Collective cognitive epidemiology: Introducing subjective parameters into disease spread models

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Abstract

Modern instances of disease emergence have shown that human subjective reactions to a novel disease can be as important as the objective reality of the disease spread. Therefore, this work introduces human cognitive heuristics and biases into epidemiological modeling. Human subjective perception and reaction to the presence of disease is represented in the difference between the objective and subjective probability of contagion. It is assumed that humans within a disease spread situation will have either limited or full information about the objective probability of contagion. From this information, humans subjectively react, forming a subjective assessment of the probability of contagion. Although the translation from the objective to the subjective probability of contagion is rooted in a biological basis, the translation has been adequately determined by previous research in Prospect Theory as developed by Daniel Kahneman and Amos Tversky. The formulation of Lotka-Volterra epidemiology models with parameters for perceived probability of contagion was followed by numerical experimentation and sensitivity analyses that determined values of the parameters that create cyclic population behavior, whether growing or dampened, as well as acyclic behavior. The results show that the model is capable of capturing stable as well as unstable behavior, and is able to model key epidemiological disease behaviors and states, such as epidemic and endemic conditions.

Keywords

Epidemiology; cognitive; heuristics; biases; prospect; objective; subjective; probability; contagion; infection; prevention

1.0 Introduction

Although medical technology and population-protecting countermeasures for preventing the contagion and spread of infectious diseases have improved steadily throughout the last century, new infectious diseases are still emerging and spreading swiftly. "Diseases are a ubiquitous part of human life. Many, such as the common cold, have minor symptoms and are purely an annoyance; but others, such as Ebola or AIDS, fill us with dread. It is the unseen and seemingly unpredictable nature of diseases, infecting some individuals while others escape, that has gripped our imagination. From prehistory to the present day, diseases have been a source of fear and superstition" [Keeling, 2001]. Human reactions to diseases are both amplifying and controlling factors in the outcome of disease spread.

Perceptions of diseases, whether informed or popular, can vary widely, influencing the timing and response of personal protective measures such as increased hygiene, procurement of remedies, and protective measures such as social distancing and self-imposed isolation. At the collective, political level, decision making ultimately controls the deployment of vaccines, medical resources and emergency response teams, as well as determining planned funding allocations, school closures and public health system responses.

Bahill [2006] identified severity amplifiers that can significantly augment the perception of danger; for example, lack of control (e.g., of disease spread), lack of choice (e.g., mandated evacuations or vaccinations), lack of trust (e.g., after misinformation), lack of warning (e.g., sudden disease emergence), lack of understanding (e.g., of an unseen disease vector), newness (e.g., SARS in 2002), dreadfulness (e.g., death from infection versus death from coronary heart disease), personalization (e.g., a risk threatening you is worse than the same risk threatening someone else), and immanency (e.g., today's outbreak versus a projected outbreak).

The mention and visualization of disease causes a disproportionately large effect on humans, as illustrated by the following fallacious human decision, in which probability can add up to less than, or the complete, 0-1 probability range. Subjects randomly assigned to two groups were asked about different insurance policies as follows [Johnson, Hershey, Meszaros and Kunreuther, 1993]:

Group A: How attractive do you find an insurance that covers hospitalizations due to any cause whatsoever?

Group B: How attractive do you find an insurance that covers hospitalizations due to diseases or accidents of any sort?

On average, subjects find the insurance mentioned to Group B more attractive than the insurance mentioned to Group A. This is not rational, since the insurance for Group A covers the complete 0-1 probability range, but the insurance for Group B does not. Descriptions of possibilities should map onto corresponding numerical probability ranges. The amplified severity of diseases and accidents extends the objective probability of dying from these causes, to the exclusion of many other real causes of death.

The modeling of infectious disease spread as influenced by human perceptions is now crucial, and includes the need to address various human reactions that may be consequential to predictability. Incorporation of the positive or negative leverage of disease perception during epidemics into mathematical epidemiological modeling will allow for the improved management and control of the spread of infectious diseases with the use of models that capture the complete dynamics of epidemics within human populations.

In fact, the threat of purposefully created viral pathogens that can be engineered to have specific characteristics is growing exponentially with the ready availability and low expense of bioengineering laboratories. The risk now exists that future epidemics will be based on diseases that have been designed to prey on the immunological and psychological debilities of human defenses, including magnified human behavioral reactions to diseases caused by different personal, cultural or societal perceptions of the innumerable characteristics of diseases and spread vectors.

