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An Epidemiological Approach to Terrorism

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THESIS

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Abstract

There are many types of models for counterterrorism, explaining different problems that the military faces in the fight against terrorism. This thesis proposes that one of the fundamental assumptions underlying existing models of counterterrorism is that the struggle with terrorists can be understood as a war in the traditional sense of the term. We propose to rethink the struggle against terrorism as a fight against an infection. The epidemic of terrorist ideology within part of the world is a result, from this perspective, of the infectiousness of that ideology. Using the insights of the field of the epidemiology of ideas, this research looks into the models and methods used to understand and fight biological epidemics. We work with the SIR model from mathematical epidemiology, which partitions populations into susceptible, infected, and recovered categories, and apply that model with notional starting rates to the epidemic of terrorist ideology. This research allows another set of assumptions for models used in counterterrorism because the insights gained from viewing terrorism as a symptom of an epidemic can expand our understanding of the problem that we fight.

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Chapter 1: Introduction

1.1 Background

The problem of terrorism and how to best fight it has become a central question for the American military. There are large numbers of papers and books that examine the sources of terrorism and how things have gotten to the point that they have with the Global War on Terror. Every group of researchers takes their particular slant to considering how best to approach this fight, and they have developed a lot of specific systems and models, all with the intent to somehow help along the fight that the United States is engaged in against transnational terrorist groups.

Within the operational research arena, many models exist that purport to allocate resources, develop strategies, and otherwise solve problems that plague the war on terror. These models engage diverse problem-solving approaches, from linear programming to simulations, but all of them seem to rely on certain fundamental presuppositions about the nature of the fight against terrorism. Even the terminology of the title of the struggle, the Global War on Terror, highlights the underpinning assumption that the United States is engaged in a struggle with terrorists that can be understood as a war.

War is defined as "a state of usually open and declared armed hostile conflict between states or nations . . . the art or science of warfare . . . a state of hostility, conflict, or antagonism . . . [or] a struggle or competition between opposing forces or for a particular end" by the Merriam-Webster Online Dictionary (http://www.m-

w.com/dictionary/war). The United States military has evolved many different strategies and doctrines that allow it to fight in wars—these have changed over time, related often to the particular conflicts that the American military has been involved in. One of the most strongly influential military philosophers who has influenced Western definitions of war was Carl von Clausewitz. His theory has been invoked, especially since the Vietnam War, as the call to principled, decisive, and rational action in warfare. (Fleming, 2004: 62-63)

Following the philosophical trail blazed by Clausewitz, much of Western thought has centered around the idea that within conflicts, rational objectives are desired on both sides and rational actions stem from those objectives. Because of this, many models of conflict have been created that include game theory and its assumptions of rational action on the part of both "players" in a conflict. However, reason is a hallmark of Western thought, and not something that is universally admired or sought after in other cultures. The fundamentalist Muslim would likely see studied rational self-preserving action, in warfare, as being something intrinsically antithetical to his or her values.

Western thought about war and the optimal strategies in which to fight war tend to concentrate on optimizing the effects of one side's weapons strikes on the other side. To this end, much inquiry has been made into centers of gravity, the idea that one can maximize the harm to the other side by choosing one's targets for their strategic and economic value to the other side in the conflict. The assumption is that, once one side experiences enough loss of manpower and infrastructure, that side will have no other option but to surrender and end the conflict. This underlying assumption also contributes

to models of rational decision-making in game theory. However, these assumptions that people operate in a purely rational manner is fundamentally Western in nature. The actions of terrorists and other such "fanatics" tend to defy rational explanation—in no game-theoretic model would the behavior of a suicide bomber maximize the expected payoff for that bomber.

In the article "Al Qaeda's Fantasy Ideology", author Lee Harris describes the tendency of Americans to see the struggle against terrorists as a traditional war as follows:

> "This common identification of 9-11 as an act of war arises from a deeper unquestioned assumption. . . . The assumption is this: An act of violence on the magnitude of 9-11 can only have been intended to further some kind of *political* objective. What this political objective might be, or whether it is worthwhile—these are all secondary considerations; but surely people do not commit such acts unless they are trying to achieve some kind of recognizably political purpose.
>
> "Behind this shared assumption stands the figure of Clausewitz and his famous definition of war as politics carried out by other means. The whole point of war, on this reading, is to get other people to do what we want them to do: It is an effort to make others adopt our policies and/or to further our interests. Clausewitzian war, in short, is rational and instrumental. It is the attempt to bring about a new state of affairs through the artful combination of violence *and* the promise to cease violence if certain political objectives are met." (Harris, 2002: 2)

The military understanding of the word “war” carries with it expectations of a defined struggle with opponents who have a clear objective—to do harm to us. We approach our problem solving, then, with the expectation that our opponents, the terrorists, have the Clausewitzian goal of maximizing the damage that they can do to American interests both overseas and here in the homeland. The problem with this assumption—which is evident in most of the models of counterterrorist applications—is that it has not been supported by the actions of terrorists. Terrorists and their greater transnational networks have not behaved in a manner that one could predict with a Clausewitzian understanding of the nature of the opponent.

Recent analyses of terrorist actions, particularly the troublesome transnational Islamist terrorist groups, have highlighted this fact. Even though they have the resources to strike us, and the clear example of ways to maximize panic and chaos through the American public, these groups have not chosen to do so. As much time and resources have been put into the problem of divesting terrorist groups of training facilities, recruits, weapons and monetary resources, it has been a difficult problem to cut these resources off entirely. Some groups such as Hamas and Hezbollah have turned to South American drug trafficking in order to fund their groups, and other transnational terrorist groups have likely done the same. Countries such as North Korea and China, traditionally antagonistic to the Western world and the United States in particular, have shown no compunction about trading in weapons with this area of the world, leading to an understanding that the groups who want these resources have the ability to obtain them. Thus it would be foolish to underestimate the resources at the hands of the terrorist

groups—but one would expect, if these groups were seeking to maximize damage and chaos, that they would have leveraged these resources against the United States in a way that we have not seen since 2001.

Additionally, terrorists who have paid attention the current events and the American media would have had ample proof for what would sow the seeds of chaos and panic through the United States. The anthrax scare after September 11th, 2001 and its resulting disruption of the country's mail could easily have been continued and leveraged to create even more panic and discord. The media has fed national audiences with scenes of biological, chemical, and nuclear threats, and any terrorist keeping track of made-for-TV movies since the attacks in New York and Washington DC would have an idea how to disrupt the American public. Because terrorist groups have not acted in such a way as to maximize terror and chaos in the American public and among Americans overseas, it does not make sense to base models on the assumption that they are doing so.

In order to reassess our assumptions about the goals of our opponents in the war on terror, we need to first understand our opponents. Next we need to look at their actions and the forces at work in their environment so that we can understand better how to fight them. For the purposes of this paper, we will consider transnational Islamist terrorist groups, largely centered in the Middle East.

If the terrorist attacks were not motivated politically or in a utilitarian way, and we of the Western world—particularly within the United States—are not going to have a good grasp of the cultural factors going on to create ripe conditions for terrorist groups, how are we to fight against attacks made on our people? We cannot fight the last war—in

this case the Cold War, a war of ideologies—because it's familiar. While we might fight with those means and methods, we are going to continue to chase down a shadow opponent and become bogged down in unpopular nation-building and guerilla warfare as we have already within the nations of Afghanistan and Iraq. It is evident that something in our presuppositions needs to change, in order that we will be able to meet this conflict head-on. Then what is the problem we are looking for, and what is a good solution for that problem?

1.2 Problem Statement and Approach

With an understanding of the roots of terrorism as a background for our understanding of the Global War on Terror, it becomes evident that the forces at work in the world that have caused our current state of war are extremely complex and entangled. The actions that terrorists take continue to baffle us because we keep expecting them to behave in a Western way, to seek to maximize the damage that they can do to us and to sow chaos and panic among Americans both at home and abroad. If the war on terror is a war in the classical Western sense, then it is not a war that transnational Islamist terrorist groups are fighting against us.

With this in mind, then, we need to readdress the assumptions upon which we are basing our research into systems, strategies, and problem solving for the war on terror. While we may still need to treat this as a struggle where we seek the greatest disruption of their networks for the least cost on our part, we know that their goals are not similarly

expressed and realized. Terrorism is not so much the tactic of a great army that seeks to bring down the Western world, even if that might be part of the side effects of their greater struggles. Instead, terrorism—particularly that directed at Western peoples—is a symptom of greater cultural currents of change. Islamist terrorism, then, could be classed as a cultural malaise, a sickness that comes about as a result of the environment surrounding people who are in the midst of great transition. Instead of having a valid cultural model for building a solid civilization for the future, people in the Middle East are falling prey to an infectious worldview that sees the Western world as being to blame for the problems of the Middle East in particular, and seeks to do harm to the Western world in large symbolic gestures such as the attacks in New York and Washington DC.

Then it becomes apparent that the most useful framework from which to consider terrorism is not that of a war against an organized opponent, but instead as a struggle to eradicate a disease. To see terrorism, and the mindset that propagates terrorism, as an epidemic would mean that we would have to reassess all of our strategies and many of our models as we work to solve problems and end the Global War on Terror. Our need to develop strategies to fight the war on terror would be better served to see the ideology of terrorists as a virulent disease that is spreading through susceptible areas as an epidemic.

Within the business world, particularly in the study of marketing, the notion of ideas as being infectious agents has become increasingly widespread over the past two or three decades. This notion encourages marketers to optimize the infectiousness of the ideas that they are promoting by adjusting the factors that go into spreading the social epidemic. This strategy has shown extraordinarily good results for many marketers, and

illustrates the benefits of considering ideas to be contagious like viruses or other infectious diseases.

As we consider the state of the Middle East at present, it becomes apparent that all of the factors necessary to spread the ideology of terrorism are present. In one sense, this is a mindset that is being marketed to a civilization, and the Western world stands to face more opposition if, indeed, the Islamic world does reach a tipping point where the ideology of terrorism becomes mainstream. It would be to our benefit within the United States to study the factors involved and find a way to do something to fend off the tipping point that could happen to turn the Islamic world as a whole against the Western world.

The study of infection and epidemics in a purely physical sense is not merely a descriptive idea, however. While the epidemiology of ideas has mainly been an area researched by people who want to market new products or ideas, there are greater applications available in the understanding of classical epidemiology. One of the first things that would be useful to consider is the SIR model, a model of the percentages of the population that are susceptible, infected or recovered from a specific epidemic. This model has been used to study the nature of diseases and to put together approaches for combating those diseases. The second part of epidemiology that would be applicable is the idea of a strategy for the eradication of a disease that causes epidemics. These have been put together with greater and lesser success depending on the nature of the epidemic involved, but both polio and smallpox have been successfully treated and removed from most of the world with these strategies.

The mathematics of SIR models is complex and fills volumes of textbooks and current research. Deterministic, probabilistic, and blended approaches are available, and the factors that are tracked in the making of these models changes from one epidemic to another. However, from these models we can see that if one understands the nature of the epidemic that one is working with, one will have a better chance of being able to keep it from getting out of hand. These models should work descriptively as well as being prognostics, and should allow us to see the points at which intervention is needed. Just as a physical epidemic can get out of hand if people are not working to track its progress and developing strategies to contain it, this epidemic of an ideology of terrorism could become out of hand if it is not being tracked closely and modeled in this way.

The susceptible population for the terrorist message has been pinpointed by much of the media and analysis available—it is largely the disaffected youth of the Middle Eastern region. What becomes more complex is discovering which of the people in this population are susceptible to or infected by the ideology of terrorism. Additionally, those who are recovered or immune from the ideology of terrorism need to be studied, in order that we have a better idea of what to look to do in order to "inoculate" people against terrorism.

At the heart of a strategy to eradicate terrorist ideology is the need for charismatic leaders with a clear vision for the Middle Eastern region who have the clout and the will to lead their people into the future without terrorism. Additionally, the Western world's insistence on standing behind leaders who do not have the interests of the larger region at heart has complicated the process. Part of the change that Western nations will need to

make is to understand that the modernization of the Islamic world will most likely not mean its Westernization, and that our best approach to encouraging a world free of the threat of terrorism from these transnational Islamist terrorist groups is to understand that we cannot make the entire world into a copy of our own civilization. Backing charismatic leaders who may not be in favor of Westernization but who are not anti-Western is more likely to be a successful measure than trying to force Western ideals into a different civilization. The key to finding a successful "inoculation" against the terrorist mentality is to look past our own cultural norms to see what the valid replacement ideologies are for the susceptible people.

The fact that the current Clausewitzian paradigm of Western nations continues to be used as a set of presuppositions for model-building and problem-solving in the Global War on Terror, particularly in political and military circles, would seem to indicate that this paradigm works. However, the current situation in the Middle East, with the United States military stretched out and working nation-building tasks as well as fighting the ongoing war on terror, would seem to indicate otherwise. If we are fighting a Clausewitzian battle, we should be seeing political and utilitarian results. Instead, we fight people who continue to fight us even after any rational model (by Western standards) would indicate that they surrender and cut their losses. Even though we have demonstrated superior firepower and the will to defend ourselves, we continue to encounter threats of action. These are not Clausewitzian strategies that our opponents are using, and by modeling them as such, we do only ourselves a disservice.

Instead, we need to look at the aspects of the terrorist message, as well as the context in which it is being relayed and the people who are relaying it, and check the message for its infectiousness. If we accept the spread of terrorist ideology within the Middle Eastern region as a social epidemic, then we can start to use the tools developed within epidemiology to characterize the epidemic and to work out strategies to fend off a lethal epidemic that spirals out of control. Much as epidemiologists develop methods to defend against a possible outbreak of SARS or the Asian bird flu, we need to be working on methods to control the spread and, optimally, to eradicate the disease of terrorism.

Once this switch has been made, models for counterterrorism, particularly those that work with resource allocation and optimal stopping, may well be changed. The expected results of given strategies would have to be recalibrated, and as parallels are made between the spread of infectious ideas and the spread of viruses, we will be better able to understand what it is about the terrorist mentality that infects the susceptible population. Additionally, we may be able to leverage marketing strategies to do some spreading of a different message, one that is not hostile to Americans, so that the same susceptible population has a chance to be "inoculated" against the terrorist mentality with another set of ideas about how to fix the problems of the present.

Switching our assumptions to reflect a change in our conception of terrorism from a political and utilitarian war of ideologies to a struggle to eradicate—or at least to slow—a social epidemic would have both positive and negative consequences. The negative consequences are primarily centered on the costs of switching to a new framework, because change means expense and difficulty. However, the positive

consequences of that type of switch would seem to outweigh the immediate problems the switch would entail.

Any fundamental shift in assumptions usually means an initial outlay of costs in order to update existing models and strategies to work with the new assumptions. It is possible that many of the legacy approaches to modeling counterterrorist activities and developing strategies to limit terrorist activities would be unable to be salvaged and thus would have to be replaced with newer tactics and strategies. Some of the models that we use to deal with the war on terror might become out of date and need replacement, and this would mean time and allocation of resources. In the short term, it could be costly.

However, with a longer view, the costliness of switching to new assumptions is overshadowed by the potential benefit of making that switch. Our frustrations with the ongoing struggle against terrorists are ample evidence that fighting a utilitarian war with these groups is not going to work. Much as the military has always done, we are attempting to fight the last war in the present one. We get bogged down in the details of rebuilding all the nations that we have had to fight, and the military continues to be deployed to more and more countries around the world. It would appear that the current paradigm is headed for a breakdown. If that happens, it could be disastrous, and switching to thinking of this fight as the fight to contain and eventually eradicate a potentially lethal epidemic that is threatening to spiral out of control is one mental shift that could help to turn things around.

Applying the concepts of epidemiology to the spread of ideas is not a new notion, but it has been applied at present in only a limited fashion. The bulk of the application

seems to be in the realm of marketing, where advertisers seek to leverage the power of person-to-person communication instead of relying on unpredictable and increasingly ineffective mass marketing techniques. We have a good basis for a descriptive application of epidemiology to the spread of the ideology of terrorism.

On the other hand, very little exists that attempts to apply the notion of disease control and/or eradication to the spread of ideas. Much of this is because at present the research into epidemics of ideas centers on ideas that people desire to spread. However, as we consider ideas and mindsets that we do not want to spread—those that make people likely to become terrorists—we will need to seek out the way in which epidemiological strategies can be applied to a purely ideas-based context.

1.3 Scope of Research

This study is a development of the idea of how to change the assumptions inherent in our models of terrorism and counterterrorism. Using the models and methodologies that exist within the field of epidemiology, the following chapters develop the notion that terrorist ideologies are an epidemic of ideas. At present, the numbers available are only notional; while a wealth of studies have been written about fighting terrorism as a war, and about how to model and counter biological epidemics, little besides marketing books has been written about epidemics of ideas.

Using a new perspective on terrorism, as an epidemic rather than a war, encourages a reframing of the questions at the heart of the study of the War on Terror. This study takes this notion and makes an initial application of the wealth of models and information within the field of epidemiology to the notion of countering terrorism. While it is still an initial application of a new idea, it has promise as a new paradigm for the fight against terrorists, especially considering the fact that most of the existing approaches to counterterrorism have been less than optimal in their results. Eventually, further studies should be conducted to continue with this idea, so that meaningful numbers can be developed, and more accurate models can be built. This will take the cooperation of people within epidemiological and social sciences realms. From that point, the models can be used as baselines for optimization problems, including resource allocation, network flows, and other types of questions about the best way to work against the propagation of terrorist notions.

1.4 Thesis Outline

This thesis continues in Chapter 2 to evaluate the literature that is relevant to the topic. The literature review includes looks at current techniques used to study terrorism, as well as at the roots of terrorism as they are currently understood. The review covers the models that are used in the field of epidemiology and considers what has been written about epidemic eradication strategies. In Chapter 3, the methodology of epidemiology is explained and illustrated, particularly through different applications of mathematical

models and discussion of eradication strategies. Chapter 4 goes on to apply the models and strategies described in Chapter 3 to the particular idea of terrorist ideology as an epidemic. Finally, Chapter 5 looks at the conclusions one can make based on the analysis in Chapter 4, and details the directions that further research could take.

Chapter 2: Literature Review

2.1 Approaches to the problem of terrorism

Now that fighting terrorism, particularly Islamist extremist terrorism, has become a priority for the American military, much has been written about the best ways in which to do so. A large portion of the literature on the subject focuses on resource allocation for counterterrorism. Hanes (2005) and Mitchell and Decker (2004) approach this problem with similar approaches. Also along this line of thought, Pruitt (2003) looks at prioritizing the factors involved in homeland security, applying a value-focused thinking approach to the problem. The General Accounting Office's report on Combating Terrorism (2002) discusses recommended actions for antiterrorism at services' installations.

The Advisory Panel to Address Domestic Response Capabilities for Terrorism Involving Weapons of Mass Destruction (2003) takes a more specialized look at the domestic protection problem, setting priorities especially centered around the threat of WMD used within the homeland. Another specialized approach comes from Yao and Edmunds, et al. (2003), who consider optimal resource allocation in electrical network defense. Again, it takes a budgeting, risk-management inspired approach, as do many of the other papers on the subject. Much of the literature, indeed, seems to focus on the aspect of counterterrorism that exists within the homeland, in the sense of managing risk and optimizing systems for the lowest risk levels. In these estimates, one allocates

resources as a response to terrorist actions, or to prevent the occurrence of such actions on domestic soil. While such an approach has value, especially in setting priorities between competing options, it can also be myopic, because it ignores the side of counterterrorism that involves action overseas.

Horowitz and Yaimes (2002) also take a risk-based approach to modeling actions that relate to counterterrorism. Their analysis involves the risks associated with intelligence gathering and then works through building scenarios which are then assessed for their risk factors. Much of this centers around Bayesian analysis of the probabilities of certain events, aggregated over decision trees that are developed from the scenarios.

Another approach to the problem of terrorism centers on the idea of applying Social Network Analysis (SNA) to terrorist networks, modeling influence factors. Renfro (2003) takes this type of approach to model the social network within the Iranian government, showing that when one works through the factors involved, the most obvious pressure points are not, after all, the most influential nodes in the influence network. Rosen and Smith (1996a, 1996b, and 1996c) work through the methodology of influence net modeling, using Bayesian analysis of probabilities and a computer approach to aggregating probabilities in order to detect sensitivity and pressure points within the influence networks. Modeling social networks of terrorists has great promise, but within the applications of SNA, there is so little consensus on the appropriate quantification of relationships, and what has been done tends to be somewhat arbitrary. Social networks have been used by social scientists to describe networks of individuals, but have not been used for mathematical modeling of the networks to any great extent so far.

Some authors approach the question of counterterrorism by modeling it in a game-theoretic mode. Mia Bloom (2004) discusses the factors that make suicide terror a "rational choice" when a person or group considers options for competition or conflict. Yaimes and Horowitz (2004) create a model based on game theory for counterterrorism intelligence analysis. It incorporates some of the same Bayesian risk analysis that some of the papers mentioned previously have used, but puts that analysis into a game model instead of an influence network.

Another perspective on counterterrorism can be found in an analysis of terrorist organizations' centers of gravity. Schweitzer (2003) does this with Al-Qaeda, discussing the foundational pillars of their brand of extremist Islamist ideology. His particular focus is on what makes the ideology possible, and how an information campaign could adequately "attack" that center of gravity. This type of analysis is useful, if still influenced by its own Western perspective. As the different models of terrorism and counterterrorism show, a better understanding of the problems at the core of terrorism is needed in order for those who want to counter the problem to make any progress.

2.2 Roots of terrorism

Much of the literature on the subject of the roots of terrorism suggests that the current dire economic outlook for the youth of the Middle East and other factors such as hunger and repressive regimes are largely to blame for the grassroots support for terrorist groups. Blomberg, et al. (2004) make their model of terrorism using these assumptions,

setting it up as an economic model. Garfinkel (2003) works off the assumption that economic factors are the motivating forces behind terrorism, modeling probabilities of terrorist action based on this assumption. This sort of thinking is facilely Western in nature, however, and oversimplifies a much more complex set of causes and effects. Politics and economics certainly play a part in the larger picture of what is causing terrorism to escalate, especially among groups with roots in the Middle East. However, if these were the only causes, we would expect to see similar situations among the rest of the world's poor and downtrodden people. And this we have not seen at present. Thus it would appear that a belief that terrorism can be eradicated by simply remedying the political and economic conditions within these countries is oversimplified.

Some authors see the roots of terrorism as being economic in nature, while others perceive them as something psychological in nature. Borum (2003) discusses the development of an extremist mindset, discussing the steps that take place as such a mindset is formed. He goes through the basic four stage model that justifies terrorist acts of violence. First, the person or group identifies an undesirable event or condition, then they frame it as an injustice, then blame some target for the existence of that event or condition, and last vilify or demonize the scapegoat. (Borum, 2003: 7-8) Michael J. Mazarr (2004) provides a similar analysis of the psychological factors at work that cause Islamic terrorism. His analysis, though, is rooted in an understanding of the current cultural predicament of the standard Muslim in a modernized world. His article concludes with a series of unconventional recommendations to propagate a "strategy of identity entrepreneurs" (Mazarr, 2004: 16)

Benjamin and Simon (2003) discuss the history behind groups such as Al-Qaeda and other extremist Islamist organizations, detailing the philosophical lineage of current movements. They work through the immediate history surrounding the September, 2001 attacks, and link the current phenomenon of radical Islamism to its historical precedents and ancestors. In *The Crisis of Islam* (2003), Bernard Lewis discusses the history behind radical Islamist terrorism, but he also considers the cultural factors at work that make terrorism seem a viable alternative to these groups when they are incomprehensible to Westerners. In particular, he focuses on the fact that the nature of Islam has always been that of a unified church and state, which makes governments and revolutions look extremely different than their Western cousins. These authors show that there is something fundamentally different at the heart of the Islamic world that Westerners do not understand. That misunderstanding, as well, creates the opportunity for differences to inflame the existing rift between the civilizations.

Another perspective on the forces at work that have caused the current clash between the Western world and some of the movements within the Islamic world is incorporated in Samuel Huntington's *Clash of Civilizations* (1996, 1993). Within this book, Huntington explores the notion that the nature of war has been changing throughout history, and that currently we are in the midst of a transition between the wars of ideology that were best exemplified by the Cold War and wars between civilizations. His definition of a civilization encompasses the things that bind together a culture such as religion, philosophy, shared history, and other similar factors. The Western world, with its Christian roots, forms one such civilization, the Islamic world another, the Orthodox

cultures of Russia and Eastern Eurasia another, and the Chinese yet another of these civilizations.

Huntington points out that the fault lines of battle exist now, in a macroscopic scale, on the borders of these civilizations, and in a microscopic scale, the clashes are happening in places where the civilizations intersect, such as the Balkans, the division between Hindu and Muslim peoples that one sees in the Kashmiri region, and the ongoing struggles in Northern Africa. As globalization marches on, the forces of modernization are becoming increasingly dissociated from the force of Westernization, and countries that are increasing their technological capabilities are starting to turn to their own cultural roots as they seek the way to approach their futures with the new resources at their disposal. The cultural dominance that the Western world has traditionally held over developed countries, then, is waning, and more of the people within other civilizations are expressing their discontent with Western norms.

