into additional categories lacking analogues in epidemiological models (e.g., militants, educators, leaders). One might consider the possibility of vertical (parent-offspring) transmission of terrorist ideology. Unlike infectious agents that attack a host and, in turn, are attacked by the host's immune system, infectious ideologies may invade a host that already harbors an ideology. An existing ideology might resist invasion or it may be replaced or altered. It seems that models of terrorist

ideology would benefit from consideration of heterogeneities in human behavior relating to spread. It would be worth considering how to incorporate terrorist intensity but, like macroparasite models, identify a currency (e.g., attack rate) useful for counterterrorist goals. Once adequate models were identified, it would be possible to determine what sorts of data might be suitable for better tracking the spread of terrorist ideology. Moreover, there is much to be gained from the study of novel pathogen outbreaks and the models used to predict their timing, size, and geography. Collaboration between ecologists, epidemiologists, and social scientists has proven beneficial to the study of various human pathogens. Keeping with the analogy, such forms of cross-disciplinary collaboration might also benefit the study of terrorism.

Summary

Goals for future research include the following:

Develop working definitions and assumptions for the variables and parameters that characterize SIR models of terrorist ideology.

Determine how behavioral, sociological, and cultural parameters would be accounted for in these models.

Consider how the strength of these models vary when temporal (e.g., political cycles) and spatial (e.g., global terrorist networks vs. local terrorist cells) scales are accounted for.

Test the models against analytical data on the spread of ideologies.

The following questions remain open:

How will ecological and epidemiological models of infectious agent spread be modified to accommodate the diversity of terrorist ideologies?

What are the ethical considerations of comparing terrorist ideology to an infectious agent and, further, using this analogy to construct counterterror campaigns?

Are the strengths and benefits of the infectious agent–terrorist ideology analogy sufficient to warrant continued pursuit of its utility?

How might operational and strategic counterterrorism use insights from the infectious agent–terrorist ideology analogy?

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