1.1 Individual Cognitive Epidemiology

The link between individual intelligence and disease contagion and health has recently been developed in a new field called cognitive epidemiology. The general hypothesis that individuals with higher intelligence have better health outcomes in life has been demonstrated with a variety of studies, using different measures of intelligence and difference diseases of interest [Arden, Gottfredson and Miller, 2009] [Deary, 20082009] [Deary and Batty, 2007] [Der, Batty and Deary, 2009] [Marmot and Kivimaki, 2009] [Arden, Gottfredson and Miller, 2009] [Rindermann and Meisenberg, 2009]. Batty et al. indicated that higher IQ correlated with a lower risk of coronary heart disease [Batty, Shipley, Mortensen, Gale and Deary, 2008]. The explanations for the connection are numerous, and include:

- A, Individuals with a higher intelligence are more likely to exercise a heightened sense of healthcare, and engage in less drinking and smoking, while exercising more and avoiding health risks,
- B, Individuals with higher intelligence are able to obtain preferred employment, housing, and healthcare and thus avoid high stress environments, or, among other possible explanations,
- C, Individuals who demonstrate higher intelligence have bodies of better health and constitution, and this differential is shown in mortality rates throughout life.

1.2 Collective Cognitive Epidemiology

This article develops a collective cognitive epidemiology with disease spread models, where the connection with disease avoidance and contagion is drawn to the robust empirical characterization of how individuals perceive gains and losses, as well as how objective probabilities are subjectively perceived. An extrapolation is drawn from individuals to groups of individuals, because of the broad commonality of decision making characteristics across similar people. This study has various objectives, including to:

- 1. Develop hybrid objective-subjective models that include observer-dependent effects,
- 2. Demonstrate that individual subjective perception can significantly impact collective disease spread outcomes,
- 3. Facilitate the assessment and characterization of the impacts of the realistic inclusion of subjectivity into numerical epidemiology models, and,
- 4. Spur the generation of countermeasures complementary to traditional disease prevention measures.

Further development of this line of research could allow the effective inclusion of epidemiological factors such as:

A. Perceptual and preventive behavioral differences based on national policies, culture and ethnicity, as well as geographical, organizational or ideological groups,

- B. Perceptual differences based on population density and living habitat,
- C. Differing and evolving perceptions and reactions to diseases during dormancy, mutation and emergence phases, as well as epidemic and pandemic conditions,
- D. Time delay between diagnosis and political decision making and organized campaigns,
- E. Differences in human reactions to various disease vectors.

2.0 Modifying the Lotka-Volterra Equations for Epidemiology

2.1. Lotka-Volterra overview

The topic of interacting competing species has been discussed for many decades. Recently, it was revisited by mathematical societies, which resulted in epidemiology models [Petrovskii and Venturino, 2007] [Fulford, Roberts and Heesterbeek, 2002] [Harvey and al., 2007] [Trottier and Philippe, 2001]. These models have been used to create and implement vaccination policies to fight and possibly eradicate infectious diseases. Currently, there are two predominant associated models, that of Hadeler and Freedman [1989], and the model of Ezio Venturino [1993], related to the subject of interacting species. Also, recent epidemiology models [Brauer and Castillo-Chavez, 2001] account for varying population sizes [Mena-Lorca and Hethcote, 1992].

Our study proposes and investigates a model that addresses the study of the spread of infectious diseases in a fixed virgin population. In this paper, we modify the predator-prey Lotka–Volterra [Holt and Pickering, 1985] equations into an observer-dependent model that allows the analysis of subjective and objective parameters that effect disease spread in Susceptible-Infectious (SI) models. Because of the simplicity of the Lotka-Volterra formulas, they are a good point for introducing cognitive heuristics and biases into disease spread models. We also examine the prediction of the spread of infectious diseases in human society by applying decision making theory. Subjective parameters are interesting in that they allow for the consideration of individual decision making in the presence of the possibility of disease contagion. More importantly, however, the inclusion of subjective parameters allows the modeling of the influence of collective social decision making that effects disease spread, including common collective behaviors that occur under during disease emergence and abatement, as well as during epidemic, pandemic and endemic conditions. Advances in modeling both objective and subjective inputs in disease spread models will significantly improve the understanding of disease reproduction in both epidemics and endemics.