We in the West still think that the goal of extending our cultural values of freedom, democracy and free market economics is something that other countries should embrace as well, and have incorporated this type of thinking into our rebuilding of the countries of Afghanistan and Iraq. However, it is likely that the people of another civilization see this influence as meddlesome and intrusive, and seen from that light, the increasing rumbles of discontent with the ongoing American presence in the Middle East can be understood, if not remedied. Huntington makes the point that the divisions between civilizations are likely only to grow more profound as the rest of the world continues to modernize and become wealthier. Thus it appears that approaching the war

on terror and expecting to rebuild the Middle East into a free-market democratic (in other words, Western) region is impractical. Expecting that the forces of modernization will do so naturally is equally problematic.

If, then, we have reached a point in history where the Middle East is ripe for change and is not looking to the Western world for guidance in that change, where will the region look? The Islamic world, deep in the throes of modernization, is ripe to be led by visionary leaders who have a distinct path for the future mapped out. Some of the factors that make this region ripe for visionaries include the large numbers of disenfranchised youth, the return to the cultural roots of the region, and the psychological need for a narrative that can be provided by a leader who understands the region and the forces at work within it.

As Harris (2002) points out in "Al Qaeda's Fantasy Ideology," much of what motivated the terrorist attacks of 9-11 was not political or utilitarian in nature. Instead, America was used as a prop in the greater story that these leaders were telling to their followers. The bringing down of the Twin Towers was, in effect, an act of high drama, a David bringing down Goliath situation. Instead of calibrating the attack for the effect it would have on America—whether the leaders of the country or the people within the country—the attacks were intended as a sweeping gesture that would capture the hearts and imaginations of the people of the Middle East, who were looking for something to believe in, since their immediate circumstances have been so hard to deal with.

2.3 Epidemiology of Ideas

Thinking of a mindset, of a particular idea or ideology as an epidemic is by no means a new concept. In the book *The Tipping Point* (Gladwell, 2000), the author goes over much of the research that has been done over the past few decades into trend research and how ideas spread between people. Many other books have been written about the concept of "viral marketing" (creating word-of-mouth marketing that spreads between people as a virus would), permission marketing (Godin, 1999) and other such approaches to marketing. In a world that is oversaturated with advertisements, the notion of leveraging the infectious nature of new ideas between people has revolutionized the arena of marketing. Emanuel Rosen (2000) discusses *The Anatomy of Buzz*, and the notion of how one creates word-of-mouth marketing, detailing the strategies and the principles behind doing this. Al and Laura Ries (2002) make a similar point in their book, detailing the need to make ideas infectious on a person-to-person level. Seth Goden (2002) discusses the need for novelty and remarkableness in marketing—essentially a blueprint for making an idea or the conveyance of an idea more "sticky" or infectious.

The typical breakdown of the factors that make an idea spread are the people who spread ideas, the nature of the message that conveys the idea, and the context of the message delivered. Once these factors are leveraged properly, an idea can be disseminated to enough people, remembered by them, and there will come a critical time—a tipping point—at which the idea will have reached saturation point and a trend will result or action will be taken. Trend research is the most innocuous of the

applications of this notion; taken further, one can see that fomenting of revolutions and coups often happen in the same way.

Gladwell speaks of the people who are instrumental in the spread of an infectious message in a chapter called “The Law of the Few”. It is important to note that as one considers the spread of ideas, there are relatively few people needed to make an ideological epidemic take hold. The three specific types of people involved are what he calls Connectors, Mavens, and Salesmen.

Connectors are people who have an exorbitant number of social contacts, due to factors including personality and their jobs. These people have larger numbers of friends and acquaintances than the average person, and keep up with their social networks. Because of this, their influence is spread over a much greater portion of the population. If the message in question gets to a Connector, it will have a better chance of reaching the number of people that it needs to in order to take hold and become an epidemic.

The next type of person who spreads a social epidemic (or ideological epidemic) is what Gladwell calls a Maven. These people are well-schooled in their areas of expertise, and because they know what they are talking about, people that they know will listen to their advice and recommendations for products. If a good product reaches a Maven, he or she will relay the message, and his or her recommendation carries more weight with other people than a remark from any other person.

Salesmen are the final type of people that Gladwell profiles as instrumental in the spreading of social epidemics. These people can move ideas farther simply by nature of their charm and persuasiveness. When convinced that they have a good idea that they

would like to recommend to others, these people have the resources to persuade those they speak to. In this sense, a Salesman will have a greater chance of making a message take hold when they speak to one of the people that they know, or even when they speak to people that they encounter by chance.

Connectors, Mavens and Salesmen are the people who make social epidemics start to spread. These people are instrumental in the beginning of an ideological epidemic. It is clear that within the Islamic world, people such as Osama bin Laden may be Salesmen, where there are imams within mosques who are Mavens, and other Muslims who are Connectors, able to spread these ideas from one locale to another. However, while the people involved in an epidemic of ideas are instrumental, the idea being conveyed within an epidemic—the message—is pivotal as well.

The important aspect of making a message work is in its packaging—a message needs to be "sticky" to be able to start a social epidemic. Gladwell highlights that while the need to make a message sticky is irrefutable, the challenge of what exactly will make the message sticky is much more complex. Marketers have been working with this difficulty as long as advertising has been used to sell products, and there is no hard and fast rule that always works. What Gladwell points out is that an adaptive and innovative approach at packaging a message that one wants to spread is what is necessary to make a message sticky in the first place, and then to continue to make it sticky. From the success of transnational terrorist groups within the Middle East to recruit and spread their message to the people of their region, it would appear that they are working on the stickiness of the messages that they wish to convey. Thus the nature of the message itself

is important as one considers its ability to start a social epidemic, but it is also important to keep in mind the context within which the message is being conveyed.

The context of the message that one wants to start a social epidemic is as important as the message itself and the people who convey it. People are greatly influenced by their environments, as Malcolm Gladwell points out, and the environment within which one experiences a message is as instrumental in its ability to cause an epidemic of ideas as the message itself or who one hears the message from. This consideration leads Gladwell into an overview of sociological research into group behavior and the different ways that people are able and willing to behave given different situations. Additionally, he discusses how there is a magic number—150—that seems to be the typical size of a real social network for any person. If a group smaller than that number attaches itself to a particular idea, most of its members will go along, for community spirit, while in a larger group, people start to become divided and alienated. The cell structure of most terrorist groups and the inherently local aspect of much of what these groups do caters to this need for an appropriate context for the spreading of the message and the continuing of the social epidemic that is the ideology of terrorism.

The other branch of the study of epidemics of ideas is the study of memetics. This somewhat controversial field of study looks at idea units—memes—as being in essence viruses of the mind, infectious agents that propagate through populations much as epidemics vector through populations. Aunger (2002) discusses this phenomenon as it occurs culturally, while Chilton (2005) discusses a particular case of ideas as viruses, as conveyed in the text *Mein Kampf*. While the field of memetics seems largely to be

philosophical and esoteric in nature, the basic central tenet of idea-units that are contagious much as viruses are contagious is a valuable insight. Specifically, this insight is how a particular meme or set of memes, like Islamist extremism, might be rightly thought of as an epidemic of ideas or a social epidemic that is raging through a part of the world.

Thus as we consider what may move terrorism from being a containable problem to a full-fledged epidemic within the Middle East, we need to track these factors. The people who are spreading the ideology of terrorism, the packaging of the message of terrorist groups, and the context within which this message is being spread are all important pieces of the puzzle of which to take note. Marketers within the Western world have been able to leverage these factors very effectively as they work on word-of-mouth or "viral marketing", and their success should give us an example of how this sort of process works. Understanding the cultural influences that make the "market" different in the Islamic world will be pivotal for an understanding of the possible success or failure of a terrorist epidemic, but it is something that needs to be tracked carefully.

2.4 SIR models: Deterministic Modeling of Infectious Diseases

The SIR model is one of a set of tools used by epidemiologists to understand the nature of a particular epidemic. The letters of the acronym stand for susceptible, infected and recovered, and refer to numbers of people within any given population that fall into one of those four categories in reference to the epidemic. A typical SIR model (shown

over time) can be seen in the figure below. This is a useful tool for tracking the progress of an epidemic, as well as to see the progress of the infection itself, with such characteristics as latency before symptoms, virulence of the infection, and other qualities. (Trottier and Philippe, 2001)

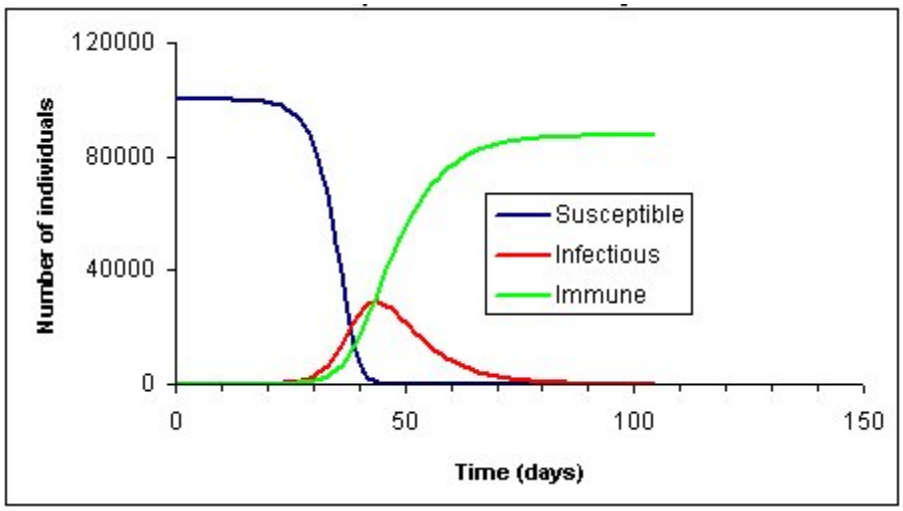


Figure 1: SIR model of measles epidemic

http://www.ispub.com/ostia/index.php?xmlFilePath=journals/ijid/vol1n2/model.xml

A SIR model, or other compartmentalized deterministic model of an epidemic, is based on modeling the rates involved in transmission of the disease, contact between members in the population, population growth, and rates of recovery. Hethcote (2000) discusses the mathematics involved in standard and more involved SIR models. Trottier and Phillippe (2001, 2002, and 2003) discuss deterministic modeling, such as SIR models, and the type of sensitivity analyses and repercussions of such tactics as immunization at birth. Li et al. (2001) discuss the relevance of adding vertical transmission into deterministic models, and show the resulting changes that occur within

the differential equations that make up the core of the deterministic model. The mathematics of these models are further explored and explained in Chapter 3.

Modeling an epidemic is a useful thing to do in order to gain insight into the particular factors that make up that epidemic. Some diseases lend themselves, more than others, to being targeted for elimination from the global community. One of the ways to measure an epidemic's potential for an elimination strategy is to look at the rates defined in the deterministic model (Kretzschmar et al, 2004). Once a meaningful model for the epidemic of terrorist ideology has been developed, it is likely that these rates could be defined.

2.5 Infectious Disease Eradication Strategies

Another aspect of epidemiology that has potential for application to the struggle against terrorism is the notion of a strategy for the eradication of a disease. Many of these have happened over time, with the most famous being the real eradication of smallpox. In the public health community, there is a distinction between the elimination of a disease (or elimination of infections) and the eradication of a disease. Elimination of a disease is defined as a reduction to zero of disease incidence in a particular geographic region as a result of deliberate efforts, while eradication is considered a permanent reduction to zero on a global scale of the incidence of infections. (Dowdle, 1999: 23)

There are two types of eradication strategies applied to infectious diseases, mass vaccination and ring vaccination. (Kretzschmar et al, 2004: 832) The original plan to

eradicate smallpox was with mass immunization, which worked for much of the developed world, where there were the resources to do that, but when the effort spread to the developing world, it was not feasible to immunize the entire population of these areas. Instead, a new strategy was developed. When a new case of smallpox was discovered, the person would be isolated, as would the people who lived within a certain radius of that person. Additionally, house-to-house searches would be made in a wider radius in order to track the progress of the epidemic. All the people within the smaller radius would be immunized and tagged as potential outbreaks, and their names would be removed from the list of these once the incubation period for the illness had passed. Using this ring vaccination strategy, the people fighting to eradicate smallpox were able to concentrate resources and effort in more focused doses, and better able to allocate what they had available to the necessary places. In this way, a better smallpox elimination strategy was developed, and this eventually allowed health workers to eradicate the disease. Fenner (2002) discusses the particular features of smallpox, both biological and sociopolitical, that made it a good candidate for an elimination strategy such as was developed. At present, the world health community is concentrating efforts such as these to eliminate poliomyelitis. Joyner and Rogers (2001) and Aylward et al. (1999) discuss this effort and the particular characteristics of the poliovirus that render it a good candidate for elimination.

If the ideology behind terrorism is an epidemic that has the potential to spread through large portions of the Islamic world, then we need to be able to not only know how it spreads, but also have a feel for what we can do to limit the spread of the epidemic

and, optimally, to eradicate that epidemic. With this goal in mind, it becomes apparent that more than marketing products could result from a careful application of epidemiological concepts to the question of the spread of the terrorist ideology. If we were to gain a good picture of the nature of the infection, using a SIR-type model and other characteristics of the ideology, and we were able to use that information to build a strategy for the eradication of the epidemic, we might have success in the fluid war on terror. Then we need to consider where the parallels between the terrorist ideology and a physical epidemic exist.

2.6 Summary

The question of how best to deal with terrorism has been tackled by many different authors in many different ways, particularly since the terrorist attacks on the United States in September, 2001. All sorts of policy and approaches to resource allocation and other ways to counter terrorism have been considered. However, most of the approaches that come from Western authors incorporate Western paradigms into their understanding of the problem of terrorism. Authors blindly assume that terrorism can be linked to poverty or other economic factors, or that a purely defensive approach is the best way to counter terrorism.

Rational behavior, such as is assumed within Western confrontations, is expected from Islamist extremists, and when their cultural paradigms allow them to operate outside of rational expectations, many of the Western models of their behavior are made useless.

Because there is a fundamental divide between the cultures of the West and of Islam, the problem of terrorism must be approached in a different manner.

To understand terrorism in a way that will allow people in the West to make appropriate models of the factors involved will require an understanding of the psychological and cultural underpinnings of extremist Islamic ideology. Unlike a state that fights a war in a pragmatic, policy-oriented manner, terrorists do not fight or recruit as Westerners might expect. It would do well to think of terrorism, or rather the ideology behind it, as an epidemic of ideas.

Epidemics have been studied extensively by scientists; a whole field of epidemiology has grown around this study. Mathematical epidemiology uses mathematics to model the spread of epidemics, so that they can be understood and countered more effectively. If the ideology of terrorism can be understood as an epidemic of ideas, then modeling it as an epidemic and studying eradication strategies in light of that epidemic would be useful.

Chapter 3: Mathematical Models of Infectious Diseases

3.1 The SIR Model: An Overview

Epidemiologists have over time developed an entire branch of epidemiology called mathematical epidemiology, which concentrates on modeling the spread of infectious disease mathematically. Different tools exist, tailored to the varying factors involved that the epidemiologists seek to track. However, a basic model that is relevant to our exploration of terrorism as a social epidemic is the classic SIR model.

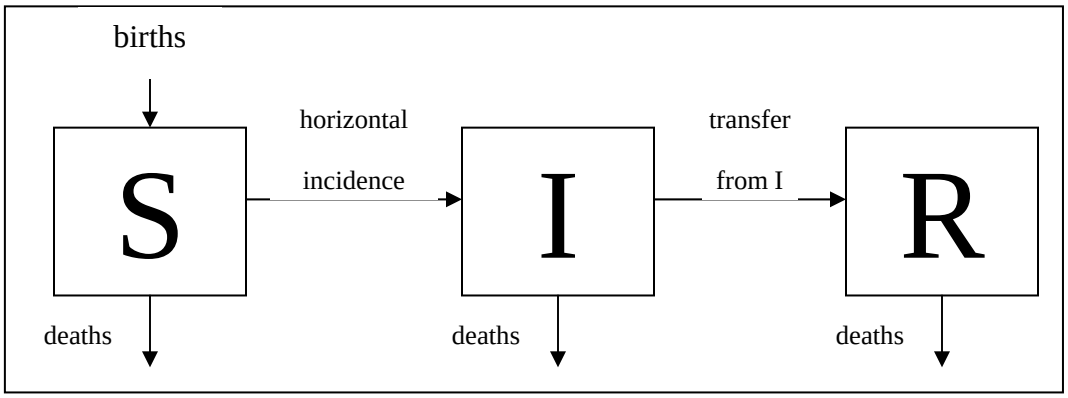


Figure 2: SIR model flow diagram

A SIR model takes the population that interests the epidemiologist and partitions it into several categories, denoted by the letters S, I, and R which, respectively, mean susceptible, infectious, and recovered. The categories are assumed to be mutually exclusive and, in the classic model, transitions only occur from S to I and I to R. This means that R, the recovered category, is an absorbing state and that eventually an epidemic will converge to this state, in a model where there are no birth and death rates

included (the SIR epidemic model, Figure 4). When birth and death rates are accounted for, there will be an equilibrium distribution of susceptible, infected and recovered populations (in the SIR endemic model, Figure 10).

Similar partition-based models include MSEIR, SEIR, SIRS, SEIRS, and MSEIRS models. (Hethcote, 2000: 601) The extra categories included here are M, infants with passive immunity that transfers from their mother, who will transition into the susceptible category, and E, the class of individuals who have been exposed to the infection and are in a latent period before becoming infectious. SIRS, MSEIRS, and SEIRS models (any models with a concluding S) include a transition back into susceptibility for recovered individuals, with a corresponding rate of leaving recovery into susceptibility.

For the sake of simplicity, our initial model of terrorism as an epidemic will be a simple SIR model. An argument could be made for inclusion of the E (exposed) category, a latent period where a person exposed to the "infection" of the terrorist mindset is not yet likely to convert others to that mindset. It would be difficult to retrieve meaningful numbers to approximate how long that latent period lasts, and it is unlikely that surveys exist with that sort of data. Because of these considerations, we will begin our exploration of terrorism as an epidemic by modeling it with a simple SIR model.

Within these models, the common assumption to make is that the time of transfer from one compartment and into another is exponentially distributed. This assumption exploits the memoryless property of exponential distributions. In a standard differential equations model, the rates of transfer will be designated by terms such as δ, ε, and γ and

multiplied by the populations within their corresponding compartments: δM εE and γI. The number of people in the population who are still in the latent period *t* time units after entering it is a function of the rate of transfer, εE.

$$P\{\text{time in E} > t\} = e^{-\varepsilon t}$$

Because the expectation of an exponentially distributed random variable is the reciprocal of its rate, we have the following correspondences between the rates and the expected time within each of these three categories.

Table 1: Expected times within categories M, E, and I

$\frac{1}{\partial}$	Period of passive immunity (time within M category)
$\frac{1}{\varepsilon}$	Mean latent period (time within E category)
$\frac{1}{\gamma}$	Mean infectious period (time within I category)

While movement between these categories is relatively simple to track, based on observations and statistics about the infection in question, when it comes to modeling the transfer into the infected category, more is needed to model the process mathematically. With that in mind, mathematical epidemiologists developed the threshold numbers for infections, and use those in a system of differential equations to complete the model.

3.2 Threshold Numbers: R_0, σ, and R

Within an epidemic or endemic SIR (MSEIR, or SEIR) model, R_0, the basic reproduction number, is the first threshold number to consider. R_0 is the average number of secondary infections that result when a single infected person is introduced into a fully susceptible population. R_0 is only defined at the beginning of an epidemic, but it relates to another significant number for SIR models, the contact number, σ.

The contact number for a SIR model is defined to be the average number of adequate contacts that a typical infected individual would have during their entire time in the infectious category. An adequate contact is one of significant duration to produce a secondary infection when an infectious individual is exposed to a susceptible individual. Thus, at time $t = 0$, $R_0 = \sigma$. Thereafter, σ is used in the mathematical characterization of the epidemic, rather than R_0. This quantity, in turn, leads to the third important number used in mathematical modeling of epidemics, which is R, the replacement number.

The replacement number, R, of a SIR model is defined as the average number of secondary infections that a typical infectious individual produces during the entire period of infectiousness. Again, at time $t = 0$, $R = R_0 = \sigma$.

Each of these threshold numbers behaves differently over time. After the time of invasion ($t = 0$), R_0 ceases to be defined. For most models, σ remains constant throughout the time of the infection. The exception to this rule is in models where after the infection has been introduced, new classes of infections arise with reduced infectiousness. In this case, σ would become smaller as the epidemic continued over time. However, this type

of model is used rarely; typically σ is treated as a constant. *R*, however, will always decrease over time within a SIR model. As the susceptible population decreases, the number of infections that an infectious individual can produce will continue to decrease proportionately.

Thus, at $t = 0$, $R_0 = \sigma = R$. Over time, though, R_0 becomes undefined, and σ remains constant while *R* decreases, because after the time of invasion, only a fraction of the people that any infected person has adequate contact with will be susceptible to infection. We can combine these results into the following formula, true for all SIR (MSEIR, or SEIR) models. (Hethcote, 2000: 601-604)

$$R_0 \geq \sigma \geq R$$

3.3 The Epidemic SIR Model

Given the compartments and parameters that we have discussed above, the table below summarizes the important notation that characterizes a SIR model. Note that the fractional representations of the population should add up to 1. Thus s + i + r = 1, a fact that the differential equations that set up the model draw upon. Note that this is the epidemic model, one developed to track an infection over a short time span.

Table 2: SIR Model Notation

S	Number of individuals within the S (susceptible) compartment
I	Number of individuals within the I (infectious) compartment
R	Number of individuals within the R (recovered) compartment
N	Number in total population (S + I + R = N)
s	Fraction of population that is susceptible (s = S/N)
i	Fraction of population that is infectious (i = I/N)
r	Fraction of population that is recovered (r = R/N)
R_0	Basic reproduction number (or rate)
σ	Contact number
R	Replacement number
t	Time, an independent variable
β	Average number of adequate contacts of an individual per time unit
γ	Rate of transfer out of I category

The definitions above lead to several relevant facts about the categories and rates within the model, some of which are detailed below.

$\sigma = \beta\left(\frac{1}{\gamma}\right) = \frac{\beta}{\gamma}$ Contact number is equivalent to contact rate, β, multiplied by mean length of infection, $1/\gamma$.

$s(t) = \frac{S(t)}{N}$ Susceptible fraction of population

$i(t) = \frac{I(t)}{N}$ Infected fraction of population

$\frac{\beta \mathrm{I}}{\mathrm{N}} = \beta \mathrm{i}$	Average number of adequate contacts with infectious individuals for one susceptible individual per time unit
$\left(\frac{\beta \mathrm{I}}{\mathrm{N}}\right)\mathrm{S} = \beta \mathrm{Nis}$	Average number of new cases per time unit, based on S = Ns being the total number of susceptible individuals within the population

The initial value problem for this model is set up with the differential equations that follow:

$$\frac{d\mathrm{S}}{dt} = \frac{-\beta \mathrm{IS}}{\mathrm{N}} \qquad \mathrm{S}(0) = \mathrm{S}_0 \geq 0$$

$$\frac{d\mathrm{I}}{dt} = \frac{\beta \mathrm{IS}}{\mathrm{N}} - \gamma \mathrm{I} \quad \mathrm{I}(0) = \mathrm{I}_0 \geq 0$$

$$\frac{d\mathrm{R}}{dt} = \gamma \mathrm{I} \qquad R(0) = R_0 \geq 0$$

This model, assuming a short time span, includes no births and deaths. When one divides the equations above by the total population (N, a constant in the epidemic model), the following equations result.

$$\frac{d\mathrm{s}}{dt} = -\beta \mathrm{is} \qquad \mathrm{s}(0) = \mathrm{s}_0 \geq 0$$

$$\frac{d\mathrm{i}}{dt} = \beta \mathrm{is} - \gamma \mathrm{i} \qquad \mathrm{i}(0) = \mathrm{i}_0 \geq 0$$

$$\frac{d\mathrm{r}}{dt} = \gamma \mathrm{i} \qquad \mathrm{r}(0) = \mathrm{r}_0 \geq 0$$

Note that $\mathrm{r}(t) = 1 - \mathrm{s}(t) - \mathrm{i}(t)$. Thus we have that the solution curves to these equations fall within the triangular region T, known as the phase plane for the epidemic model, where

$$\mathrm{T} = \{(\mathrm{s},\mathrm{i}) : \mathrm{s} \geq 0, \mathrm{i} \geq 0, \mathrm{s} + \mathrm{i} \leq 1\}.$$

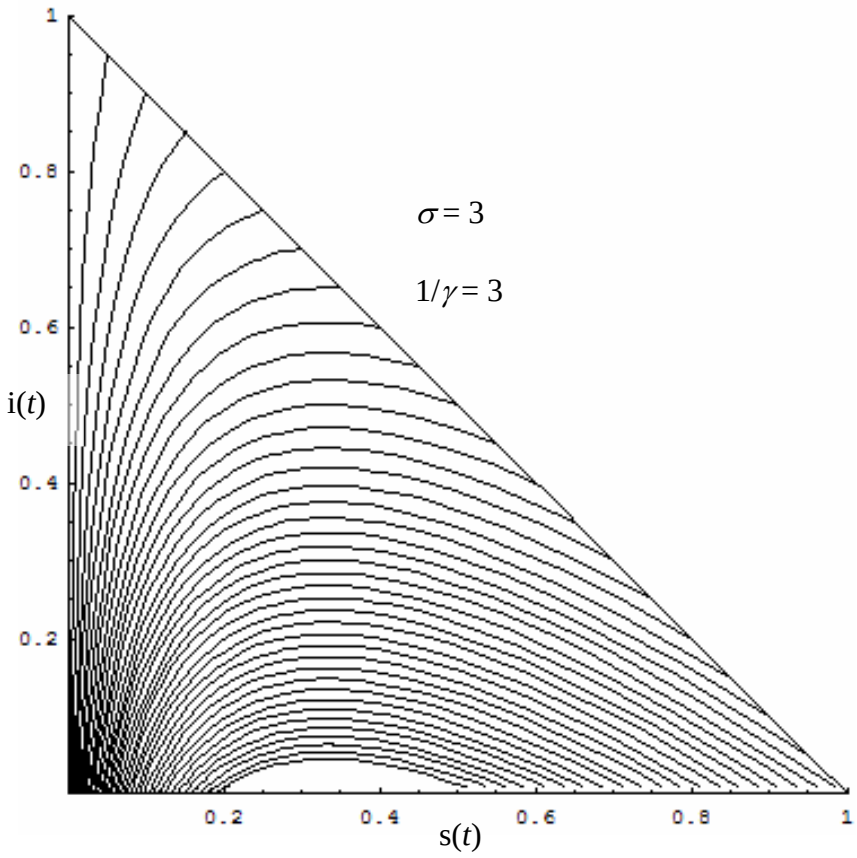


Figure 3: Phase plane portrait of epidemic SIR model

Note that when $\sigma = 3$ and $1/\gamma = 3$ (days), the paths over time curve from the right side of T to the left, eventually dying out as i(t) = 0.