2.2. Lotka-Volterra (LV) equations

The Lotka-Volterra differential equations model predator-prey dynamics in the case of one predator population and one prey population. The model includes several simplifying assumptions:

- 1) The prey population will grow exponentially when the predator is absent;
- 2) The predator population will starve in the absence of the prey population (as opposed to switching to another type of prey);
- 3) predators can consume infinite quantities of prey;

4) There is no environmental complexity (in other words, both populations are moving randomly through a homogeneous environment).

An ecological predator-prey, or parasite-host, model thus exists for a set of four fixed positive constants as follows:

A' is a proportionality constant for the growth rate of prey,

C' is a proportionality constant for the rate at which predators destroy prey,

B' is a proportionality constant for the death rate of predators, and,

D' is a proportionality constant for the rate at which predators increase by consuming prey.

The application of these four assumptions and constants follows:

1. Prey population growth is shown in Equation 1,

$$\frac{dx}{dt} \approx A'x \tag{1}$$

Where, dx/dt shows the prey population growth rate, which is a proportional to number of prey.

The influence of predators is contributed by Equation 2,

$$\frac{dx}{dt} \approx -C'xy \tag{2}$$

Where, the decay rate of the prey population is a proportional to the product of the number of prey and predators.

2. Predator population decay rate is shown below by Equation 3,

$$\frac{dy}{dt} \approx -B'y \tag{3}$$

Where, the predator decay rate is proportional to the number of predators.

The growth rate in the predator population is shown by Equation 4,

$$\frac{dy}{dt} \approx D'xy \tag{4}$$

Where, the predator growth rate dy/dt is proportional to the product of the numbers of prey and predators.

3. Combining the decreasing and increasing components of both the prey population *x*, and the predator population *y*, yields the two Lotka-Volterra differential Equations 5 and 6:

$$\frac{dx}{dt} = A'x - C'xy \tag{5}$$

$$\frac{dy}{dt} = -B' y + D' xy \tag{6}$$

2.3. Modifying LV equations to LV-Epidemiology equations

Assuming that the prey population x becomes the Susceptible population S, and the predator population y becomes the Infectious population I, the LV equations can be modified and made applicable to infectious disease epidemiology.

The LV-Epidemiology constants accordingly become:

A is a proportionality constant for growth rate of the susceptible population,

C is a proportionality constant for the growth rate of the susceptible population by birth,

B is a proportionality constant for the decreasing rate of the infectious population, and

D is a proportionality constant for the rate at which the infectious population increases by stricken susceptible persons, or by birth.

The Lotka-Volterra equations (5) and (6) become modified by considering Infectious (*I*) and Susceptible (*S*) populations, become the LV-Epidemiology Equations 7 and 8.

$$\frac{dS}{dt} = AS - CIS \tag{7}$$

$$\frac{dI}{dt} = -BI + DIS \tag{8}$$

2.4. Lotka-Volterra-Epidemiology numerical study

The Lotka-Volterra equations for epidemiology, Equations 7 and 8, were applied over 20,000 discrete time steps by assuming that A=2, B=2, C=0.2 and D=0.3505, generating evolving susceptible and infectious populations, S and I, respectively. Figure 1 graphically shows the interacting populations S and I.

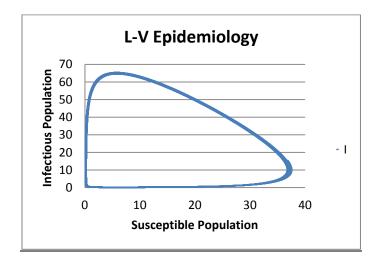


Figure 1: Susceptible and Infectious populations generated from the LV-Epidemiology equations, with populations growing in counter-clockwise rotations

The system evolves counterclockwise, as the Susceptible population S initially increases without a significant number of infections, the Infectious population I is ultimately able to increase markedly on the diminution of the Susceptible population S. However, the limited Susceptible

population S chokes off the growth of infections, bringing about a collapse of both populations. When the Susceptible population S is reduced to extremely low numbers, the rate of infection decreases dramatically, allowing an almost unfettered recovery of the Susceptible population S. The cycle repeats itself rather regularly for a limited range of model coefficients. In the figure shown above, there is slight but clear increase in overall population as the model evolves. If the constants A, B, C and D are subject to chance fluctuations, the process lifecycle may not be stable, and the Susceptible population S can actually go extinct, and as a consequence the Infectious population will go extinct as well.