The same functions can be seen in the graph below, where s(t) and i(t) are shown against time. The tendency of a typical epidemic is for i(t) to peak and then die down, as the susceptible fraction reaches a steady-state number.

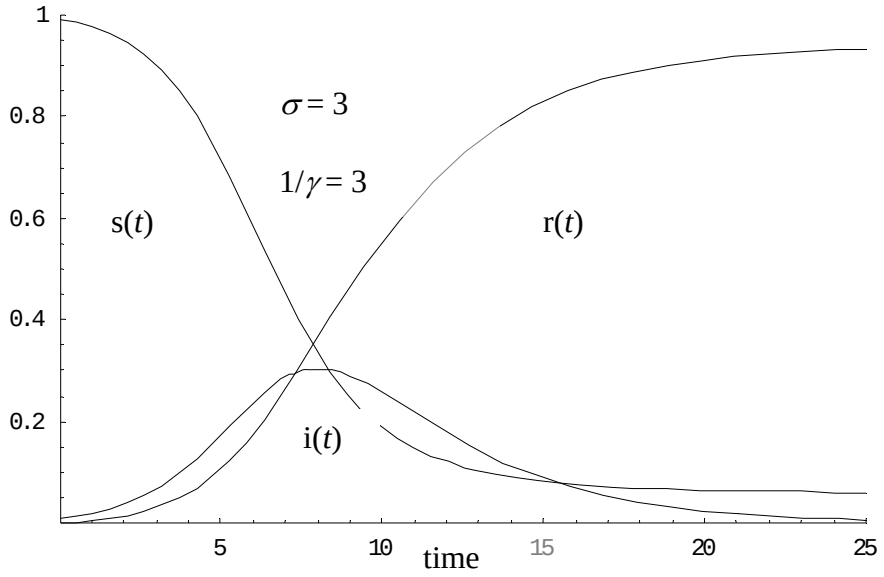


Figure 4: Susceptible and infected population proportions over time

When σ increases, the peak proportion of the population that is infectious increases dramatically. This peak of infected population happens in a shorter period of time, but the duration of the epidemic becomes correspondingly shorter. Figure 5 and Figure 6 illustrate this graphically.

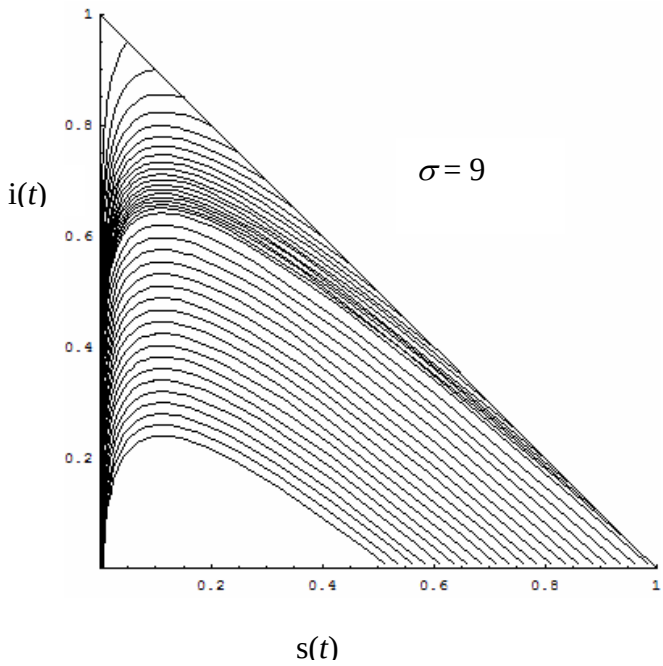


Figure 5: Higher contact number SIR model phase plane

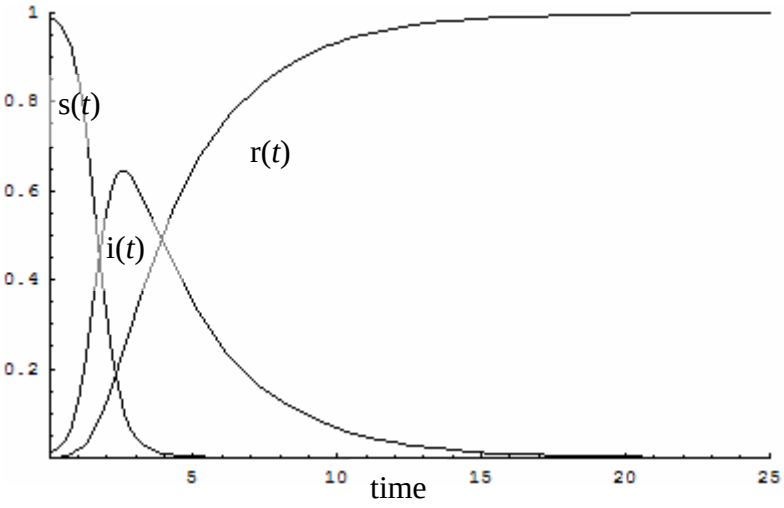


Figure 6: Higher contact number SIR model over time

Conversely, when σ decreases, the peak infectious proportion of the population becomes much smaller. Because the proportion of infectious individuals is not large

enough at any point to expose the entire susceptible population to infection, the infection tapers out before the susceptible population is reduced to proportions near zero. When σ is small enough, an epidemic's end state will result in only part of the population having been exposed to the infection. Figure 7 and Figure 8 graphically illustrate the impact of a low contact number for an epidemic.

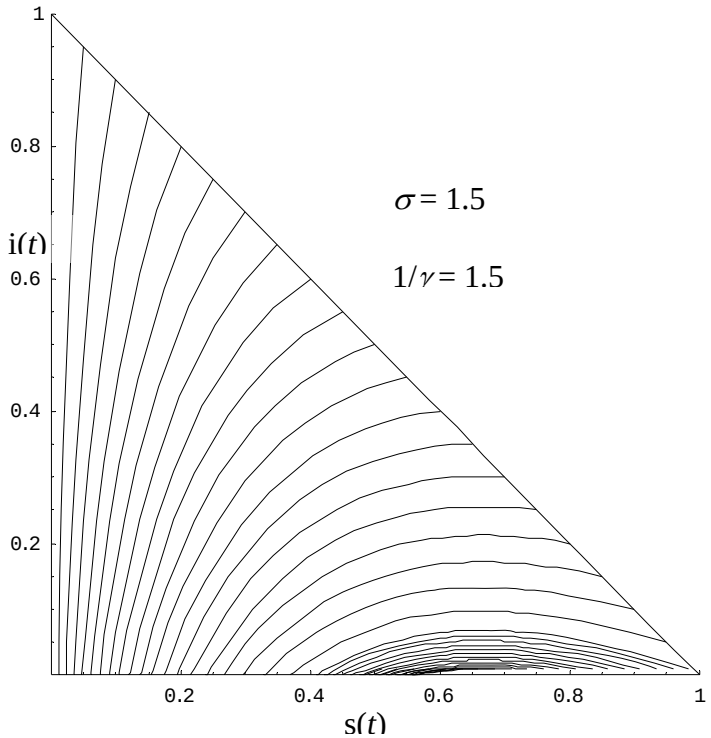


Figure 7: Low contact number SIR model phase plane

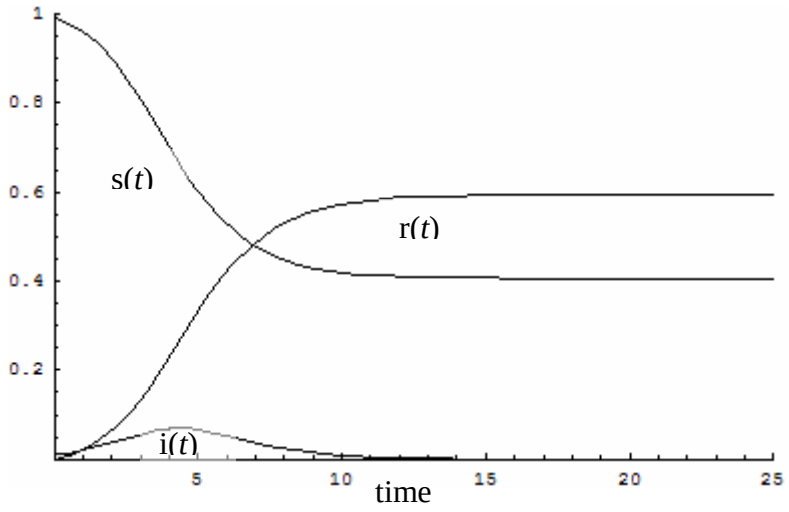


Figure 8: Low contact number SIR model over time

The solution paths within T, characterized by the following equation

$$i(t) + s(t) - \frac{\ln(s(t))}{\sigma} = i_0 + s_0 - \frac{\ln(s_0)}{\sigma}$$

are found from the quotient differential equation below.

$$\frac{di}{ds} = -1 + \frac{1}{\sigma s}$$

Equilibrium points (the end state) can be found along the s axis in the phase plane portrait. These are neutrally unstable for $s > 1/\sigma$ and neutrally stable for $s < 1/\sigma$. (Hethcote, 2000: 607)

3.4 The Endemic SIR Model

An endemic model accounts for births and deaths within the population and thus tracks an infection over a potentially longer time span than the epidemic model. If nothing else, after an entire recovered generation dies out, the new generation will become fully susceptible again, leaving the population open to another epidemic. The behavior of endemic infections is actually more complex than this, however.

The model for the endemic SIR model is very much the same as for the epidemic model, except that it incorporates a birth and death rate. The birth rate, μN, balances the death rate, also μN. N = S + I + R, which implies that μN = μ(S + I + R) = μS + μI + μR. Because of this, μS, μI, and μR are the death rates within susceptible, infectious, and recovered categories, respectively, in the SIR endemic model. This assumption ensures well-behaved movement within the model but is somewhat optimistic; in our terrorism example it is quite likely that the population is growing exponentially. The mean lifetime for a given individual is $1/\mu$, which can be found easily from demographic reports about regions of interest.

The initial value problem is set up as follows:

$$\frac{dS}{dt} = \mu N - \mu S - \frac{\beta IS}{N} \qquad S(0) = S_0 \geq 0$$

$$\frac{dI}{dt} = \frac{\beta IS}{N} - (\gamma + \mu)I \qquad I(0) = I_0 \geq 0$$

$$\frac{dR}{dt} = \gamma I - \mu R \qquad R(0) = R_0 \geq 0$$

Because the proportions of the population are assumed to remain constant whether the population grows or shrinks, we divide by N to produce the following equations.

$$\frac{ds}{dt} = \mu - \mu s - \beta is \qquad s(0) = s_0 \geq 0$$

$$\frac{di}{dt} = \beta is - (\gamma + \mu) i \qquad i(0) = i_0 \geq 0$$

$$\frac{dr}{dt} = \gamma i - \mu r \qquad r(0) = r_0 \geq 0$$

The threshold numbers are slightly different for this model as well:

$R_0 = \sigma = \frac{\beta}{(\gamma + \mu)}$ where $\frac{1}{(\gamma + \mu)}$ is the average death-adjusted infectious period

Behavior within an endemic model tends to alternate between rapid epidemic outbreaks and slow regeneration of the susceptible class. As time continues, an endemic equilibrium will eventually be reached, characterized by the equations below.

$$s_e = \frac{1}{\sigma} \qquad i_e = \frac{\mu(\sigma - 1)}{\beta}$$

Figure 9 below shows the characteristic path of a solution within the phase plane, spiraling toward an endemic equilibrium. In this case, the average lifespan is 60 days, in order to show clearly the behavior of the solution paths.

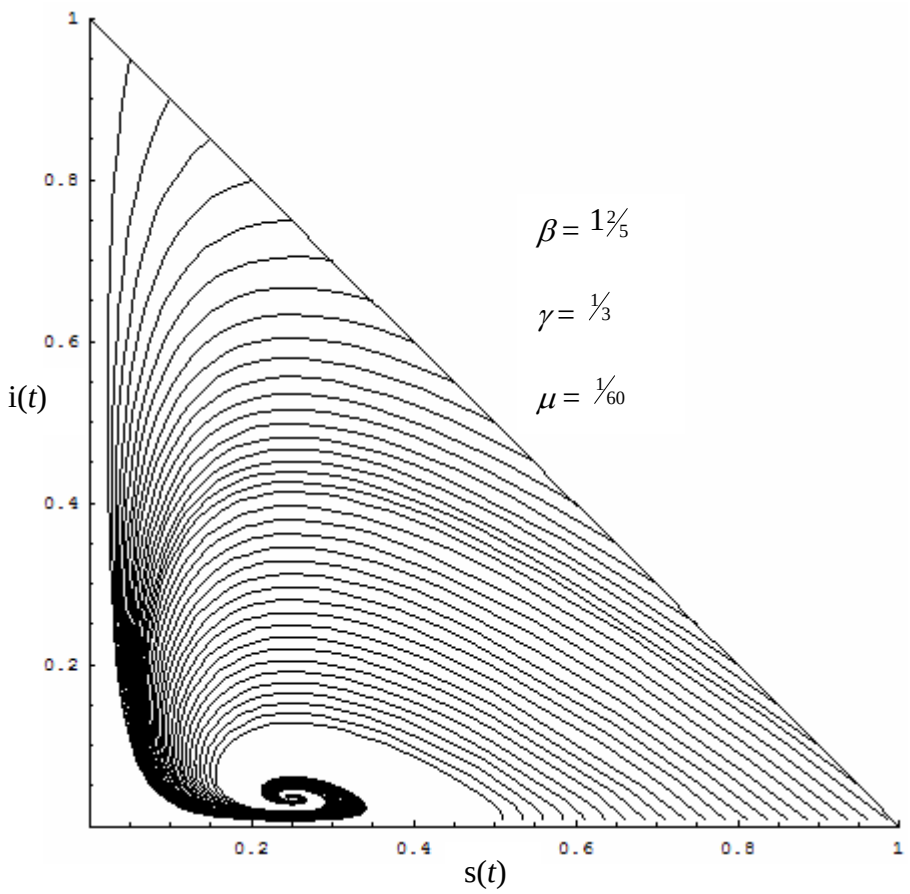


Figure 9: Endemic SIR model phase plane

In Figure 10, below, the damped oscillation in the infectious population becomes clearly visible. After the first peak of epidemic outbreak, the susceptible population gradually grows, due to birth rates in the susceptible category. When the susceptible proportion of the population reaches a high enough point, another epidemic peak occurs, but this time the peak looks characteristic of an epidemic with a smaller contact rate. Because the secondary epidemics when the susceptible proportion of the population is reduced, this effectively reduces the rate at which any infectious person is making

adequate contact with susceptible people—because he or she will also be encountering recovered (and thus immune) individuals.

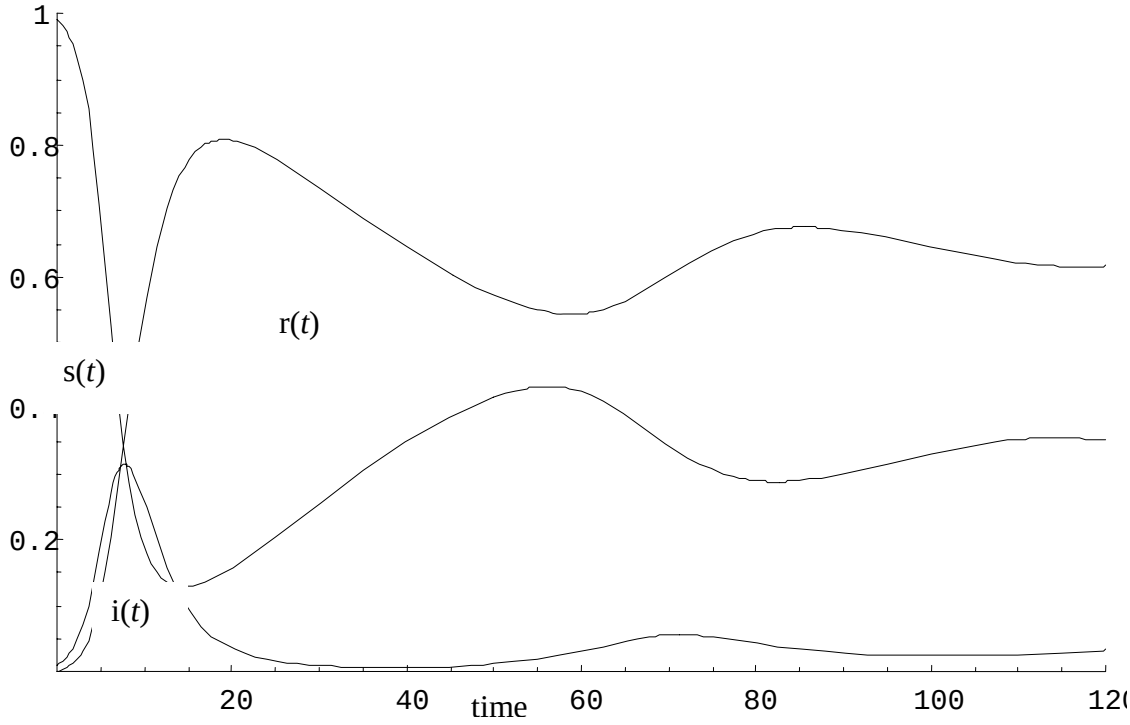


Figure 10: Endemic model over time

A more typical endemic model would characteristically show the infectious population becoming negligible over time, as a result of the reduction of the birth (and death) rate, μ. When an infection occurs over a period of days (or weeks), and expected lifespan ($1/\mu$) is measured in years, the results are illustrated in Figure 11, below.

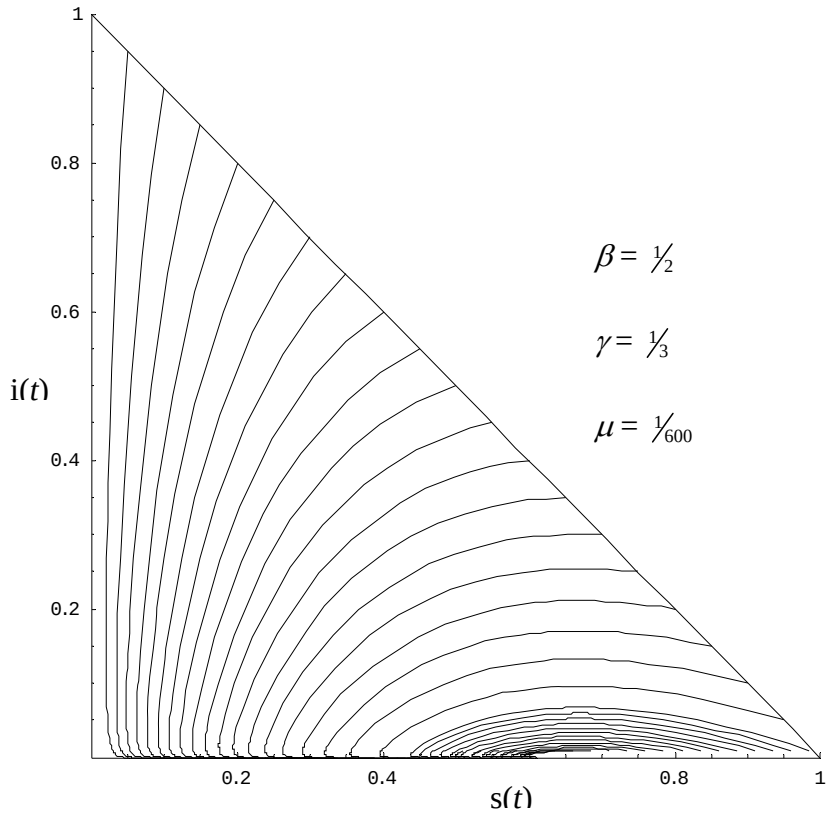


Figure 11: Typical paths for an endemic phase plane

3.5 Adding age groups to the SIR model

Because epidemics often affect different age groups differently, epidemiological models often incorporate partitions between different age groups, in order to better show the rates at which the disease affects these age groups. With a standard infection, it is likely that an older individual will have more resistance to the disease, and contact rates can vary between different age groups. Just as the endemic model incorporates another

level of detail to the model, SIR models that incorporate age groups add another layer of detail to the picture of the infection in question.

Age is treated as an independent variable, just as time is, and can be handled as either a continuous function or stepwise as a series of age groups. We will choose the age group approach as it is easier to collect and report statistical data about age groups than to create continuous functions that express birth, death, and recovery rates in terms of age. Age group models typically involve an assumption of exponential population growth (Hethcote, 2000: 619-620), modeled by the following equation, where

$\mathrm{N}(t)$	size of the population at time t
$\mathrm{N}'(t)$	rate of change of the population at time t
b	mean natural birth rate
d	mean natural death rate.

$$\mathrm{N}'(t) = (b - d)\mathrm{N}(t)$$

Letting $q = b - d$, we have that

$$\frac{d\mathrm{N}}{dt} = q\mathrm{N}.$$

Given that our population is growing (or shrinking) exponentially, we assume the distribution of proportions within the populations of n different age groups remains constant. Let P_i denote the proportion of the total population that is in age group i, where $i \in \{1, \ldots, n\}$, and

$$\sum_{i=1}^{n} P_i = 1$$

This leads to the understanding that the total number of individuals within each age group, N_i, where $i \in \{1, \ldots, n\}$, can be found with the following equation. (Hethcote, 2000: 635)

$$N_i(t) = e^{qt} P_i$$

Note that in the SIR model, where $s_i = S_i/N_i$, $i_i = I_i/N_i$, and $r_i = R_i/N_i$, we have the following property.

$$P_i = s_i + i_i + r_i \quad \forall \ i \in \{1, \cdots, n\}$$

Part of the problem of modeling age groups is the underlying assumption that contact rates (which previously we modeled as a constant rate, β, for all individuals within the system) will be different between the differing age groups. Thus we have a new set of rates that we will model as follows.

Table 3: Contact rates in the age category SIR model

β_i	The contact rate between individuals in the same age group
$\beta_i \tilde{\beta}_j$	The contact rate between individuals in separate age groups

The total force of infection for each age group is λ_i, where $i \in \{1, \ldots, n\}$. This is equivalent to the rate of contact across all age groups multiplied by the number of infected in each of those age groups, and is the more complex variant of β that we used in our previous equations. (Hethcote, 2000: 635)

$$\lambda_i = \sum_{j=1}^{n} \beta_i \tilde{\beta}_j \, \mathrm{i}_j, \text{ where } \beta_i \tilde{\beta}_i = \beta_i$$

Note that instead of using the notation $\beta_i \tilde{\beta}_j$, the two age category model developed later will use the notation β_{ij}, because it is simpler to understand. The table below details the meaning of each of these rates.

Table 4: Meanings of β rates

β_{11}	The rate at which infectious individuals in group 1 infect susceptibles in group 1
β_{12}	The rate at which infectious individuals in group 2 infect susceptibles in group 1
β_{21}	The rate at which infectious individuals in group 1 infect susceptibles in group 2
β_{22}	The rate at which infectious individuals in group 2 infect susceptibles in group 2

Another rate that we need to be concerned with in the age category SIR model is the rate at which individuals leave age category i and enter category $i + 1$. This rate is labeled c_i, where $i \in \{1, \ldots, n\}$. This rate must be distinguished from the death rate, d_i, at which people in category i are dying when they are within that age group.

The flow diagram of the two-category model is detailed in Figure 12 below. This illustrates all the important states that any particular individual can exist within and the flow rates between these states, where the time spent in every state is assumed to be exponentially distributed.

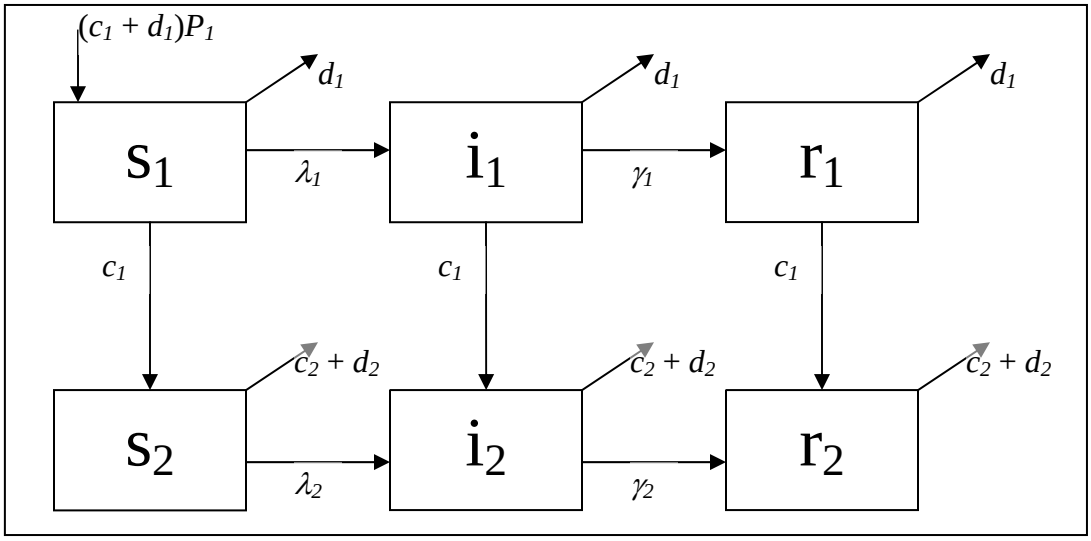


Figure 12: Age category model flow diagram

With these equations and the terms that we have previously defined, we are ready to set up the differential equations that define our age group model. This model is characterized by a set of 3*n* equations, with *n* being the number of age groups within the model. Note that the

$$\frac{d\mathrm{s}_1}{dt} = (c_1 + d_1 + q)P_1 - [\lambda_1 + c_1 + d_1 + q]\mathrm{s}_1$$

$$\frac{d\mathrm{i}_1}{dt} = \lambda_1 \mathrm{s}_1 - [\gamma_1 + c_1 + d_1 + q]\mathrm{i}_1$$

$$\frac{d\mathrm{r}_1}{dt} = \gamma_1 \mathrm{i}_1 - [c_1 + d_1 + q]\mathrm{r}_1$$

$$\frac{d\mathrm{s}_i}{dt} = c_{i-1}\mathrm{s}_{i-1} - [\lambda_i + c_i + d_i + q]\mathrm{s}_i \qquad i \geq 2$$

$$\frac{d\mathrm{i}_i}{dt} = c_{i-1}\mathrm{i}_{i-1} + \lambda_i \mathrm{s}_i - [\gamma_i + c_i + d_i + q]\mathrm{i}_i \qquad i \geq 2$$

$$\frac{d\mathrm{r}_i}{dt} = c_{i-1}\mathrm{r}_{i-1} + \gamma_i \mathrm{i}_i - [c_i + d_i + q]\mathrm{r}_i \qquad i \geq 2$$

This definition of equations can be simplified, in the two-category case, to the equations below. For the sake of example, we take the case where population growth rate is zero. That is, there is a static population size and the rate at which people enter the system is the same as the rate at which they leave the system.

$$\frac{d\mathrm{s}_1}{dt} = (c_1 + d_1)P_1 - [\lambda_1 + c_1 + d_1]\mathrm{s}_1$$

$$\frac{d\mathrm{i}_1}{dt} = \lambda_1 \mathrm{s}_1 - [\gamma_1 + c_1 + d_1]\mathrm{i}_1$$

$$\frac{d\mathrm{r}_1}{dt} = \gamma_1 \mathrm{i}_1 - [c_1 + d_1]\mathrm{r}_1$$

$$\frac{d\mathrm{s}_2}{dt} = c_1 \mathrm{s}_1 - [\lambda_2 + c_2 + d_2]\mathrm{s}_2$$

$$\frac{d\mathrm{i}_2}{dt} = c_1 \mathrm{i}_1 + \lambda_2 \mathrm{s}_2 - [\gamma_2 + c_2 + d_2]\mathrm{i}_2$$

$$\frac{d\mathrm{r}_2}{dt} = c_1 \mathrm{r}_1 + \gamma_2 \mathrm{i}_2 - [c_2 + d_2]\mathrm{r}_2$$

Demographic data is available for the proportions of the population within age groups, P_i, and the death rates within each age category, d_i. The rate at which people leave each category and enter the next, c_i, can be calculated by the length of time that they spend within each age group. The final age group's exit rate, c_n, or in this case c_2, has to be calculated so that the rate at which people leave the final age group is equal to

the rate at which they enter it. Note that, in the final category, c_n just means the rate at which individuals exit the system. For the two-category model, this means

$$c_1 P_1 = (c_2 + d_2) P_2$$

Note that our initial values, shown in Table 5 below, include a larger category P_1 than P_2. For the example problem, we assume that age group 1 dies at a slower rate than age group 2, but it sustains the infection for a longer time than the second age group. Additionally, while each age category is infected by members within itself at the same rate, members of category 2 will have a greater likelihood of infecting those of category 1 than vice-versa.

Table 5: Two age group SIR model initial values

$P_1 = 0.6$	$P_2 = 0.4$
$\lambda_1 = \beta_{11} \mathrm{i}_1 + \beta_{12} \mathrm{i}_2$	$\lambda_2 = \beta_{21} \mathrm{i}_1 + \beta_{22} \mathrm{i}_2$
$\beta_{11} = 0.75$	$\beta_{12} = 1.0$
$\beta_{21} = 0.25$	$\beta_{22} = 0.75$
$\gamma_1 = \frac{1}{3}$	$\gamma_2 = \frac{1}{2}$
$d_1 = \frac{1}{200}$	$d_2 = \frac{1}{100}$
$c_1 = \frac{1}{20}$	$c_2 = \frac{c_1 P_1}{P_2} - d_2 = \frac{13}{200}$

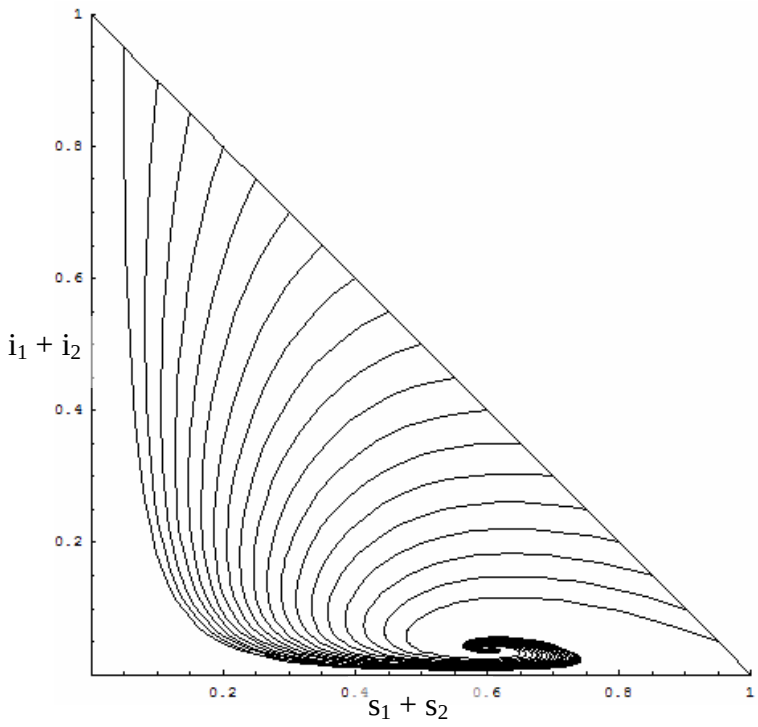


Figure 13: Two age group SIR model phase plane

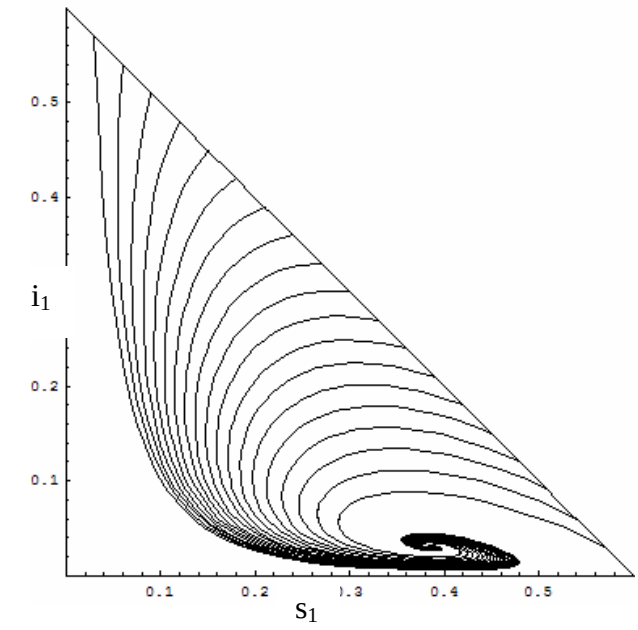


Figure 14: Two age group SIR model phase plane for category 1 only

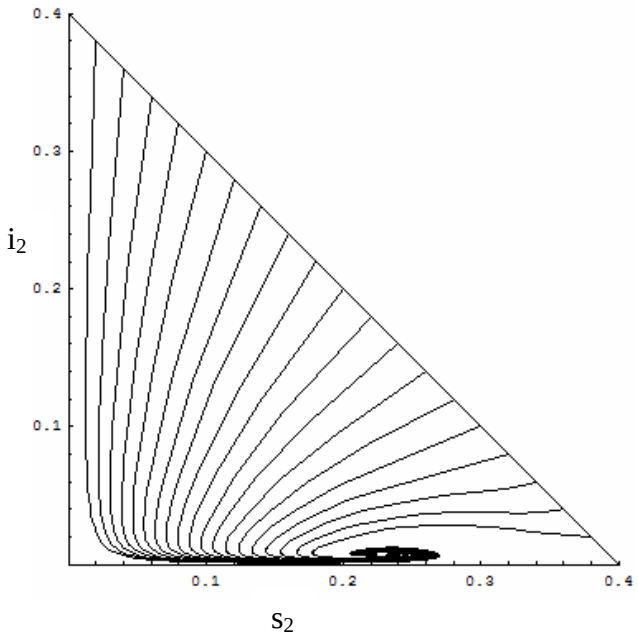


Figure 15: Two age group SIR model phase plane for category 2 only

One can clearly see in the age group phase planes that the initial values that we have set have changed the outcome for the different age categories. In particular, the longer time of infection for the first age bracket makes the curves in the phase plane have a much more spiral nature than they do in the second age bracket. Additionally, one can see that the systemic equilibrium for the different age categories is going to be different for each. In this model, the first age group converges to an endemic equilibrium with a higher proportion of infectious individuals than does the second age group, because of the longer duration of the infection and the inflow of susceptible individuals.

The next set of valuable information about the age group model is what the different populations are doing over time. With this in mind, we graph the value of the populations over time, assuming a negligible number of infected population (0.05 of the total population), and the results follow in Figure 16, Figure 17, and Figure 18.

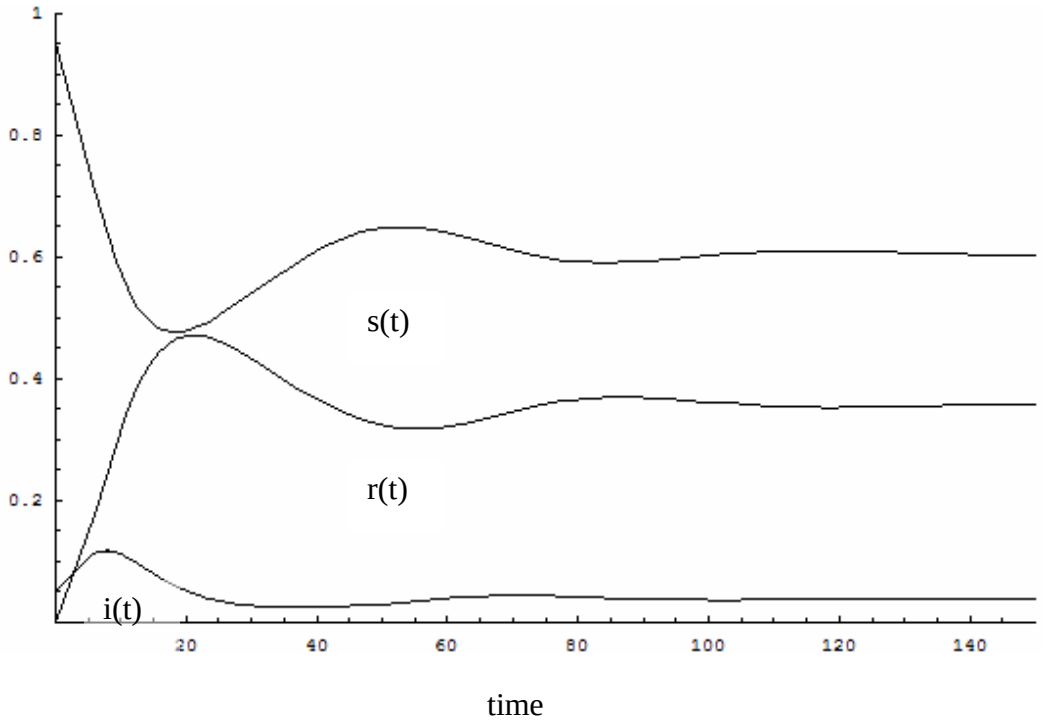


Figure 16: Susceptible, infectious, and recovered fractions of age group model

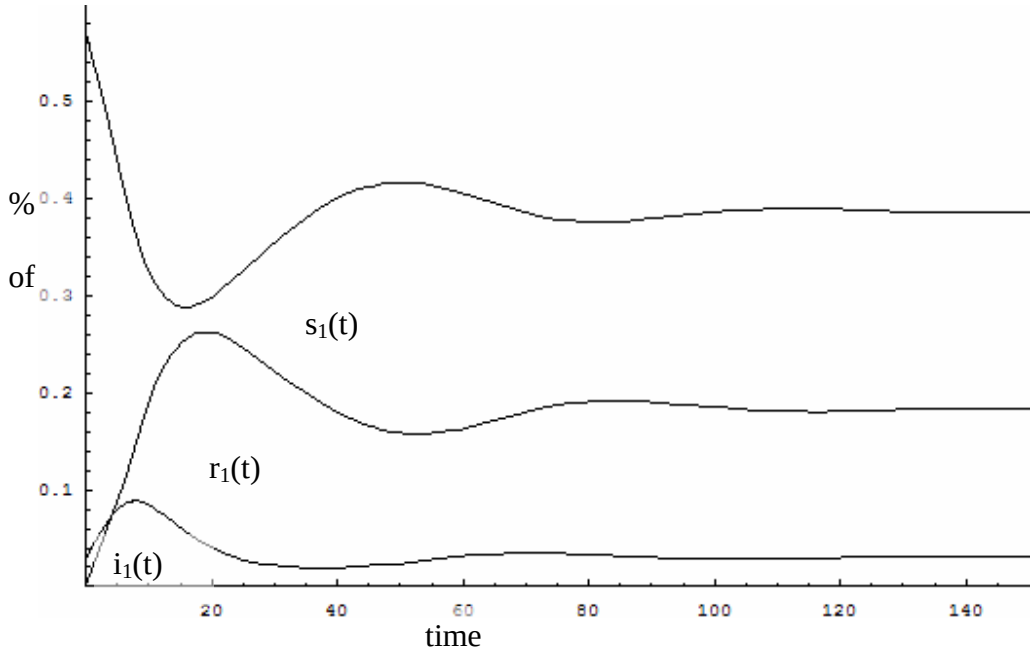


Figure 17: Susceptible, infectious, and recovered fractions of category 1

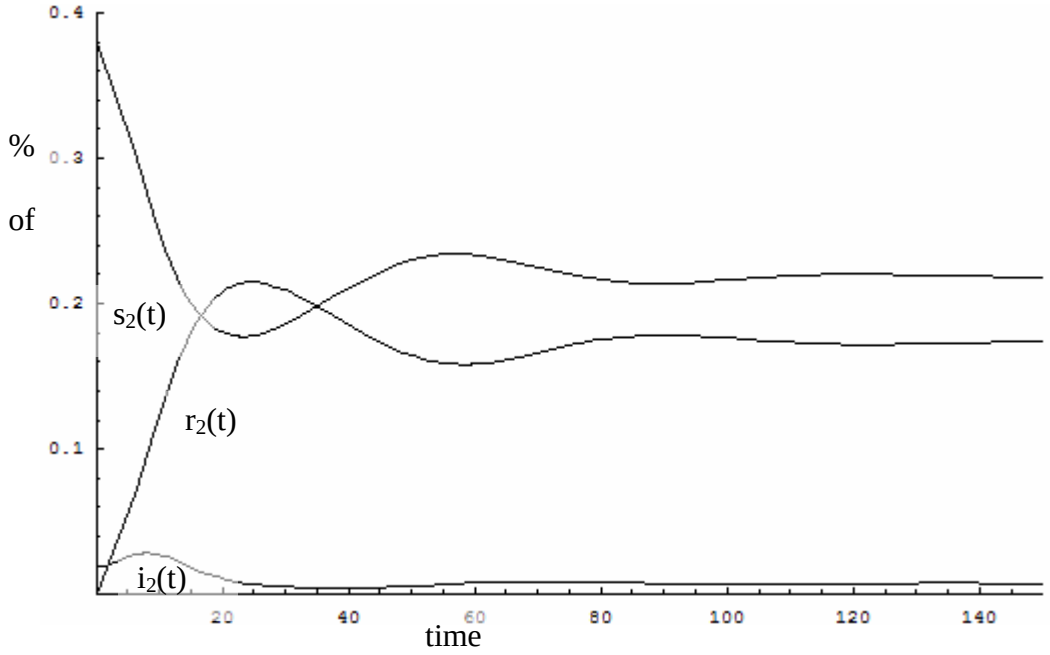


Figure 18: Susceptible, infectious, and recovered fractions of category 2

One can see that the shapes of the curves associated with each part of the population are different. In this case, the overall epidemic occurs with more force in the younger part of the population, which consequently results in an endemic equilibrium with a larger steady-state number (relative to the total population of the younger age category) for infected and susceptible groups, and a smaller equilibrium state for the recovered portion of the younger population. However, since one can assume that a younger person will eventually transition into the older category, this makes sense; the younger age bracket is steadily losing people from its later categories to the older age bracket. Since the older category includes the absorbing state, r_2, it has a much larger proportion (relative to the population) of recovered individuals than does the first category.

Both epidemic and endemic models serve a purpose, and have potential application to the problem of terrorism spreading as a social epidemic. It is a matter of which parts of the problem that one chooses to focus on that determines which model one would use. An epidemic model looks at a short-term problem and focuses on what the initial outbreak of an infection is going to look like, while an endemic model tends to be of more use to determine what the eventual steady-state distribution of the infection will become. An endemic model assumes a longer time horizon than an epidemic model does.

Additionally, the age category model has potential use to model the spread of the "infection" of the terrorist mindset. This would allow partitioning of the entire population of a given region into more and less susceptible groups, and might give a more detailed picture of the dynamics involved in the spread of terrorist ideology. However, this model

also requires more initial settings for rates, and while many of them come directly from known demographic data, others need to be estimated.

3.6 Applying the partition principle to varying levels of influence

Using similar partitioning methodology to that we used to develop the age category model, we develop a model based on categories of influence. This type of model will look similar to the age category model, but will incorporate the understanding that the proportion of highly influential individuals will be very much less than that of normal people in the population. However, these influential individuals can be expected to have much greater infectiousness, which will change the force of infection values. This incorporates the notion that some individuals will have a much greater tendency of carrying the infection to others, the idea that *The Tipping Point* emphasizes in its discussion of the people factors involved in epidemics of ideas. (Gladwell, 2000)

Partitioning can also be used to model the way an epidemic spreads through multiple populations, such as in different regions or cities, because in this way the differing rates of interaction can be illustrated. (Zaric and Brandeau, 2002) Such partitions can become very complicated, especially when rates of contact between populations are factored in, because the flow is not simply out of one population and into another, but in and out of both. SIR models that incorporate partitioning thus have the potential to display complex interactions of factors that influence the spread and duration of epidemics.

3.7 One step further: the effects of immunization on the model

Though SIR models are based on the assumption that the only way out of the susceptible category is into the infected category, it makes sense to take the model one step farther. In this immunization SIR model, we theorize that individuals within the recovered category are capable of having a positive influence on susceptible individuals. This means that, if exposed for a sufficient length of time to a recovered person, a susceptible individual could be effectively "immunized" against the contagion in question. Immunization, in this sense, means that the disease never infected the individual. While this assumption would not make sense within a typical biological epidemic model, it can be incorporated into a model of an epidemic of ideas. In this case, one can expect the recovered category to be composed of people who have a positive (or neutral) influence on susceptible people. The main item of importance is that the influence of the recovered category will enable those who are susceptible to infection to bypass the infected state. In essence, their influence is an inoculation against the infection.

This model will look very much like the model partitioned by influence, but will incorporate the effects of normal and highly influential people in both infectious and immunizing ways. Figure 19, below, is the flow diagram of this model. Category 1 stands for normal individuals and category 2 for highly influential individuals.

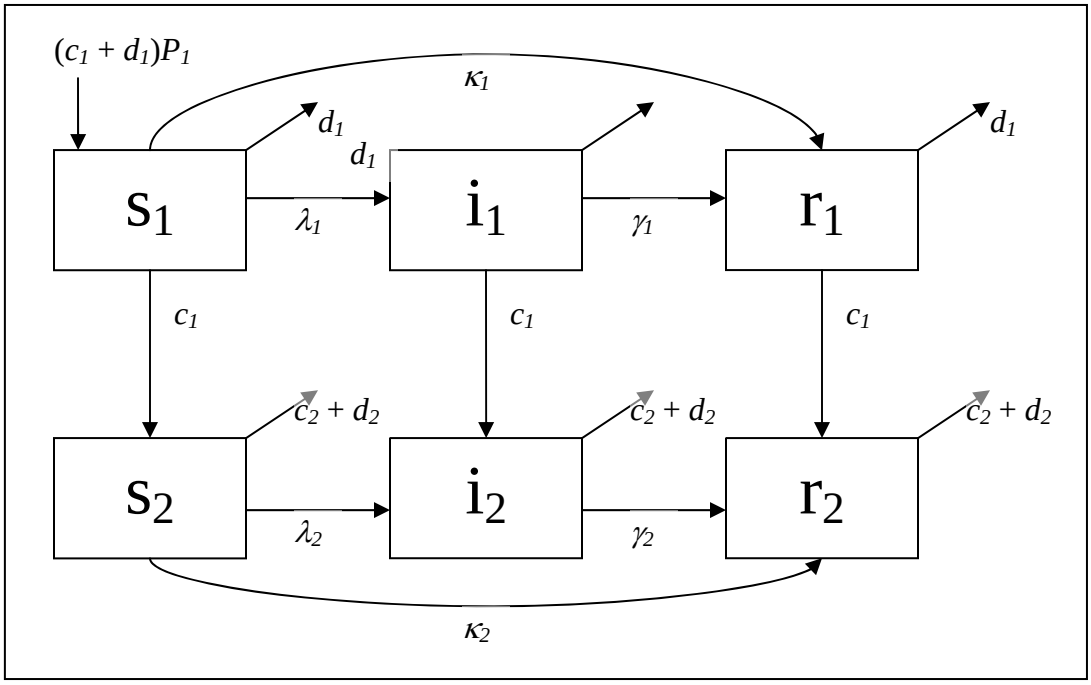


Figure 19: Partition model flow diagram with immunization effects

While all our rates from before remain what they were before, there is an additional factor of the κ rates, which will be the forces of immunization for each of the susceptible groups. These can be defined as following:

$$\kappa_1 = (\alpha_{11}\, r_1 + \alpha_{12}\, r_2)\, s_1 \qquad \kappa_2 = (\alpha_{21}\, r_1 + \alpha_{22}\, r_2)\, s_2$$

The α values here measure the rates at which one population of recovered will immunize the susceptible population of the category in question. These α values are analogous to the β values associated with force of infection rates.