2.5. Perception of Probability of Contagion in LV-Epidemiology

Upon the observation and awareness of disease presence, it is common knowledge that human will exhibit preventive reflexes and thoughtful behavioral changes meant to thwart the personal contagion of disease. It order to model the epidemiological effects of individual and collective cognitive behavioral changes, a perception term must be added to the LV-Epidemiology equations. A review of the theoretical and empirical knowledge of the relation between objective reality and the subjective assessment of objective reality is now necessary to inform the construction and addition of a perception term into the LV-Epidemiology equations. Edwards [1955] noted that translations between objective values and objective perceptions should be carefully defined. Expected Value, when either the objective "Expected" probability or objective "Value" term become subjective, can become either Subjective-Expected Value, Expected Utility, or Subjective-Expected Utility.

Theories of human behavior can either be Normative, Descriptive, or Prescriptive. Normative models propound views that humans should make decisions according to ideals. For example, rational calculations may be based on the sum of objective probabilities multiplied by corresponding objective values, giving the overall expected value of a gamble. The descriptive model (view) of human decision making seeks only to describe human behavior. The most accepted descriptive theory is Prospect Theory [Kahneman and Tversky, 1979], which explains the nature of the subjective human decision-making process in terms of the heuristics and biases employed in assessing information, and the common deviations from rational decision-making that result [Smith, 2006]. Prescriptive theories seek to ameliorate mistakes.

3.0. Prospect Theory

In 1979, two brilliant psychologists, Daniel Kahneman and Amos Tversky, wrote the second-most cited paper ever to appear in Econometrica, the prestigious academic journal of economics. They presented an analysis of expected utility theory, called von-Neumann Morgenstern utility, as a descriptive model of decision making under risk. In addition, they developed an alternative model, which they called Prospect Theory. Expected utility theory is incapable of describing the biases of people who are simultaneously attracted to both insurance and gambling. Following empirical studies by Kahneman and Tversky, it was found that people under-weigh outcomes that are merely probable, in comparison with outcomes that are obtained with certainty Also, people generally discard components that are shared by all prospects under consideration. In order to explain these seeming aberrations, in prospect theory value is assigned to gains and losses rather than to final assets, and objective probability is converted to a weighted subjective probability before consideration.

3.1. Objective versus Subjective Value

A properly formulated decision theory of human decision making requires a comprehensible model which translates objective value into subjective utility, and vice versa. Fortunately, such a first order model relating objective value and subjective utility has been given in Prospect Theory (PT). Using prospect theory, the process of subjective decision making is divided into a preliminary screening stage, and a secondary evaluation stage. As a result of this break into two stages, values are considered not in an absolute sense (from zero), but subjectively from a reference point established by the subject's wealth perspective before a decision. This process is called *framing*. The key graph that shows how objective values translate into subjective utilities is shown in Figure 2. Remarkable is the significant disparity in magnitude with which gains and losses are subjectively valued: Losses can have a subjective absolute magnitude that is about 2.5 times greater than gains, depending on the human subject.

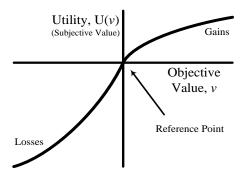


Figure 2: Subjective utility of objective gains and losses in value

Using deviations from a reference point, the value function is defined and is normally concave for gains (implying risk aversion), and normally convex for losses (implying risk seeking), and is generally steeper for losses than for gains (loss aversion). The empirically obtained figure shows that losses have as much as twice the psychologically impact as gains.

People more strongly prefer to avoid losses than to acquire gains. That is, given a loss, they prefer to move from a unit loss back to the status quo, more than they prefer to move from the status quo to a unit gain. Considering a box of cereal with 100 grams of cereal: Would you rather avoid a loss of 5 grams of cereal, or get 5 grams more cereal in the box? Similarly, would you rather avoid paying 5% extra, or save 5%? Most people would rather avoid the loss. The equal changes in amount, framed differently, have significantly different effects on consumer behavior. Traditional economists consider this to be completely irrational, because the implicit assumption of conventional economics is that the only relevant metric is the magnitude of the absolute change in expenditure. The unequal utilities, however, are highly important to the fields of marketing and behavioral finance. In epidemiology, an illustrative example could be posed as follows. Example:

Suppose that an experimental hormone treatment will either raise or lower the objective quality of your life by 5 objective value units, with equal chances. From a personal subjective perspective, it makes no sense to take the treatment while healthy. However, after contracting a disease that lowers the objective quality of life by 5 units, people will engage in risk seeking behavior and choose to take the treatment, because they are trying to recover their recently lost health. The person is falsely using their previous health reference point. However, the utility

curve indicates that they should still refrain from the treatment, since the treatment can still cause a loss of health as readily as a gain in health.

3.2. Objective versus Subjective Probability

Prospect Theory describes the subjective evaluation of probabilities according to the experimentally-obtained graph in Figure 3, which is used to modify the epidemiological equations in this study.

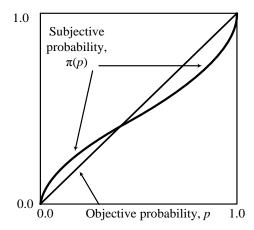


Figure 3: Subjective Probability compared to Objective Probability

Empirically observed overestimation of small probabilities and underestimation of high probabilities can be explained using the experimentally determined PT subjective probability function. Generally, people will overestimate small probabilities, gambling on the unlikely, but possibly important, appearance of rare phenomenon. The subjective probability of dangerous rare events will be considered elevated, and will lead people to try to eliminate such possibilities as part of the Certainty Effect. On the other hand, people will underestimate large probabilities – perhaps because a more commonly occurring phenomenon loses psychological impact.

The Certainty Effect occurs in the range of low probabilities close to 0.0 (and in the range of high probabilities close to 1.0). In this region, prospect theory's subjective probability curve shows that people prefer reducing chances of something bad happening from something to nothing, more than reducing chances of something bad happening by the same amount but not to zero. Note the slope of equal-length consecutive ranges of probabilities close to zero. This phenomenon is called the Certainty Effect. Plous [1993, p. 99] cites economist Richard Zeckhauser: "Zeckhauser observed that most people would pay more to remove the only bullet from a gun in Russian roulette than they would to remove one of four bullets." Notice that the reduction in the probability of being shot is the same in both cases, namely, a 1/6th reduction in probability in both cases. Under the Certainty Effect, considering gains, people are inclined to value a gain that is definite significantly more than a gain that is less than definite; this behavior can hold true even when the expected value of each gain is the same.

Looking a the high end of the subjective probability function, a similar phenomenon occurs for the situation of a loss with either a probability of 1.0, or a probability that is high but less 1.0. Note that a probability that is high but less than 1.0 is severely discounted below a linear discounting. In the range of these probabilities, people will grasp at straws to keep away from a

certain loss, even if it means taking even higher risks with a high probability of loss – because the high probability is subjectively discounted significantly more than the objective probability. Consequently, often people will focus on taking higher risks when they take a certain loss and are trying to regain the loss.

3.3 Prospect Theory versus Expected Value theory

Prospect theory, which uses subjective utility, is inconsistent with expected value theory because of two fundamental reasons. Firstly, expected value theory uses a linear, objective probability, while prospect theory employs a subjectively weighted probability that is not linear. Secondly, expected value theory uses a linear objective value, while prospect theory employs a subjective utility function that is defined differently for gains and losses, where the subjective utility is dependent on deviations from current wealth.

A classic experiment by Richard Thaler [1988] posed three survey questions:

(a) Assume you have been exposed to a disease which if contracted leads to a quick and painless death within a week. The probability that you have the disease is 0.001.

What is the maximum you would be willing to pay for a cure?

Average answer: \$800

(b) Suppose volunteers were needed for research on the above disease.

What is the minimum payment you would require to volunteer for this program?

Average answer: \$10,000

(c) Assume you have been exposed to a disease which if contracted leads to a quick and painless death within a week. The probability that you have the disease is 0.002. The only available cure works in 1 patient out of 2.

What is the maximum you would be willing to pay for this cure?

Average answer: \$100

These result do not make sense under the lens of rational expected value theory, but they do make sense under prospect theory. The Certainty Effect explains these results by ordering the scenarios in term of certainty. Scenario (b) poses option of trading the certainty of not having the disease with the possibility of contracting the disease, Scenario (a) poses a probability of contagion of 0.001, and Scenario (c) poses the probability of contagion of 0.002 with a lowered probability of a cure. Scenario (c) could be said to pose a probability of death of 0.004. The difference in monetary value of the scenarios is remarkable, specifically, \$10,000, \$800, and \$100, in order of decreasing certainty of a good result. Note that the drop off in monetary value is almost complete within an extremely limited probability range of 0.004.