Table 6: Meanings of α rates

α_{11}	The rate at which recovered individuals in group 1 immunize group 1 susceptibles
α_{12}	The rate at which recovered individuals in group 2 immunize group 1 susceptibles
α_{21}	The rate at which recovered individuals in group 1 immunize group 2 susceptibles
α_{22}	The rate at which recovered individuals in group 2 immunize group 2 susceptibles

The immunization factor will change the shape of the curves associated with the epidemic, depending on what initial values are chosen. The equations below and the rates in Table 7 set up the influence model with immunization.

$$\frac{d\,\mathrm{s}_1}{dt} = (c_1 + d_1)P_1 - [\lambda_1 + \kappa_1 + c_1 + d_1]\mathrm{s}_1$$

$$\frac{d\,\mathrm{i}_1}{dt} = \lambda_1 \mathrm{s}_1 - [\gamma_1 + c_1 + d_1]\mathrm{i}_1$$

$$\frac{d\,\mathrm{r}_1}{dt} = \kappa_1 \mathrm{s}_1 + \gamma_1 \mathrm{i}_1 - [c_1 + d_1]\mathrm{r}_1$$

$$\frac{d\,\mathrm{s}_2}{dt} = c_1 \mathrm{s}_1 - [\lambda_2 + \kappa_2 + c_2 + d_2]\mathrm{s}_2$$

$$\frac{d\,\mathrm{i}_2}{dt} = c_1 \mathrm{i}_1 + \lambda_2 \mathrm{s}_2 - [\gamma_2 + c_2 + d_2]\mathrm{i}_2$$

$$\frac{d\,\mathrm{r}_2}{dt} = c_1 \mathrm{r}_1 + \kappa_2 \mathrm{s}_2 + \gamma_2 \mathrm{i}_2 - [c_2 + d_2]\mathrm{r}_2$$

Table 7: Influence model with immunization initial values

$P_1 = 0.99$	$P_2 = 0.01$
$\lambda_1 = \beta_{11}\,\mathrm{i}_1 + \beta_{12}\,\mathrm{i}_2$	$\lambda_2 = \beta_{21}\,\mathrm{i}_1 + \beta_{22}\,\mathrm{i}_2$
$\beta_{11} = 0.75$	$\beta_{12} = 100$
$\beta_{21} = 0.2$	$\beta_{22} = 25$
$\kappa_1 = \alpha_{11}\,\mathrm{r}_1 + \alpha_{12}\,\mathrm{r}_2$	$\kappa_2 = \alpha_{21}\,\mathrm{r}_1 + \alpha_{22}\,\mathrm{r}_2$
$\alpha_{11} = 0.5$	$\alpha_{12} = 75$
$\alpha_{21} = 0.1$	$\alpha_{22} = 20$
$d_1 = \frac{1}{100}$	$d_2 = \frac{1}{100}$
$c_1 = \frac{1}{20}$	$c_2 = \frac{c_1 P_1}{P_2} - d_2 = \frac{247}{50}$
$\gamma_1 = \frac{1}{3}$	$\gamma_2 = \frac{1}{2}$

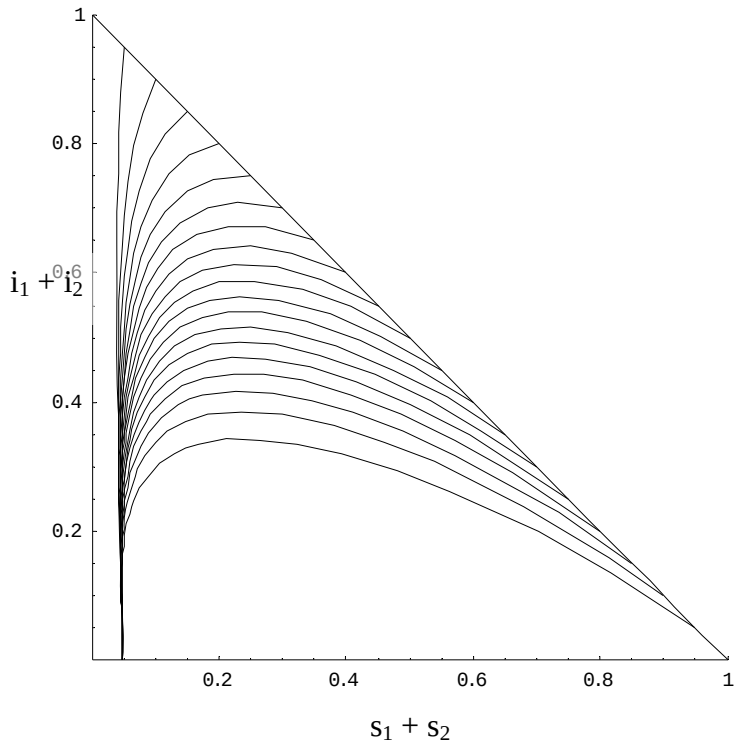


Figure 20: Influence category SIR model with immunization phase plane

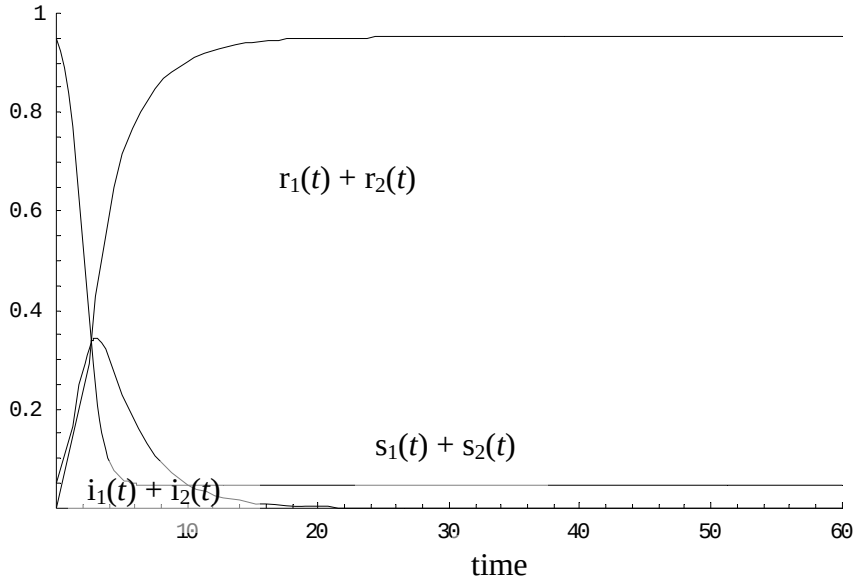


Figure 21: Influence category SIR model with immunization over time

Since normal people, who compose P1, are such a large percentage of the population, the graphs barely change for their statistics considered alone. However, the difference between these graphs and the graphs for influential people is considerably greater, and one can see a great difference between the epidemic at large compared to that within those people of influence. One can see this influence in Figure 22 and Figure 23 below.

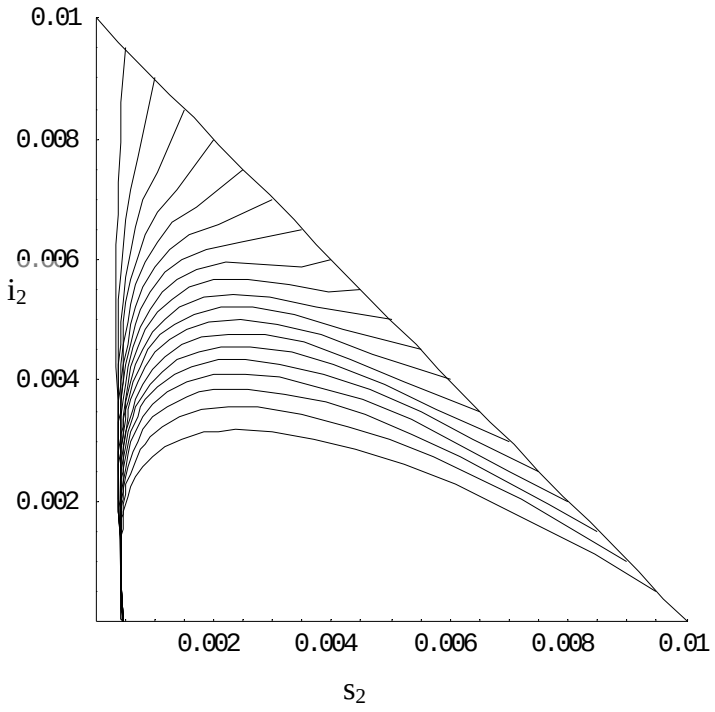


Figure 22: Highly influential individuals phase plane

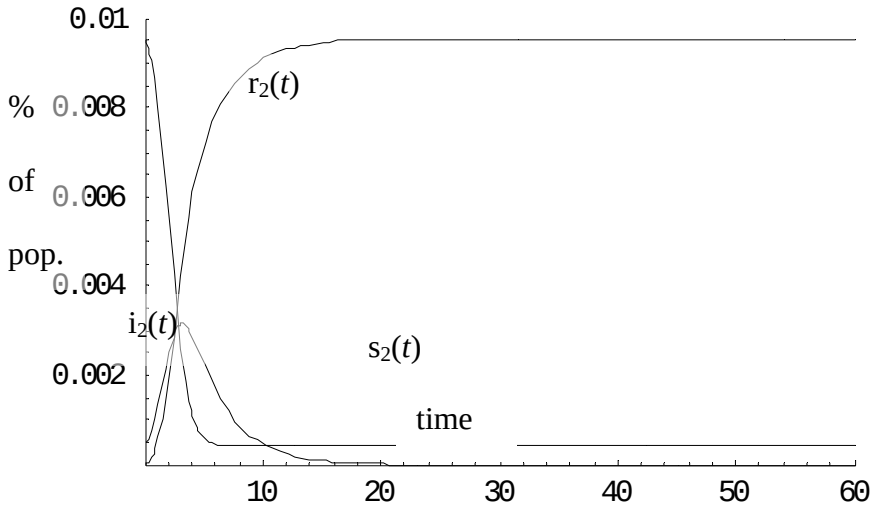


Figure 23: Highly influential individuals over time

Modeling immunization in this way, the epidemic curve scarcely changes in shape. However, instead of settling to an endemic equilibrium point, the curves for s(*t*) and i(*t*) trend toward zero, meaning that r(*t*) trends toward 1. In this sense, the growing influence of the recovered population will eventually obliterate the incidence of the infection. This is not a realistic expectation to make about a biological epidemic, but when one is modeling a social epidemic, it could be close to the mark. When an idea stops having followers, it tends to die out.

For most epidemiological models for biological infections, immunization is modeled by reducing the inflow into the susceptible category and correspondingly increasing the inflow into the recovered category. Trottier and Phillippe (2003) discuss the expected change that inoculation will have on an epidemic, showing that it lengthens the intervals between epidemic outbreaks (initial and following peaks in a model like

Figure 10), and each epidemic outbreak is smaller in scale until they disappear altogether. Modeled into the differential equations for the SIR model, this change is a reduction of the inflow into the susceptible category. The equations that make up an endemic SIR model become the following, where μ is the rate of people entering (and leaving) the system, γ is the rate at which people recover from infection, β is the rate at which people become infected, and *p* is the rate of immunization of the susceptible population.

$$\frac{d\mathrm{s}}{dt} = \mu(1-p) - \mu\mathrm{s} - \beta\mathrm{is} \qquad \mathrm{s}(0) = \mathrm{s}_0 \geq 0$$

$$\frac{d\mathrm{i}}{dt} = \beta\mathrm{is} - (\gamma + \mu)\mathrm{i} \qquad \mathrm{i}(0) = \mathrm{i}_0 \geq 0$$

$$\frac{d\mathrm{r}}{dt} = \mu p + \gamma\mathrm{i} - \mu\mathrm{r} \qquad \mathrm{r}(0) = \mathrm{r}_0 \geq 0$$

The disadvantage of modeling immunization this way is that it does not capture the influence that "recovered" individuals will have on susceptible individuals. Because ideas that are contrary to the epidemic in question are infectious (though perhaps less so than the epidemic), those who are "recovered" from the infection exert their influence on susceptible individuals. In that sense, our proposed model of immunization to a social epidemic makes more sense than the typical biological model.

3.8 Applications of SIR models

Not only is a SIR model descriptive of an epidemic, but it also can be used to solve problems related to the control of such epidemics. Zaric and Brandeau (2002) work

through a resource allocation problem based on a multiple population model. In their case, expected years of life for HIV/AIDS infected individuals are maximized, based on the assumption of a group of control measures that will reduce “risky behavior” (sufficient contact rates) within given populations. Alternately, they show, one can minimize the expected number of new infections over time, based on a dynamic allocation of resources into these control measures. The SIR model, in any form, shows the boundaries of the feasible region that any optimization problem for control measures would have to exist within. Thus, not only resource allocation problems, but control strategies could be evaluated over the modeled epidemic, once the changes that a control measure would have on a population are modeled appropriately.

3.9 Infectious Disease Eradication: Necessary Conditions and Strategies

3.9.1 Necessary Conditions for Eradication

According to Frank Fenner (2002: 9-13), the necessary conditions that make a particular infectious disease a good candidate for eradication are as follows in Table 8. These factors, while targeted to biological infections, do have correlates when one considers social epidemics.

Table 8: Factors contributing to eradication candidacy

Severity (high mortality rates or terrible side effects)
No animal reservoir of the virus/infectious agent
Slight incidence of undiagnosable cases
No recurrence of disease
Limited number of subtypes
Effective methods to confirm diagnosis
A stable, effective vaccine is available
Cases not infectious until diagnosable

It is apparent that for an eradication strategy to be possible, the infection needs to be something clearly defined, easily and reliably diagnosed, and an effective vaccine needs to be available to use in any eradication strategy. Additionally, a perceived need for the eradication strategy needs to exist as well. If the world community does not agree that the disease is a threat sufficient to warrant a costly intervention, then containing the epidemic will be much more difficult. Assuming that these conditions exist for an infection, then there are different strategies available to effect the eradication of the disease.

3.9.2 Eradication strategies

The most straightforward strategy to eradicate a disease is mass vaccination. This has been applied regionally with good results for many of the common diseases such as smallpox, polio, mumps, measles, rubella, and even chickenpox. However, such a

strategy depends on the existence of enough vaccine and enough economic resources to pay for the vaccine for the entire population. Additionally, until the infection is eradicated globally, mass vaccination still needs to be applied for all newborns into a population, so this is a high-cost but very effective strategy.

However, when resources are limited and eradication is still sought, ring vaccination is the strategy of choice. This strategy depends on surveillance in all the affected regions and quick diagnosis of new infections. Once new infections occur, the social network surrounding the newly infected person is inoculated, usually measured by a radius of a certain distance from the home of the infected person. Outside that radius, house-to-house searches are made for signs of infection that may have come out that far, and if infection is discovered, the same set of actions are applied starting there. This strategy targets infections as they occur, and allows a much more limited amount of resources, both funding and vaccines.

The success of ring vaccination is very much dependent upon the level of surveillance in the affected regions and the accuracy of diagnosis early on in an infection. This is why a disease prone to recurrence or difficult to diagnose early in its infectious stages would be difficult to eradicate with this sort of strategy. In the case of a less-obvious infection, the only effective strategy would be mass vaccination.

With any eradication strategy, the results may not be immediately observable. An infection that has become an epidemic usually will run its course. However, an eradication program will ensure that the disease does not become an endemic infection within a population, and will reduce the successive outbreaks of a disease. An eradication

program is a long-term solution to the problem of infectious diseases, not an immediate fix of the epidemic at hand.

3.10 Summary

The mathematical models of epidemiology are diverse and can incorporate many different interactions of variables involved. The simplest form of these models, the SIR epidemic model, shows over the short term how a single epidemic will play out through time. An endemic model, which requires more entry data, also gives more information about the long term and where endemic levels of a disease can be expected to settle.

These models can then be partitioned, either by age groups or by influence categories or by separate populations affected by the same epidemic. Conceivably, all these factors could be incorporated into one extremely large and complex model. These partitioned models, again, require more rates to determine the shape of the model, but they also give a more detailed picture of the epidemic in question when they are developed.

SIR models also can be used to inform the application of eradication strategies to epidemics. The different characteristics of an epidemic are what make it a better candidate for one or another eradication strategy. All of this information that is incorporated into mathematical models of epidemics has direct use for modeling of an epidemic of ideas, specifically the outbreak of extremist Islamist thought that encourages terrorism.

Chapter 4: Applying Epidemic Models and Strategies to Terrorism

This chapter's goal is to demonstrate how the mathematical epidemiological models developed in Chapter 3 may be applied to the epidemiology of ideas through several notional examples.

4.1 The epidemic SIR model of terrorism

The first challenge to modeling terrorism in a SIR model is to decide what is the population within which to model. Judging from the literature on the subject, the likely susceptible population to the terrorist mindset is that of males between the ages of 15 and 35, approximately. (Urdal, 2004) There are terrorist leaders who are much older than that, but those leaders seem to be the exception rather than the rule, and even if older people might be sympathetic to the extremist mindset, they appear to be less likely to act on it. Another characteristic of the susceptible population is that it exists within the Muslim world; for the case of our model we will choose to model with the young male demographic within a single region. This is a notional model just to exemplify how epidemiological concepts could be applied to better understand the dynamics of the spread of terrorism.

Within our young male regional population, then, we have to partition the population, N, into the classes of S, I, and R. Susceptible individuals are those who have

not yet been exposed to the terrorist mindset for a sufficient amount of time to become infected with it. For our model we will start with the assumptions that $s_0 = 1$ and $i_0 = r_0 = 0$. These will be starting conditions. Infective individuals are those who have been converted to an extremist Islamist mindset, who are ready and willing to be a part of the narrative that the terrorist leaders have presented them with. Finally, recovered individuals are those who have, in the past, been "infected" with the terrorist mindset but who have since changed their minds or become disaffected with that perspective on the world. We will proceed on the assumption that a recovered individual is not likely to become re-infected with the terrorist mindset.

Table 9: SIR Model Notation

S	Number of individuals within the S (susceptible) compartment
I	Number of individuals within the I (infectious) compartment
R	Number of individuals within the R (recovered) compartment
N	Number in total population (S + I + R = N)
s	Fraction of population that is susceptible (s = S/N)
i	Fraction of population that is infectious (i = I/N)
r	Fraction of population that is recovered (r = R/N)
R_0	Basic reproduction number (or rate)
σ	Contact number
R	Replacement number
t	Time, an independent variable

β	Average number of adequate contacts of an individual per time unit
γ	Rate of transfer out of I category

Unlike most of the diseases modeled by epidemiologists in SIR (or SEIR, or MSEIR) models, the time periods associated with the spread of terrorism are not typically measured in days. An adequate contact, one that would be sufficient to spread the "infection" to a susceptible individual, is likely to be one that is the result of weeks or months of exposure to an infectious individual. Additionally, once one is infected with the terrorist mindset, the time that one remains infectious is likely to be measured in months or years.

Table 10: Notional rates for terrorist SIR model

Time unit	6 months
β	2 per time unit on average
$1/\gamma$	10 time units (5 years) on average
σ	20 over the average "lifespan"

Using these rates in the differential equations below, the epidemic model of the infection of terrorism can be seen in Figure 24 and Figure 25. Bear in mind that the rates are notional numbers. A more extensive study of how a person becomes "infected" by terrorist ideology and a development of more rigorous numbers would be needed to

develop models to rely on and predict with. However, the notional numbers can give a sense of what the epidemic may look like when modeled mathematically.

$$\frac{ds}{dt} = -\beta is \qquad s(0) = s_0 \geq 0$$

$$\frac{di}{dt} = \beta is - \gamma i \qquad i(0) = i_0 \geq 0$$

$$\frac{dr}{dt} = \gamma i \qquad r(0) = r_0 \geq 0$$

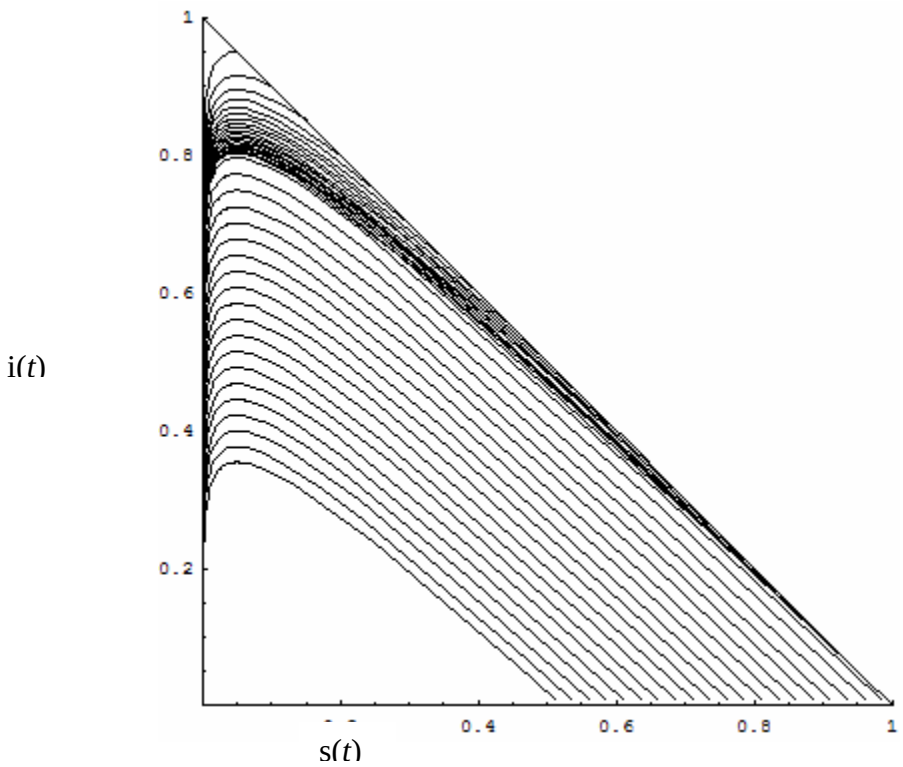


Figure 24: Terrorism SIR epidemic phase plane—s(*t*) vs. i(*t*)

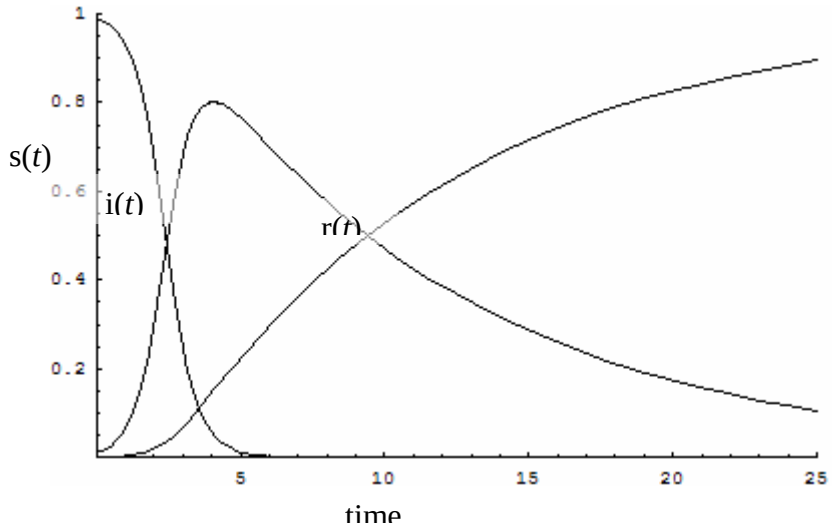


Figure 25: Terrorism SIR epidemic model over time

We can see that these assumptions leave us with a fairly steep epidemic curve, showing that the susceptible proportion of the population decreases rapidly and the infected portion peaks around five time periods, after which it slowly decreases. Modeling without birth and death rates, if we continued to model through time, the infected proportion should be expected to converge to zero eventually. This is, however, a simplistic representation of the population susceptible to terrorism. The problem of terrorism is not a short-term epidemic that can be expected to fade out, as experience has shown us over time, and thus modeling it as solely an epidemic, without accounting for the new people who will enter the system (and leave it), leaves an unrealistic picture of the problem at large. Our initial curves are most likely near correct, but when the rates of incoming and outgoing members of the system are added, the system will stabilize differently than an epidemic model would suggest, given its simplified assumptions.

3.2 The endemic SIR model of terrorism

An endemic model applied to terrorism gives us a longer time-scale look at what levels of infection are likely to become constant for the epidemic of terrorism. Assuming the rates that were used in the previous section, and adding $\mu = 1/40$ time periods (20 years), the equations below are used to model the epidemic. Since the population consists of a specific demographic group, we will consider the "lifespan" of a typical individual within the population to be 20 years, and use that number for $1/\mu$ in the endemic version of the SIR model. The differential equations and rates below set up the endemic model of terrorist ideology.

$$\frac{ds}{dt} = \mu - \mu s - \beta is \qquad s(0) = s_0 \geq 0$$

$$\frac{di}{dt} = \beta is - (\gamma + \mu)i \qquad i(0) = i_0 \geq 0$$

$$\frac{dr}{dt} = \gamma i - \mu r \qquad r(0) = r_0 \geq 0$$

Table 11: Notional rates for endemic terrorism model

Time unit	6 months
β	2 per time unit on average
$1/\gamma$	10 time units (5 years) on average
$1/\mu$	40 time units (20 years)
σ	16 over the average "lifespan"

Since the model is exactly the same, except for the addition of rates into and out of the system, the model should look very similar to the epidemic model developed in the previous section. However, because this is an endemic model, it will converge to the endemic equilibrium of the infection, and will give a sense of what these numbers will mean in the long term given that rates are close to the notional ones chosen. The endemic equilibrium will give a sense of steady-state percentages of the population to expect to be infected with the terrorist ideology. This means that one can guess at levels of willing participants in terrorist activities, as well as how many people continue to be within the susceptible population. This model, if given reasonable estimates, could inform decision-makers about the potential effects of counterterrorism strategies when they encounter several alternatives.

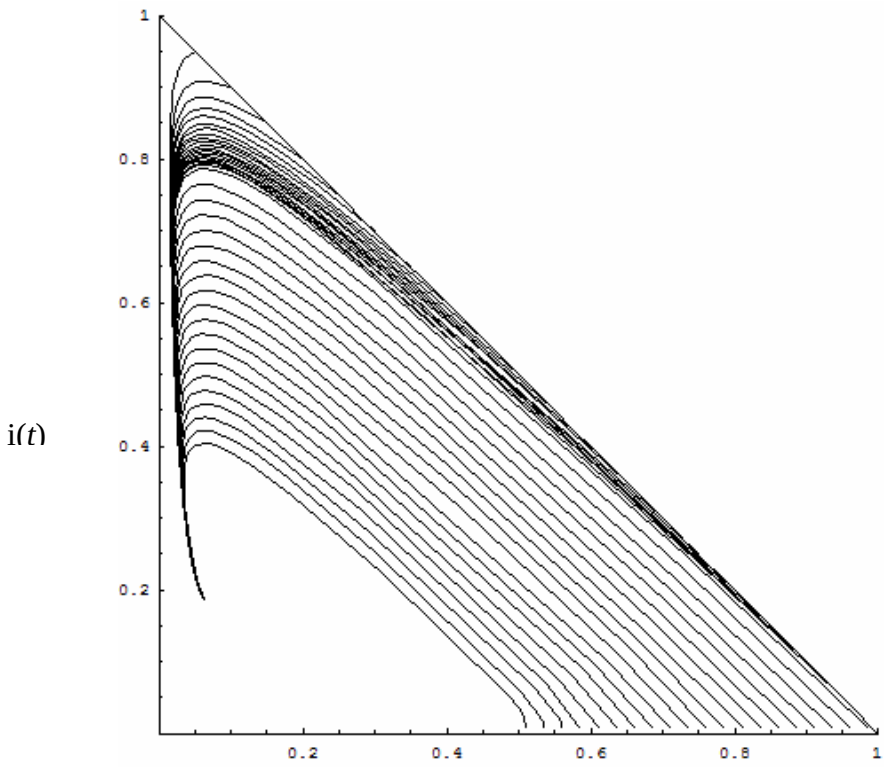


Figure 26: Terrorism SIR endemic phase plane—s(*t*) vs. i(t)

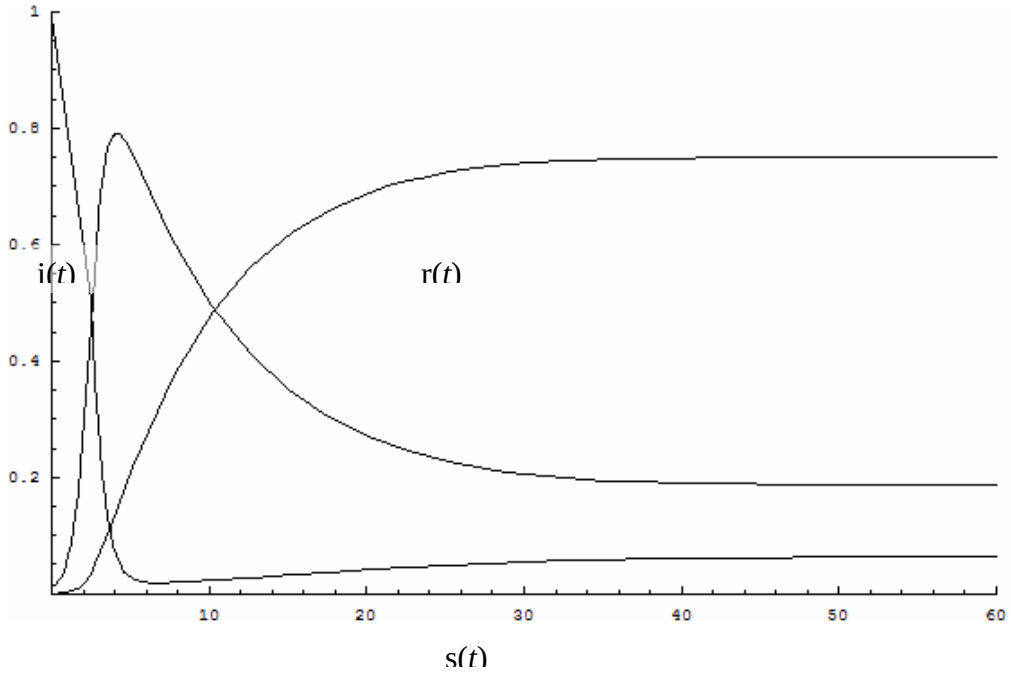


Figure 27: Terrorism SIR endemic model over time

Using an endemic model shows more dynamics within the system, with the interesting outcome that the infected population, given these vital dynamics, can be expected to reach a steady-state distribution (the endemic equilibrium). This does show that the initial peak of terrorist mindset infection is the greatest; if our assumptions are reasonable, it would appear that it is likely that within a given population, the susceptible population will be rapidly exposed. After the initial exposure, the proportions of those likely to act on the terrorist mentality will decline until terrorists can be expected to make up only twenty percent or so of the population at any given time. While this is not good news—one might wish to see a smaller proportion of infected individuals—it does show that the initial surge of terrorist recruits cannot be something sustained.

Assuming that the first 2.5 years (5 time units) of exposure to terrorist mindsets have already elapsed, it is conceivable that for any given population of young Muslim males, the "infected" proportions are already declining, rather than continuing to peak as some literature suggests.

The benefit of such models is that one can conduct sensitivity analyses with regards to the targeting of parameters such as forces of infection and immunization, where even slight perturbations might make a great difference in the behavior of the model. It will be necessary to study the sociological makeup of the region of interest and the terrorists operating within that region to come up with numbers that have more objective value. With a sense of steady-state levels within the population, the next challenge is to model the system partitioned into its various component populations, so

that more complex interactions and dynamics within the system will become apparent in the model.

4.3 Two age category model

For this model we subdivide the 15 to 35 age group into two equal portions. Category 1 will be the 15-25 age group and Category 2 will be 25-35. We will assume that we have a youth bulge (Urdal, 2004) weighted on the younger side of the spectrum, and thus our percentages will be skewed accordingly over our 20-year spectrum. We will assume that a younger person is less able to "infect" an older person, and that an older person is less likely to become infected from either group. As well, we will assume a longer time period span for the infection of an older person—if they become convinced of the validity of their cause, they will be less likely to change their minds again later.

The differential equations below and the rates in Table 12 set up the age category model for the epidemic of terrorist ideology.

$$\frac{d\,\mathrm{s}_1}{dt} = (c_1 + d_1)P_1 - [\lambda_1 + c_1 + d_1]\mathrm{s}_1$$

$$\frac{d\,\mathrm{i}_1}{dt} = \lambda_1 \mathrm{s}_1 - [\gamma_1 + c_1 + d_1]\mathrm{i}_1$$

$$\frac{d\,\mathrm{r}_1}{dt} = \gamma_1 \mathrm{i}_1 - [c_1 + d_1]\mathrm{r}_1$$

$$\frac{d\,\mathrm{s}_2}{dt} = c_1 \mathrm{s}_1 - [\lambda_2 + c_2 + d_2]\mathrm{s}_2$$

$$\frac{d\mathrm{i}_2}{dt} = c_1 \mathrm{i}_1 + \lambda_2 \mathrm{s}_2 - [\gamma_2 + c_2 + d_2]\mathrm{i}_2$$

$$\frac{d\mathrm{r}_2}{dt} = c_1 \mathrm{r}_1 + \gamma_2 \mathrm{i}_2 - [c_2 + d_2]\mathrm{r}_2$$

Table 12: Age category rates for terrorism SIR model

$P_1 = 0.7$	$P_2 = 0.3$
$\beta_{11} = 1$	$\beta_{12} = 3$
$\beta_{21} = 0.1$	$\beta_{22} = 0.5$
$d_1 = \frac{1}{200}$	$d_2 = \frac{1}{100}$
$c_1 = \frac{1}{20}$	$c_2 = \frac{c_1 P_1}{P_2} - d_2 = \frac{8}{75}$
$\gamma_1 = \frac{1}{8}$	$\gamma_2 = \frac{1}{14}$

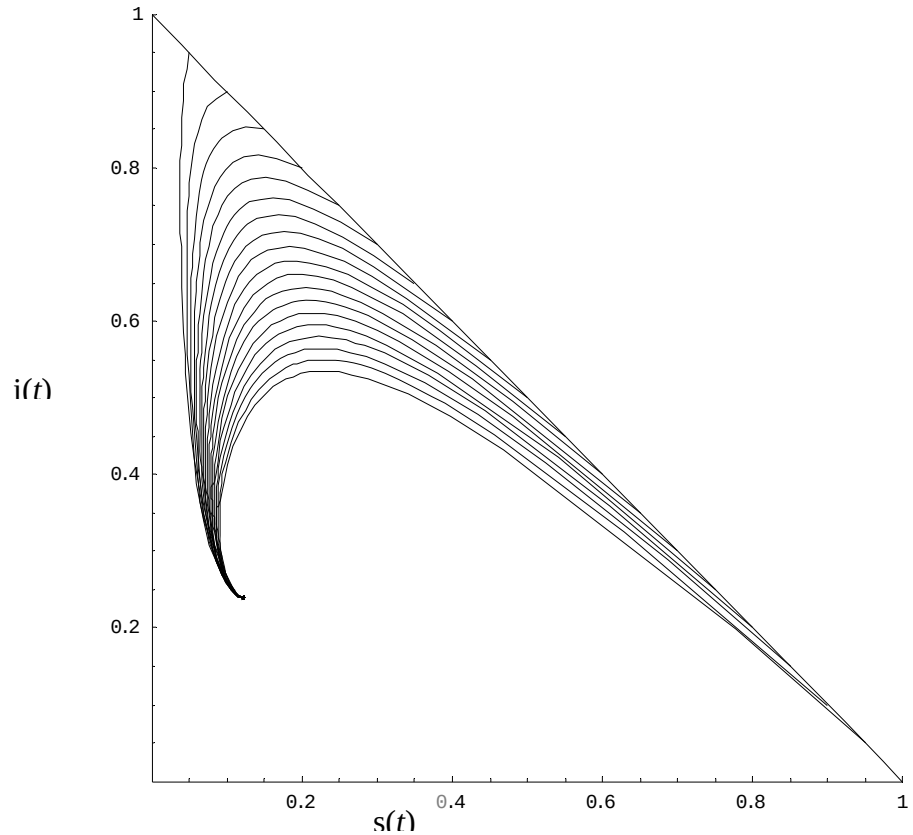


Figure 28: Two age category terrorism SIR model phase plane

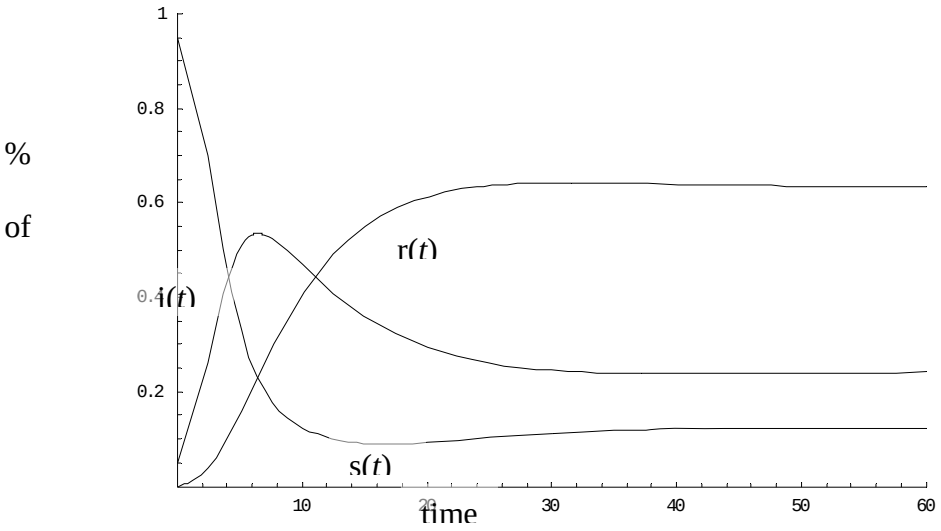


Figure 29: Two age category terrorism SIR model over time

Figure 28 and Figure 29 show the shape of the model when the rates from Table 12 are used within the partitioned model. The entire population is accounted for in these figures, and it is evident that the model looks different than the endemic model. Adding the complexity of interactions within states makes the model look different than the endemic model developed in the section before. Each age category, as well, looks different when it is modeled separately, as one can see in the figures below. Since these are modeled as proportions of the total population, the partitioned models have different ranges for the values within the graphs.

The younger age group, modeled this way, will have a higher steady-state incidence of both susceptible and infectious populations. The older group will have a much lower incidence of both, a fact that can be clearly seen as its levels over time are shown in Figure 33.

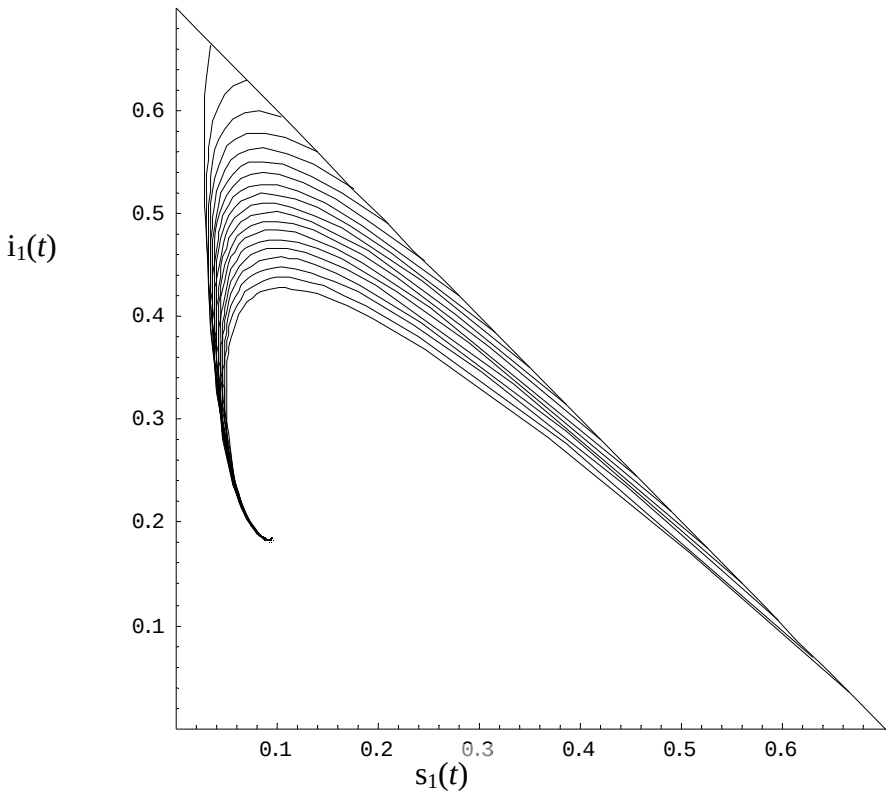


Figure 30: Age category 1 phase plane

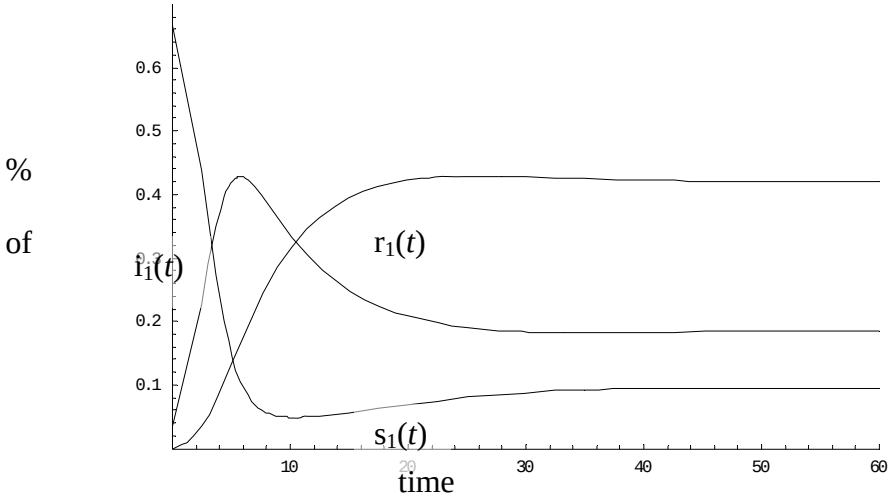


Figure 31: Age category 1 over time

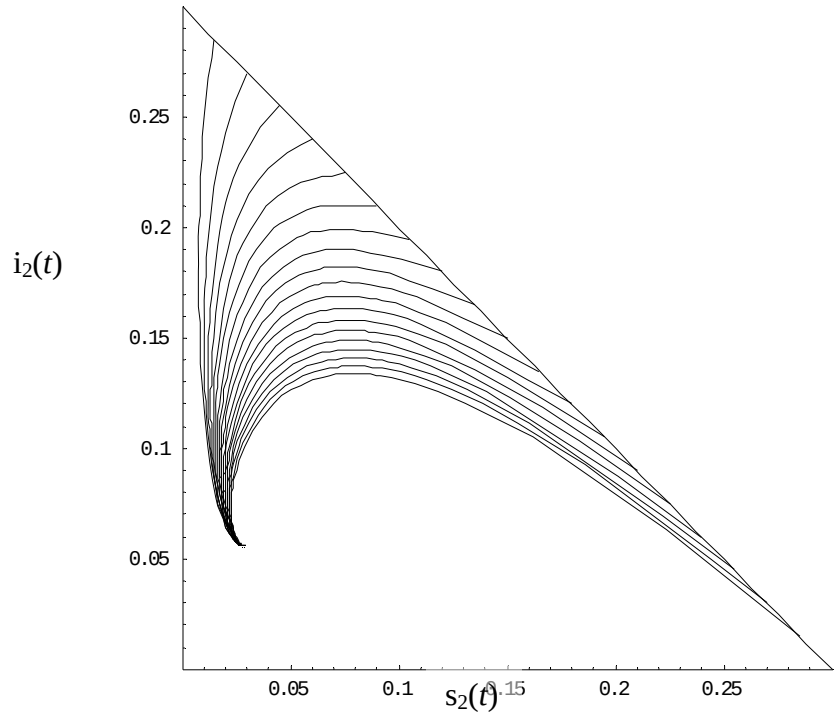


Figure 32: Age category 2 phase plane

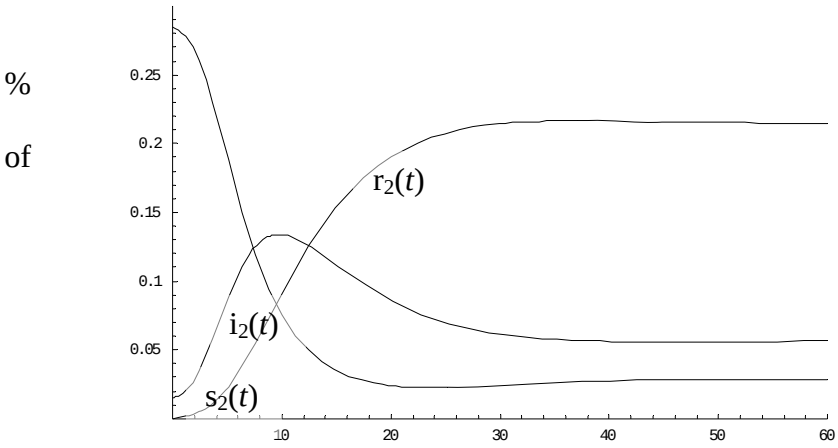


Figure 33: Age category 2 over time

From these notional numbers, one can see that our assumptions have changed the SIR model. The total population stabilizes to an endemic equilibrium that is very close to the numbers we achieved with the simpler endemic model. However, when one looks at the model by category, two very different dynamics can be observed. It is evident that a smaller chance of infection (β value) will curtail the effects of the epidemic to a large degree, though in this case the long period of infectiousness (γ value) still creates the peak of infectious population that is associated with an epidemic.

4.4 Terrorism model partitioned by influence, including "immunity"

Using the methodology developed for influence category models, we again partition the population, this time into category 1, normal, and category 2, highly influential, individuals. The second category could be thought of as people who have a large degree of connections to others, unusual persuasiveness, or merely those who can effectively use the media to gain the sufficient amount of exposure to "infect" or "immunize" their audience. An imam in a large mosque might be one such person, or the leader of a terrorist network who produces tapes that are distributed to local television networks. While this is only based on two categories of people, an argument could be made for having more partitions, based on local influence.

While the first category is overwhelmingly the largest, the second category has such a large ability to persuade others that it makes its mark even though it has small numbers. We also assume that those with an extremist viewpoint will have more

likelihood of “infecting” people than those with a more moderate viewpoint have to “immunize” people. We base this assumption on the observation that the current state of the Islamic world seems to favor apocalyptic paradigms, and most moderate interpretations of Islam tend to be less apocalyptic in character than are the fundamentalist or extremist versions.

The differential equations below and the rates in Table 13 set up the influence category with immunity model of the terrorist epidemic.

$$\frac{d\,s_1}{dt} = (c_1 + d_1)P_1 - [\lambda_1 + \kappa_1 + c_1 + d_1]s_1$$

$$\frac{d\,i_1}{dt} = \lambda_1 s_1 - [\gamma_1 + c_1 + d_1]i_1$$

$$\frac{d\,r_1}{dt} = \kappa_1 s_1 + \gamma_1 i_1 - [c_1 + d_1]r_1$$

$$\frac{d\,s_2}{dt} = c_1 s_1 - [\lambda_2 + \kappa_2 + c_2 + d_2]s_2$$

$$\frac{d\,i_2}{dt} = c_1 i_1 + \lambda_2 s_2 - [\gamma_2 + c_2 + d_2]i_2$$

$$\frac{d\,r_2}{dt} = c_1 r_1 + \kappa_2 s_2 + \gamma_2 i_2 - [c_2 + d_2]r_2$$

Remember that λ and κ are defined as follows:

$$\lambda_1 = \beta_{11} i_1 + \beta_{12} i_2 \qquad \lambda_2 = \beta_{21} i_1 + \beta_{22} i_2$$

$$\kappa_1 = (\alpha_{11} r_1 + \alpha_{12} r_2) s_1 \qquad \kappa_2 = (\alpha_{21} r_1 + \alpha_{22} r_2) s_2$$

Table 13: Terrorism influence category model with "immunity" rates

$P_1 = 0.99$	$P_2 = 0.01$
$\beta_{11} = 1$	$\beta_{12} = 200$
$\beta_{21} = 1$	$\beta_{22} = 50$
$\alpha_{11} = 1$	$\alpha_{12} = 75$
$\alpha_{21} = 1$	$\alpha_{22} = 25$
$d_1 = \frac{1}{100}$	$d_2 = \frac{1}{100}$
$c_1 = \frac{1}{200}$	$c_2 = \frac{c_1 P_1}{P_2} - d_2 = \frac{97}{200}$
$\gamma_1 = \frac{1}{10}$	$\gamma_2 = \frac{1}{20}$

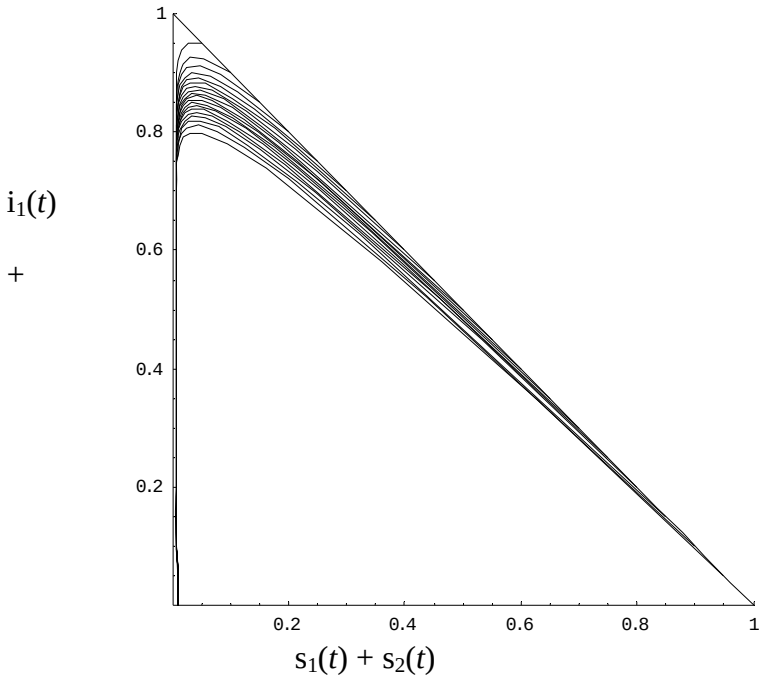


Figure 34: Terrorism influence model with "immunity" phase plane

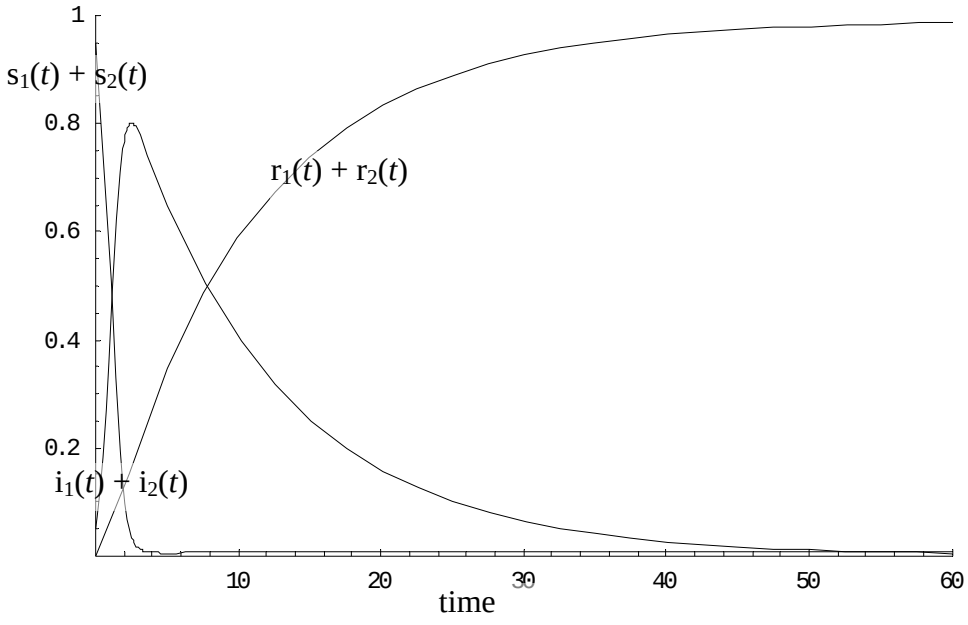


Figure 35: Terrorism influence category model with "immunity" over time

In this model, the breakdown diagrams do not differ significantly in shape from the diagrams for the entire population, so they have been omitted.