An experiment by Tversky and Kahneman [1981] gathered experimental data from human subjects. They asked people to assume that there was a disease affecting 600 persons, and then gave the experimental subjects two choices:

- Plan A, where 200 of the 600 persons will survive.
- Plan B, where there is a 33% chance that all 600 persons will be survived, and a 66% chance that nobody will survived.

The majority of subjects selected Plan A, which shows a preference for certainty.

In another test with the same assumptions, the human subjects were offered two other choices:

- Plan C, where 400 persons will die.
- Plan D, where there is a 33% chance that nobody will die, and a 66% chance that all 600 people will die.

In this test, most subjects picked Plan D. The results of this test showed risk seeking to avoid the loss of 400 persons. Notice how the framing as either a certain proportion or a risk probability makes the differences. Plan A and C are rationally equivalent according to expected value theory, as are Plan B and D. However, the framing of Plan A and Plan B in terms of gains creates a premium for certainty, while the framing of Plan C and Plan D in terms of losses causes a risk seeking behavior in order to try to avoid losses, along with the human use of a subjectively heightened 33% objective probability, juxtaposed with a subjectively lowered 66% objective probability.

It is apparent that epidemiologically relevant situations are assessment much differently under the empirically descriptive prospect theory, rather than under the normatively rational expected value theory.

4. Perception Effects included in the LV-Epidemiology model

Now, with some understanding of the key differences between objective and subjective decision making, we apply knowledge of subjective human decision-making to the construction of LV-Epidemiology models.

4.1. Loss versus Gain framing

Using the function for the subjective utility of objective gains and losses in value (Figure 2), it is possible to incorporate the subjective assessment of objective value into epidemiology models. For example, suppose that the government maintains and publicizes a "Disease Security Index" (DSI) with a numerical range from 0-10. The original value of the DSI will set the subjective status quo for the population. Suppose that the index first falls by 1, and then rises by 1, staying at the level 9 and level 10. The function for the subjective utility of objective gains and losses indicates that the fall by 1 will be perceived as a loss of approximately twice the utility as the rise by 1 provides. In the short term, this leverage may be useful for stimulating the population to take actions such as attending vaccination tents. Cumulatively, repetitive oscillations down and up by the index, in the medium term, will be perceived as a general degradation in security, with the population becoming increasingly uneasy with the volatility in the index, although a degradation in the objective disease security situation is not true in reality. However, in the long term, the population may become inured to movements in the index [Pusa and Smith, 2010], and may eventually ignore the index, whose repeated calls to alarm do not correlate to the relatively stable objective disease security situation. An example of this conditioned inurement occurred in the Homeland Security Advisory System [Shapiro and Cohen, 2007].

4.2. Objective Probability of Contagion Perception and Prevention Response

Adding an objective probability infection prevention term to the epidemiology modified Lotka-Volterra Equations 7 and 8 results in Equations 9 and 10, where I' and S' represent the infected and susceptible populations under the influence of the objective probability perception term.

$$\frac{dS'}{dt} = AS' - CI'S' + EP_{obj}$$

$$\frac{dI'}{dt} = -BI' + DI'S' - EP_{obj}$$
(10)

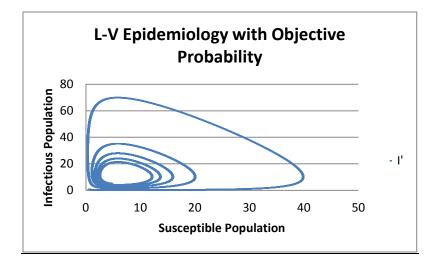
$$\frac{dI'}{dt} = -BI' + DI'S' - EP_{obj} \tag{10}$$

E is a proportionality constant that depends on different prevention conditions, and P_{obj} is the objective probability of contagion perceived by humans and applied toward prevention measures. The EP_{obj} term essentially models the decrease in the rate of infection made possible by the objective perception of disease spread by humans, and by the consequential preventive measures taken by humans.

As an assumption, the P_{obj} parameter is calculated with Equation 11,

$$P_{obj} = \frac{I'}{I' + S'} \tag{11}$$

Application of these modified equations with the initial conditions used previously results in Figure 4, where the population cycles counterclockwise.