The first item of note in this model is that the overall shape of the diagrams is very similar to that of the endemic model produced earlier. The difference we can see here is that the endemic model found a steady-state distribution with proportions of the population in a fixed ratio to one another, while this model shows an eventual trend of susceptible and infected populations down to zero, or very close. In one sense, this model incorporates a time limit into the epidemic, because if one assumes that any recovered individual will have the possibility of having an immunizing effect on susceptible individuals, the gradual trend toward a larger and larger recovered class (recovery is, after all, an absorbing state) will create its own epidemic of ideas. This secondary epidemic of ideas—but ideas that are not considered dangerous to the Western world—can be thought of as the eventual replacement for the dangerous infection of terrorist ideology.

In a sense, then, introducing a counter-paradigm to that of the terrorism propagators would be an eradication strategy. Just as biological epidemics have been eradicated by strategies created by the world health community (Dowdle, 1999: 23), it stands to reason that a social epidemic of extremist ideology could be eliminated by an eradication strategy. Of course, the stakes are high, and finding an “inoculation” that has a high probability to take hold would be difficult. Typical information operations and psychological operations that focus on promoting West-friendly ideas might never

achieve the “stickiness” needed to combat the much more tempting extremist ideology. (Mazarr, 2004)

One can see from these models, however, that extremist ideology, like any contagion, could hold within itself the seeds of its own destruction, were the right approach toward eliminating it employed. The key to eliminating the steady-state levels of terrorists or, in our models, “infectious” individuals is to introduce a counter-epidemic of ideas that has a similar ability to propagate within the Muslim world. With such a strategy, the problematic epidemic of terrorist ideology might come to an end within a reasonable timeframe.

4.5 Additional applications of a partitioned model

A partitioned model could also be used to show the epidemic as it affects multiple populations over multiple time periods (Zaric, 2002) In this case the interactions are more complex, because flows between the partitions of the population are rarely going to be in one direction only. This will require another set of rates for interactions between populations. Additionally, all of these ideas could be used together to create a more complex problem. However, the more factors that are included into the model, the more difficult the solution becomes.

SIR models have been used to define the boundaries of the feasible region for problems relating to epidemic control. When this has been done, one can start using the feasible solutions to an epidemic distribution to develop different controls over

epidemics. Some of these are resource allocation problems and some discuss minimization of new infections over a time horizon. With an understanding of the factors involved in the modeling of epidemics, operations research analysts could apply this sort of thinking to a model of the spread of terrorist ideology and potentially work out a strategy to limit and even to eradicate the ideology of terrorism.

4.6 Eradication strategies applied to the epidemic of terrorist ideology

As one considers the spread of the ideology of terrorism, it becomes apparent that this infection of ideas spreads like a biological epidemic. With that in mind, the strategies that have been used to eradicate infectious diseases take on new meaning. If immunization has a counterpart in the realm of ideas, it is likely the propagation of an alternate mindset from that which backs terrorism, something that is not necessarily pro-Western, but is also not virulently anti-Western in nature. In short, the West needs to seek an alternate epidemic of ideas for the Islamic world that the people in the Western world can live with. This is not just a marketing scheme, however.

The idea of mass-marketing a particular idea, or new epidemic, is an equivalent to the mass immunization strategy. Both require a good budget, extensive research and understanding of the markets into which they wish to inject their message, and adaptation and creativity to keep up with the easily-bored audience of the message in question. Because of these needs, a mass-marketing approach is probably not an optimal strategy to contain the epidemic of terrorism.

Ring immunization, though, has a promising correlation to the epidemic of ideas. It would require careful surveillance of the areas in question, however, to be workable. Any sign of "infection," such as a person caught participating in terrorist activities, and the social network surrounding that person should immediately be explored for signs of infection. Law enforcement agencies likely already do this—albeit unofficially—as they track down the terrorist cells that have perpetrated acts of terrorism. There could be a careful inspection of the first-degree associates of the infected person, and then an alternate idea, tailored to the local culture, could be introduced into that group. Second-degree associates should be carefully surveyed and tracked for signs of infection, and treated accordingly.

Ring immunization, or its ideational equivalent, has the potential to be tailored to the local conditions that surround outbreaks of terrorist ideology. Because the Islamic world contains such a wide variety of subcultures, this would give any eradication strategy a greater ability to adapt to each subculture and region. The epidemic of terrorist ideology could be slowed down in this manner and eventually stopped, if the program were carefully applied.

4.7 Summary

Looking at terrorist ideology as an epidemic makes it possible to apply the models used by epidemiologists. SIR models are good descriptors of epidemics, and can be changed in order to increase complexity and show more interactions that exist within

populations in question. With notional rates, these models show the sorts of things to look at and where the target populations might be for any eventual eradication strategy.

Just as eradication strategies apply to biological epidemics, they have correlates when it comes to ideational epidemics. In this sense, a mass-marketing campaign could be considered to be like a mass immunization program. Accordingly, a more targeted and surveillance-rich ring immunization strategy would be similar to a program that followed the social networks of known terrorists and tailored "immunization" ideas to the surrounding networks of people. Modeling and eradication of the social epidemic of terrorism may be a far-reaching goal, but it provides a fresh paradigm for the study of how to fight against the spread of terrorism.

Chapter 5: Conclusions and Recommendations

5.1 Summary

People who wish to solve problems associated with the spread of terrorist ideology have taken many different approaches to their solutions. Within Western culture, it is a common assumption that the struggle against terrorists is a war similar to wars that have been fought against other ideologies. This assumption creates a problem, however, because the cultural barriers between radical Islamists and Westerners are great. Rather than modeling problems associated with the War on Terror in a way that incorporates Western expectations about the nature of war and conflict, it would make more sense to see the spread of extremist Islamic ideology as an epidemic of ideas.

With this core idea in mind, this thesis has explored the methodologies associated with epidemiology. Particularly, it has focused on the mathematical models used by epidemiologists to model the spread of epidemics. Using different variants of the basic deterministic mathematical model in epidemiology, the SIR model, it has shown the way that different factors that influence the spread of epidemics change the way that an epidemic can be modeled. From short-term and simple in the SIR epidemic model to complex partitioned models that show interactions between age groups or influence categories, this paper looks through the types of models one can develop to understand the dynamics of an epidemic. Additionally, epidemic eradication strategies are considered and described, along with when and where they would work best.

From there the thesis continues on to apply the models developed to the specific problem of the epidemic of terrorist ideology. Each type of model from the simple to the complex is considered, and applied with notional initial rates. From there, it discusses the application of different eradication strategies to the epidemic of terrorist ideology. While a mass-marketing strategy might be the most obvious approach, a targeted ring strategy with adaptability to local subcultures might have a greater effect. While no strategy will immediately end the spread of terrorist ideology, either of these could eventually end the epidemic.

This is a first look at how to approach the question of terrorism and the fight against it from another angle. Since almost all of the problem-solving models that have been developed to fight terrorism incorporate ideas about the struggle being a war of sorts, this is a new set of assumptions to use to base models on. Instead of talking about target value, risk assessment, and resource allocation, it might be better to think about eradication strategies, control measures, and threshold numbers. Since the current paradigms that back studies of counterterrorism seem to be breaking down, this new approach might be of value as another perspective on the fight.

5.2 Recommendations

5.2.1 Populating SIR models with demographic data

There are an abundance of possible future directions for research into the application of epidemiology to the study of terrorism. Even the models presented within this paper need to be populated with demographic data. The rates of infection, recovery, contact rates, and sufficient exposure would need to be collected from intelligence or surveys done in the region of interest by social scientists. Demographic data about the regions of interest and data about the sociopolitical structure of these regions should be studied and applied where necessary. An abundance of data exists; it would take looking into which studies are applicable and how the studies could be quantified into meaningful numbers for epidemiological models.

5.2.2 Other epidemiological models

SIR models are not the extent of models of epidemics that exist within the realm of epidemiology. Indeed, this overview barely scratches the surface of the models available. It is intended to be an example of how the tools of epidemiologists could be used to study and understand terrorism. Another type of epidemiological models that would be of interest are geographical models that show the spread of an infection throughout a region with contour plots for each time period. These have been used with biological epidemics to track the source of an infection, and might have a parallel use for ideational epidemics. Used over a “landscape” of social networks, this might clarify the source nodes for an infection and perhaps offer insight into where future infections might develop.

Even the compartmentalized model could be developed further, so that instead of a basic SIR model, some vertical transmission of immunity (the category M) is incorporated into the model. This would model the influence that parents would have over their offspring. Additionally, a latent period, where an individual may be infected but is not yet infectious (the category E) could shed light on the model.

5.2.3 Using other applications of epidemiology

The applications of epidemiology that have been developed by marketers should be studied. “Viral marketing” might well be incorporated in the eventual eradication strategy applied to the spread of extremist Islamist ideology. Marketers have spent more time studying ideational epidemics than any other group of people, and thus their methods and findings about epidemics of ideas are the best source of data and strategies for someone who would seek to apply the idea to another discipline. While the study of counterterrorism must be kept distinct from trend research, there are some valid correspondences between these disciplines as well.

5.2.4 Using SIR models for optimization

Some applications of SIR models of biological epidemics include using the model as a definition of the feasible region within which to optimize some function of the variables in the system. For instance, one paper optimizes resource allocation by showing

the effects that several control measures would have on a SIR model and using a function of the infected population as the objective value function. One seeks to minimize the numbers of infected within the model by applying the control measures that are constrained by budgetary requirements. (Zaric and Brandeau, 2002) Similarly, one could model different eradication strategies and their perceived effects, and optimize using the SIR model to show what the effects might be. The SIR model is a different conception of the shape of terrorism within a region, and may have some applicability as a background for other often-used methods of problem-solving. The idea that terrorism could be conceived of as an epidemic of ideas crosses disciplinary boundaries and will require some synthesis of disciplines and approaches in order to be well applied, but it carries with it enormous promise as well.

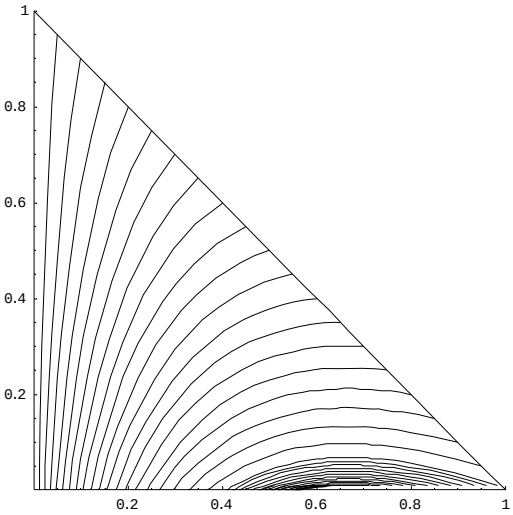
5.3 Conclusion

Reassessing our assumptions about terrorism and how to fight it has the potential to change all of the approaches to fighting terrorists. Since the United States is now engaged in a War on Terror, this is a good avenue to explore. It is evident that, while some of the strategies of warfare that have been used to fight the war on terrorism are effective, many are not, and the struggle continues to become increasingly more bogged down. The military is notorious for “fighting the last war,” because that is the war that we understand, but paradigms from the Cold War break down when applied to the struggle against extremist Islamist ideologies. If we consider terrorism the symptom of a disease,

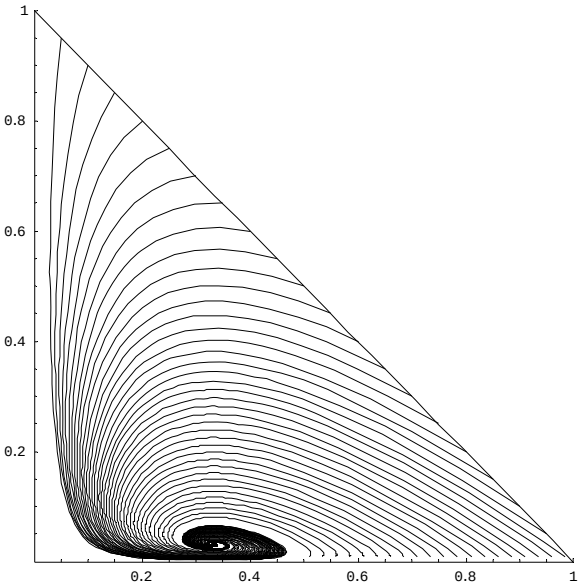
and model that disease as we would an epidemic, it might well cast light on the problems with the current state of affairs and highlight better courses of action for the future. A new perspective on an ongoing problem cannot be a detriment to understanding that problem.

Appendix: Mathematica 5.2 Code for SIR plots

```
(*This plots the phase plane: i(t) against s(t) for the epidemic SIR model*)
beta=2.25; gamma=1.5;
p0=Plot[1-x,{x,0,1},DisplayFunction→Identity];
sols={};
plots={p0};
For[k=1,k<20,
    sols=Append[sols,NDSolve[{s'[t]==-beta*i[t]*s[t],i'[t]==beta*i[t]*s[t]-gamma*i[t], s[0]==1-
0.05*k,i[0]==0.05*k},{s,i},{t,0,50}]];
plots=Append[plots,ParametricPlot[Evaluate[{s[t],i[t]}/.sols[[k]]],{t,0,50},DisplayFunction→Identity]]
;
    k++];
For[k=20,k<40,
    sols=Append[sols,NDSolve[{s'[t]==-beta*i[t]*s[t],i'[t]==beta*i[t]*s[t]-gamma*i[t], s[0]==0.51+(k-
20)*0.025,i[0]==0.01},{s,i},{t,0,50}]];
plots=Append[plots,ParametricPlot[Evaluate[{s[t],i[t]}/.sols[[k]]],{t,0,50},DisplayFunction→Identity]]
;
    k++];
Show[plots,AspectRatio→1,PlotRange→{{0,1},{0,1}},DisplayFunction→$DisplayFunction]
Clear[solution]
```



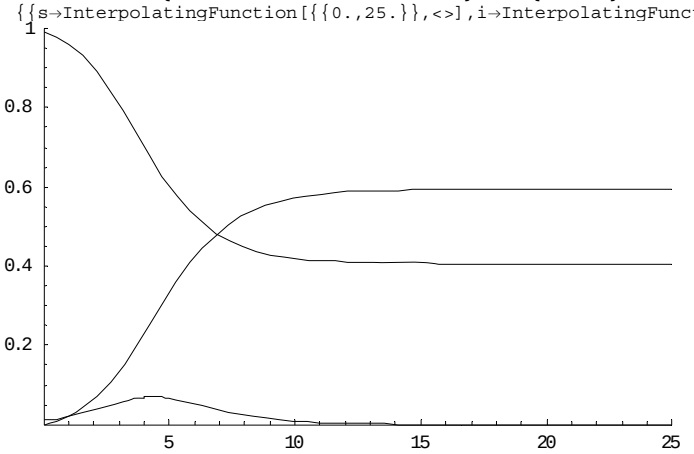

```
 -Graphics-
 (*This plots the endemic phase plane*)
beta=21/20; gamma=1/3; mu = 1/60;
p0=Plot[1-x,{x,0,1},DisplayFunction→Identity];
sols={};
plots={p0};
For[k=1,k<20,
    sols=Append[sols,NDSolve[{s'[t]==-beta*i[t]*s[t]+mu-mu*s[t],i'[t]==beta*i[t]*s[t]-gamma*i[t]-
mu*i[t], s[0]==1-0.05*k,i[0]==0.05*k},{s,i},{t,0,200}]];
plots=Append[plots,ParametricPlot[Evaluate[{s[t],i[t]}/.sols[[k]]],{t,0,200},DisplayFunction→Identity]
];
    k++];
For[k=20,k<40,
    sols=Append[sols,NDSolve[{s'[t]==-beta*i[t]*s[t]+mu-mu*s[t],i'[t]==beta*i[t]*s[t]-gamma*i[t]-
mu*i[t], s[0]==0.51+(k-20)*0.025,i[0]==0.01},{s,i},{t,0,100}]];
plots=Append[plots,ParametricPlot[Evaluate[{s[t],i[t]}/.sols[[k]]],{t,0,100},DisplayFunction→Identity]
];
    k++];
Show[plots,AspectRatio→1,PlotRange→{{0,1},{0,1}},DisplayFunction→$DisplayFunction]
Clear[solution]
```



-Graphics-

```
(*This plots s[t], i[t] and r[t] over time for the epidemic SIR model*)
beta=2.25; gamma=1.5;
NDSolve[{s'[t]==-beta*i[t]*s[t], i'[t]==beta*i[t]*s[t]-gamma*i[t], s[0]==.99, i[0]==.01}, {s,i},
{t,0,25}]
Plot[Evaluate[{s[t], i[t], 1-(s[t]+ i[t])}/.%],{t,0,25}, PlotRange→{{0,25},{0,1}}]
```

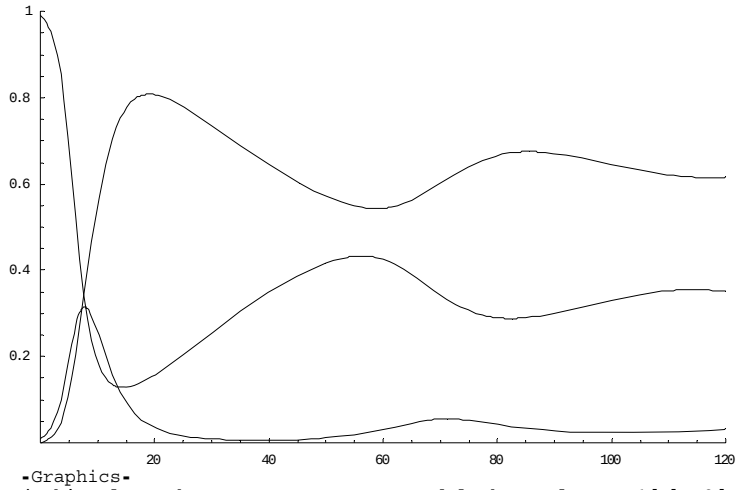
{{s→InterpolatingFunction[{{0.,25.}},<>],i→InterpolatingFunction[{{0.,25.}},<>]}}



-Graphics-

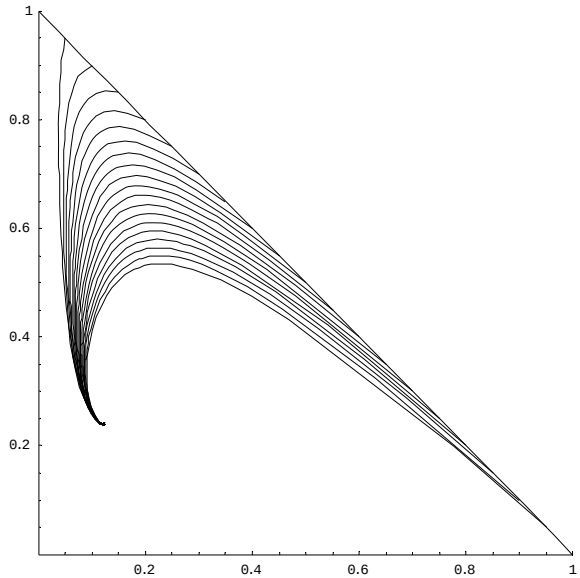
```
(*This plots s[t], i[t] and r[t] over time for the endemic SIR model*)
beta=21/20; gamma=1/3; mu = 1/60;
NDSolve[{s'[t]==-beta*i[t]*s[t]+mu-mu*s[t], i'[t]==beta*i[t]*s[t]-gamma*i[t]-mu*i[t], s[0]==.99,
i[0]==.01}, {s,i}, {t,0,120}]
Plot[Evaluate[{s[t], i[t], 1-(s[t]+ i[t])}/.%],{t,0,120}, PlotRange→{{0,120},{0,1}}]
```

{{s→InterpolatingFunction[{{0.,120.}},<>],i→InterpolatingFunction[{{0.,120.}},<>]}}



-Graphics-

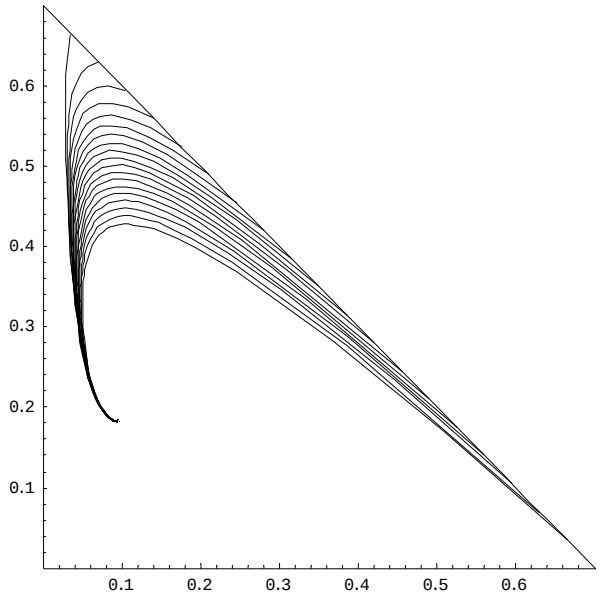
```
(*This plots the two age group SIR model phase plane: s1[t]+s2[t] vs. i1[t]+i2[t]*)

beta11=1;beta12=3;beta21=0.1;beta22=0.5; gamma1=1/8;gamma2=1/14; d1=
1/200;d2=1/100;c1=1/20;c2=8/75;P1=.7;P2=.3;
p0=Plot[1-x,{x,0,1},DisplayFunction→Identity];
sols={};
plots={p0};
For[k=1,k<20,
    sols=Append[sols,NDSolve[{s1'[t]==(c1+d1)*P1-(beta11*i1[t]+beta12*i2[t])*s1[t]-
(c1+d1)*s1[t],s2'[t]==c1*s1[t]-(beta21*i1[t]+beta22*i2[t])*s2[t]-
(c2+d2)*s2[t],i1'[t]==(beta11*i1[t]+beta12*i2[t])*s1[t]-
(gamma1+c1+d1)*i1[t],i2'[t]==c1*i1[t]+(beta21*i1[t]+beta22*i2[t])*s2[t]-(gamma2+c2+d2)*i2[t], s1[0]==P1-
0.05*P1*k,s2[0]==P2-0.05*P2*k,i1[0]==0.05*P1*k,i2[0]==0.05*P2*k},{s1,s2,i1,i2},{t,0,200}]];
plots=Append[plots,ParametricPlot[Evaluate[{s1[t]+s2[t],i1[t]+i2[t]}/.sols[[k]]],{t,0,200},DisplayFunc
tion→Identity]];
    k++];
Show[plots,AspectRatio→1,PlotRange→{{0,1},{0,1}},DisplayFunction→$DisplayFunction]
Clear[sols]
```



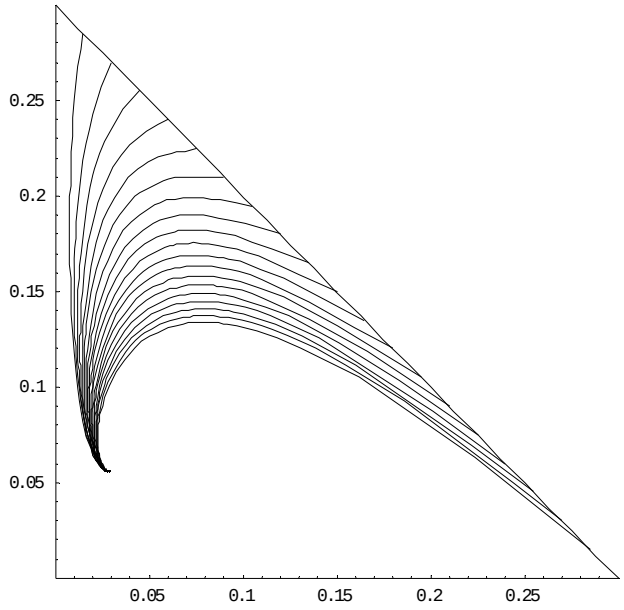
-Graphics-

```
(*This plots the two age group SIR model phase plane: s1[t] vs. i1[t]*)

beta11=1;beta12=3;beta21=0.1;beta22=0.5; gamma1=1/8;gamma2=1/14; d1=
1/200;d2=1/100;c1=1/20;c2=8/75;P1=.7;P2=.3;
p0=Plot[P1-x,{x,0,P1},DisplayFunction→Identity];
sols={};
plots={p0};
For[k=1,k<20,
    sols=Append[sols,NDSolve[{s1'[t]==(c1+d1)*P1-(beta11*i1[t]+beta12*i2[t])*s1[t]-
(c1+d1)*s1[t],s2'[t]==c1*s1[t]-(beta21*i1[t]+beta22*i2[t])*s2[t]-
(c2+d2)*s2[t],i1'[t]==(beta11*i1[t]+beta12*i2[t])*s1[t]-
```

```
(gamma1+c1+d1)*i1[t],i2'[t]==c1*i1[t]+(beta21*i1[t]+beta22*i2[t])*s2[t]-(gamma2+c2+d2)*i2[t], s1[0]==P1-
0.05*P1*k,s2[0]==P2-0.05*P2*k,i1[0]==0.05*P1*k,i2[0]==0.05*P2*k},{s1,s2,i1,i2},{t,0,200}]];
plots=Append[plots,ParametricPlot[Evaluate[{s1[t],i1[t]}/.sols[[k]]],{t,0,200},DisplayFunction→Identit
y]];
    k++];
Show[plots,AspectRatio→1,PlotRange→{{0,P1},{0,P1}},DisplayFunction→$DisplayFunction]
Clear[sols]
```




```
 -Graphics-
 (*This plots the two age group SIR model phase plane: s2[t] vs. i2[t]*)
beta11=1;beta12=3;beta21=0.1;beta22=0.5; gamma1=1/8;gamma2=1/14; d1=
1/200;d2=1/100;c1=1/20;c2=8/75;P1=.7;P2=.3;
p0=Plot[P2-x,{x,0,P2},DisplayFunction→Identity];
sols={};
plots={p0};
For[k=1,k<20,
    sols=Append[sols,NDSolve[{s1'[t]==(c1+d1)*P1-(beta11*i1[t]+beta12*i2[t])*s1[t]-
(c1+d1)*s1[t],s2'[t]==c1*s1[t]-(beta21*i1[t]+beta22*i2[t])*s2[t]-
(c2+d2)*s2[t],i1'[t]==(beta11*i1[t]+beta12*i2[t])*s1[t]-
(gamma1+c1+d1)*i1[t],i2'[t]==c1*i1[t]+(beta21*i1[t]+beta22*i2[t])*s2[t]-(gamma2+c2+d2)*i2[t], s1[0]==P1-
0.05*P1*k,s2[0]==P2-0.05*P2*k,i1[0]==0.05*P1*k,i2[0]==0.05*P2*k},{s1,s2,i1,i2},{t,0,200}]];
plots=Append[plots,ParametricPlot[Evaluate[{s2[t],i2[t]}/.sols[[k]]],{t,0,200},DisplayFunction→Identit
y]];
    k++];
Show[plots,AspectRatio→1,PlotRange→{{0,P2},{0,P2}},DisplayFunction→$DisplayFunction]
Clear[sols]
```

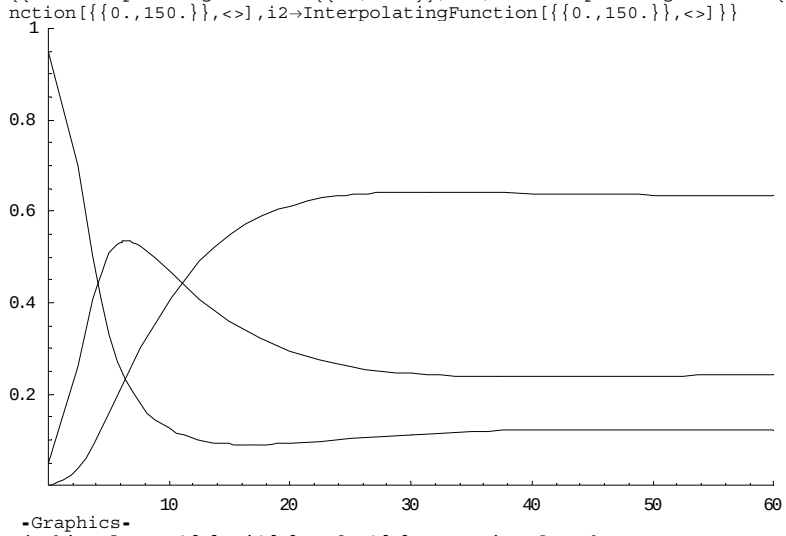


-Graphics-

(*This plots s[t], i[t] and r[t] over time for the age category SIR model*)

```
beta11=1;beta12=3;beta21=0.1;beta22=0.5; gamma1=1/8;gamma2=1/14; d1=
1/200;d2=1/100;c1=1/20;c2=8/75;P1=.7;P2=.3;
NDSolve[{s1'[t]==(c1+d1)*P1-(beta11*i1[t]+beta12*i2[t])*s1[t]-(c1+d1)*s1[t],s2'[t]==c1*s1[t]-
(beta21*i1[t]+beta22*i2[t])*s2[t]-(c2+d2)*s2[t],i1'[t]==(beta11*i1[t]+beta12*i2[t])*s1[t]-
(gamma1+c1+d1)*i1[t],i2'[t]==c1*i1[t]+(beta21*i1[t]+beta22*i2[t])*s2[t]-(gamma2+c2+d2)*i2[t], s1[0]==P1-
0.05*P1,s2[0]==P2-0.05*P2,i1[0]==0.05*P1,i2[0]==0.05*P2}, {s1,s2,i1,i2}, {t,0,150}]
Plot[Evaluate[{s1[t]+s2[t],i1[t]+i2[t], 1-(s1[t]+s2[t]+i1[t]+i2[t])}/.%],{t,0,60},
PlotRange→{{0,60},{0,1}}]
```

{{s1→InterpolatingFunction[{{0.,150.}},<>],s2→InterpolatingFunction[{{0.,150.}},<>],i1→InterpolatingFunction[{{0.,150.}},<>],i2→InterpolatingFunction[{{0.,150.}},<>]}}

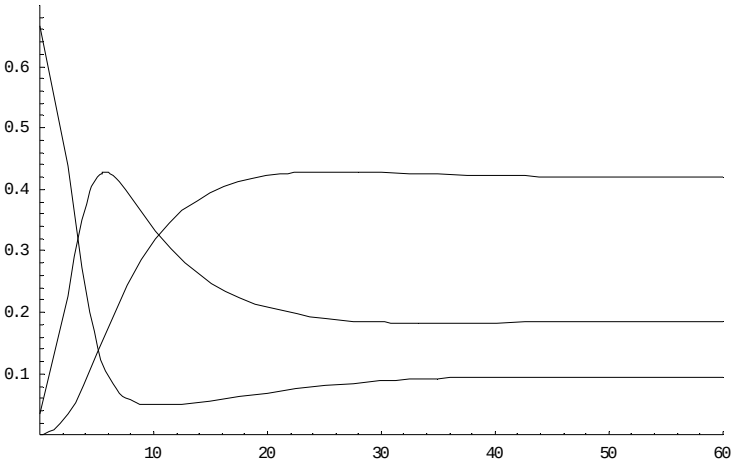


-Graphics-

(*This plots s1[t], i1[t] and r1[t] over time for the age category SIR model*)

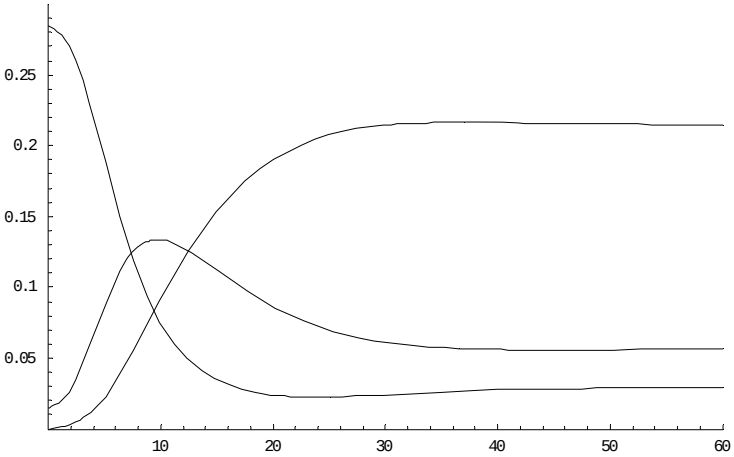
```
beta11=1;beta12=3;beta21=0.1;beta22=0.5; gamma1=1/8;gamma2=1/14; d1=
1/200;d2=1/100;c1=1/20;c2=8/75;P1=.7;P2=.3;
NDSolve[{s1'[t]==(c1+d1)*P1-(beta11*i1[t]+beta12*i2[t])*s1[t]-(c1+d1)*s1[t],s2'[t]==c1*s1[t]-
(beta21*i1[t]+beta22*i2[t])*s2[t]-(c2+d2)*s2[t],i1'[t]==(beta11*i1[t]+beta12*i2[t])*s1[t]-
(gamma1+c1+d1)*i1[t],i2'[t]==c1*i1[t]+(beta21*i1[t]+beta22*i2[t])*s2[t]-(gamma2+c2+d2)*i2[t], s1[0]==P1-
0.05*P1,s2[0]==P2-0.05*P2,i1[0]==0.05*P1,i2[0]==0.05*P2}, {s1,s2,i1,i2}, {t,0,150}]
Plot[Evaluate[{s1[t],i1[t], P1-(s1[t]+i1[t])}/.%],{t,0,60}, PlotRange→{{0,60},{0,P1}}]
```

{{s1→InterpolatingFunction[{{0.,150.}},<>],s2→InterpolatingFunction[{{0.,150.}},<>],i1→InterpolatingFunction[{{0.,150.}},<>],i2→InterpolatingFunction[{{0.,150.}},<>]}}



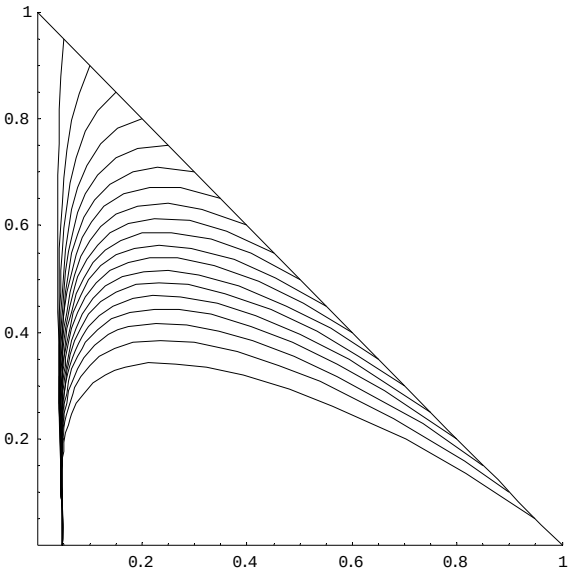
-Graphics-

```
(*This plots s2[t], i2[t] and r2[t] over time for the age category SIR model*)

beta11=1;beta12=3;beta21=0.1;beta22=0.5; gamma1=1/8;gamma2=1/14; d1=
1/200;d2=1/100;c1=1/20;c2=8/75;P1=.7;P2=.3;
NDSolve[{s1'[t]==(c1+d1)*P1-(beta11*i1[t]+beta12*i2[t])*s1[t]-(c1+d1)*s1[t],s2'[t]==c1*s1[t]-
(beta21*i1[t]+beta22*i2[t])*s2[t]-(c2+d2)*s2[t],i1'[t]==(beta11*i1[t]+beta12*i2[t])*s1[t]-
(gamma1+c1+d1)*i1[t],i2'[t]==c1*i1[t]+(beta21*i1[t]+beta22*i2[t])*s2[t]-(gamma2+c2+d2)*i2[t], s1[0]==P1-
0.05*P1,s2[0]==P2-0.05*P2,i1[0]==0.05*P1,i2[0]==0.05*P2}, {s1,s2,i1,i2}, {t,0,150}]
Plot[Evaluate[{s2[t],i2[t], P2-(s2[t]+i2[t])}/.%],{t,0,60}, PlotRange→{{0,60},{0,P2}}]
{{s1→InterpolatingFunction[{{0.,150.}},<>],s2→InterpolatingFunction[{{0.,150.}},<>],i1→InterpolatingFu
nction[{{0.,150.}},<>],i2→InterpolatingFunction[{{0.,150.}},<>]}}
```



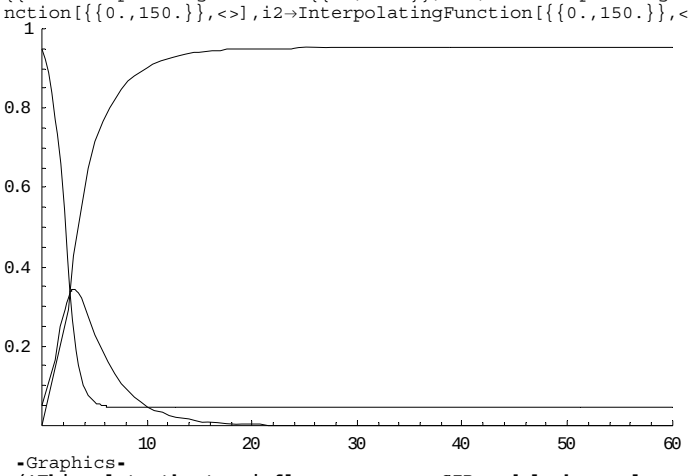
-Graphics-

```
(*This plots the two influence groups SIR model phase plane with immunization: s1[t]+s2[t] vs.
i1[t]+i2[t]*)

beta11=.75;beta12=100;beta21=0.2;beta22=25;alpha11=.5;alpha12=75;alpha21=.1;alpha22=20;
gamma1=1/3;gamma2=1/2; d1= 1/100;d2=1/100;c1=1/20;c2=247/50;P1=.99;P2=.01;
p0=Plot[1-x,{x,0,1},DisplayFunction→Identity];
sols={};
plots={p0};
For[k=1,k<20,
    sols=Append[sols,NDSolve[{s1'[t]==(c1+d1)*P1-(beta11*i1[t]+beta12*i2[t])*s1[t]-(alpha11*(P1-s1[t]-
i1[t])+alpha12*(P2-s2[t]-i2[t]))*s1[t]-(c1+d1)*s1[t],s2'[t]==c1*s1[t]-
(beta21*i1[t]+beta22*i2[t])*s2[t]-(alpha21*(P1-s1[t]-i1[t])+alpha22*(P2-s2[t]-i2[t]))*s2[t]-
(c2+d2)*s2[t],i1'[t]==(beta11*i1[t]+beta12*i2[t])*s1[t]-
(gamma1+c1+d1)*i1[t],i2'[t]==c1*i1[t]+(beta21*i1[t]+beta22*i2[t])*s2[t]-(gamma2+c2+d2)*i2[t], s1[0]==P1-
0.05*P1*k,s2[0]==P2-0.05*P2*k,i1[0]==0.05*P1*k,i2[0]==0.05*P2*k},{s1,s2,i1,i2},{t,0,200}]];
plots=Append[plots,ParametricPlot[Evaluate[{s1[t]+s2[t],i1[t]+i2[t]}/.sols[[k]]],{t,0,200},DisplayFunc
tion→Identity]];
    k++];
Show[plots,AspectRatio→1,PlotRange→{{0,1},{0,1}},DisplayFunction→$DisplayFunction]
Clear[sols]
```



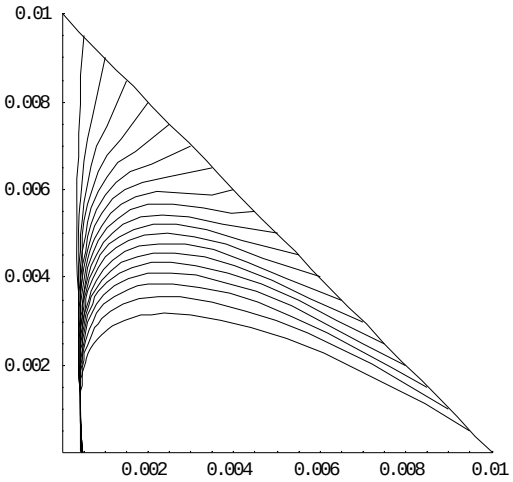
-Graphics-

```
(*This plots s[t], i[t] and r[t] over time for the influence category SIR model with immunization*)

beta11=.75;beta12=100;beta21=0.2;beta22=25;alpha11=.5;alpha12=75;alpha21=.1;alpha22=20;
gamma1=1/3;gamma2=1/2; d1= 1/100;d2=1/100;c1=1/20;c2=247/50;P1=.99;P2=.01;
NDSolve[{s1'[t]==(c1+d1)*P1-(beta11*i1[t]+beta12*i2[t])*s1[t]-(alpha11*(P1-s1[t]-i1[t])+alpha12*(P2-
s2[t]-i2[t]))*s1[t]-(c1+d1)*s1[t],s2'[t]==c1*s1[t]-(beta21*i1[t]+beta22*i2[t])*s2[t]-(alpha21*(P1-
s1[t]-i1[t])+alpha22*(P2-s2[t]-i2[t]))*s2[t]-(c2+d2)*s2[t],i1'[t]==(beta11*i1[t]+beta12*i2[t])*s1[t]-
(gamma1+c1+d1)*i1[t],i2'[t]==c1*i1[t]+(beta21*i1[t]+beta22*i2[t])*s2[t]-(gamma2+c2+d2)*i2[t], s1[0]==P1-
0.05*P1,s2[0]==P2-0.05*P2,i1[0]==0.05*P1,i2[0]==0.05*P2}, {s1,s2,i1,i2}, {t,0,150}]
Plot[Evaluate[{s1[t]+s2[t],i1[t]+i2[t], 1-(s1[t]+s2[t]+i1[t]+i2[t])}/.%],{t,0,60},
PlotRange→{{0,60},{0,1}}]
{{s1→InterpolatingFunction[{{0.,150.}},<>],s2→InterpolatingFunction[{{0.,150.}},<>],i1→InterpolatingFu
nction[{{0.,150.}},<>],i2→InterpolatingFunction[{{0.,150.}},<>]}}
```



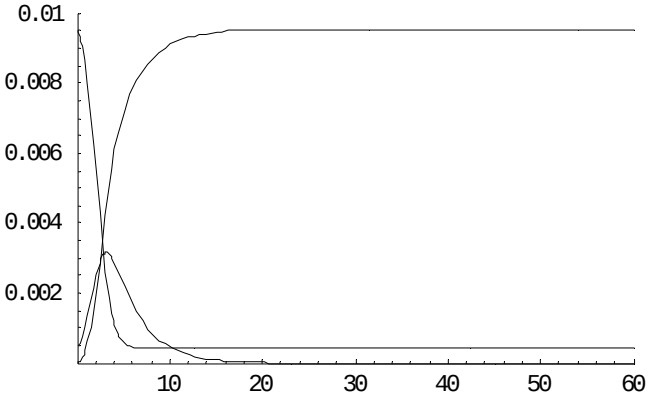
-Graphics-

```
(*This plots the two influence groups SIR model phase plane with immunization: s2[t] vs. i2[t]*)

beta11=.75;beta12=100;beta21=0.2;beta22=25;alpha11=.5;alpha12=75;alpha21=.1;alpha22=20;
gamma1=1/3;gamma2=1/2; d1= 1/100;d2=1/100;c1=1/20;c2=247/50;P1=.99;P2=.01;
p0=Plot[P2-x,{x,0,P2},DisplayFunction→Identity];
sols={};
plots={p0};
For[k=1,k<20,
    sols=Append[sols,NDSolve[{s1'[t]==(c1+d1)*P1-(beta11*i1[t]+beta12*i2[t])*s1[t]-(alpha11*(P1-s1[t]-
i1[t])+alpha12*(P2-s2[t]-i2[t]))*s1[t]-(c1+d1)*s1[t],s2'[t]==c1*s1[t]-
(beta21*i1[t]+beta22*i2[t])*s2[t]-(alpha21*(P1-s1[t]-i1[t])+alpha22*(P2-s2[t]-i2[t]))*s2[t]-
(c2+d2)*s2[t],i1'[t]==(beta11*i1[t]+beta12*i2[t])*s1[t]-
(gamma1+c1+d1)*i1[t],i2'[t]==c1*i1[t]+(beta21*i1[t]+beta22*i2[t])*s2[t]-(gamma2+c2+d2)*i2[t], s1[0]==P1-
0.05*P1*k,s2[0]==P2-0.05*P2*k,i1[0]==0.05*P1*k,i2[0]==0.05*P2*k},{s1,s2,i1,i2},{t,0,200}]];
plots=Append[plots,ParametricPlot[Evaluate[{s2[t],i2[t]}/.sols[[k]]],{t,0,200},DisplayFunction→Identit
y]];
    k++];
Show[plots,AspectRatio→1,PlotRange→{{0,P2},{0,P2}},DisplayFunction→$DisplayFunction]
Clear[sols]
```



-Graphics-

```
 (*This plots s2[t], i2[t] and r2[t] over time for the influence category SIR model with
immunization*)

beta11=.75;beta12=100;beta21=0.2;beta22=25;alpha11=.5;alpha12=75;alpha21=.1;alpha22=20;
gamma1=1/3;gamma2=1/2; d1= 1/100;d2=1/100;c1=1/20;c2=247/50;P1=.99;P2=.01;
NDSolve[{s1'[t]==(c1+d1)*P1-(beta11*i1[t]+beta12*i2[t])*s1[t]-(alpha11*(P1-s1[t]-i1[t])+alpha12*(P2-
s2[t]-i2[t]))*s1[t]-(c1+d1)*s1[t],s2'[t]==c1*s1[t]-(beta21*i1[t]+beta22*i2[t])*s2[t]-(alpha21*(P1-
s1[t]-i1[t])+alpha22*(P2-s2[t]-i2[t]))*s2[t]-(c2+d2)*s2[t],i1'[t]==(beta11*i1[t]+beta12*i2[t])*s1[t]-
(gamma1+c1+d1)*i1[t],i2'[t]==c1*i1[t]+(beta21*i1[t]+beta22*i2[t])*s2[t]-(gamma2+c2+d2)*i2[t], s1[0]==P1-
0.05*P1,s2[0]==P2-0.05*P2,i1[0]==0.05*P1,i2[0]==0.05*P2}, {s1,s2,i1,i2}, {t,0,150}]
Plot[Evaluate[{s2[t],i2[t], P2-(s2[t]+i2[t])}/.%],{t,0,60}, PlotRange→{{0,60},{0,P2}}]
{{s1→InterpolatingFunction[{{0.,150.}},<>],s2→InterpolatingFunction[{{0.,150.}},<>],i1→InterpolatingFu
nction[{{0.,150.}},<>],i2→InterpolatingFunction[{{0.,150.}},<>]}}
```



-Graphics-

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Vita

1st Lieutenant Kjirstin A. Bentson graduated from Gar-Field High School in Woodbridge, Virginia. She entered undergraduate studies at Covenant College in Lookout Mountain, Georgia where she graduated with a Bachelor of Arts degree in Mathematics in May 1997. She went on to work in the civilian world as a mathematics and science teacher in Miami, Florida, a customer service representative in Tucson, Arizona , and a bookkeeper for a law firm in Seattle, Washington. She was commissioned through Air Force Officer Training School in May 2002.

Her first assignment was at Ft. Detrick, Maryland where she worked as a Warfare Analyst with the Air Force Manpower Readiness Flight in May 2002. She was assigned in July 2003 to the Air Force Research Lab, Human Effectiveness Directorate, Logistics Readiness Branch where she served as a project manager. In August 2004, she entered the Graduate School of Engineering and Management, Air Force Institute of Technology. Upon graduation, she will be assigned to the College of Aerospace Doctrine, Research, and Education at Maxwell AFB, Alabama.

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14. ABSTRACT

There are many types of models for counterterrorism, explaining different problems that the military faces in the fight against terrorism. This thesis proposes that one of the fundamental assumptions underlying existing models of counterterrorism is that the struggle with terrorists can be understood as a war in the traditional sense of the term. We propose to rethink the struggle against terrorism as a fight against an infection. The epidemic of terrorist ideology within part of the world is a result, from this perspective, of the infectiousness of that ideology. Using the insights of the field of the epidemiology of ideas, this research looks into the models and methods used to understand and fight biological epidemics. We work with the SIR model from mathematical epidemiology, which partitions populations into susceptible, infected, and recovered categories, and apply that model with notional starting rates to the epidemic of terrorist ideology. This research allows another set of assumptions for models used in counterterrorism because the insights gained from viewing terrorism as a symptom of an epidemic can expand our understanding of the problem that we fight.

15. SUBJECT TERMS

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