Susceptible and Infectious populations interacting according to the epidemiology Lotka-Volterra equations with the addition of the Objective Probability Perception and Prevention term

The loops in Figure 4 look similar to the loops in Figure 1. With E=1.68, the susceptible population S' is generally increasing, indicating that, indeed, the human perception of the objective probability of contagion, and subsequent prevention measures, works to increase the susceptible (non-infected) population.

4.3. Subjective Probability of Contagion Perception and Prevention Response

Considering that human use of subjective probability estimates in assessing objective probabilities leads to the modification of Equation 9 and 10 into Equation 12 and 13,

$$\frac{dS''}{dt} = AS'' - CI''S'' + EP_{sub} \tag{12}$$

$$\frac{dI''}{dt} = -BI'' + DI''S'' - EP_{sub} \tag{13}$$

where E is a proportionality constant depending on different prevention conditions, and P_{sub} is the subjective probability of infection contagion. A double apostrophe notation is used, appearing as S'' and I'', to show that the susceptible and infectious populations are different than under conditions of the objective perception of probability.

 P_{sub} results from the experimentally determined curve in Figure 3 and from the objective probability P_{obj} , and is calculated with Equation 12,

$$P_{sub} = \frac{(P_{obj})^{\delta}}{[(P_{obj})^{\delta} + (1 - P_{obj})^{\delta}]^{(1/\delta)}}$$
(12)

where $\delta \sim 0.68$ under general conditions of subjective probability assessment. Applying this modified equation to the initial conditions used previously results Figure 5.

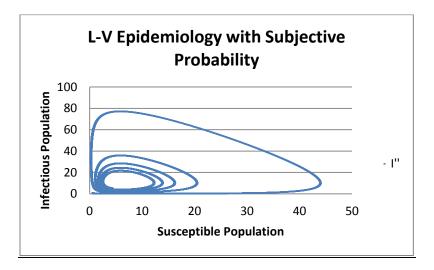


Figure 5: Susceptible and Infectious population interaction according to epidemiology modified Lotka-Volterra equations that include a Subjective Probability Perception and Prevention term

It is apparent that the susceptible population S'' decreases somewhat in comparison with Figure 4, indicating that the use of subjective probability assessment, which actually lowers the perceived sense of danger for situations of high probability of contagion, actually works to increase the overall growth of the susceptible population. This influence predominates – for the employed values of the constants of proportionality – over the counter influence of the significant increase of subjective probability for small objective probabilities.

Figure 6 shows the subjective and objective probabilities as actually employed by the Lotka-Volterra epidemiological equations in the parametric study. As prospect theory describes, overestimation of small probabilities and underestimation of high probabilities occurs.

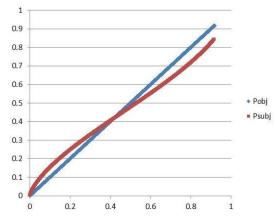


Figure 6- Relation of Objective and Subjective Probabilities

Note that the objective probabilities of contagion from approximately 0.91 - 1.00 did not occur in the simulation.

Analysis of the population interactions shown in Figures 4 and 5 indicates that the averages of total populations produced by the Subjective versus the Objective Probability Perception and Prevention terms are not equal.

This issue can be clarified by more involved data analyses that elucidate the nuances between the models. The models have the potential of showing that actual collective human reactions can be significantly different from reactions predicted by current theoretical modeling.

A numerical investigation of the impact of basic models, and these specialty models, follows.

5. Numeric Parametric study

In order to better understand the behavior of the modified L-V epidemiology equations with the addition of Objective and Subjective probability perceptions and prevention terms, the modified equations were simulated using Euler's method, a well know mathematical computational method.

Euler's method provides an approximation to the solution of differential equations. It uses the concept of linearity to combine small approximations to zero in to an actual solution. The explicit values for A, B, C, D and E were used to explore the behavior and adequacy of the modified equations.

As illustrated in figure 4 and figure 5, the behavior of the modified equations as assumed to suppress the growth of infectious population is not observed. In order to better illustrate this, we use Runge-Kutta method to obtain better approximation of the differential equations. Runge-Kutta method is similar to Euler's method to provide a solution of a differential equation except that Runge-Kutta method employed multiple applications on intermediate time intervals.

5.1. Comparison of No, Objective, and Subjective Perception of Probability of Contagion

Figure 7 illustrates the overlapped trajectories of L-V epidemiology equation, L-V epidemiology equation with addition of the Objective Probability Perception and Prevention term and the equation with the addition of the Subjective Probability Perception and Prevention terms using Runge-Kutta method.

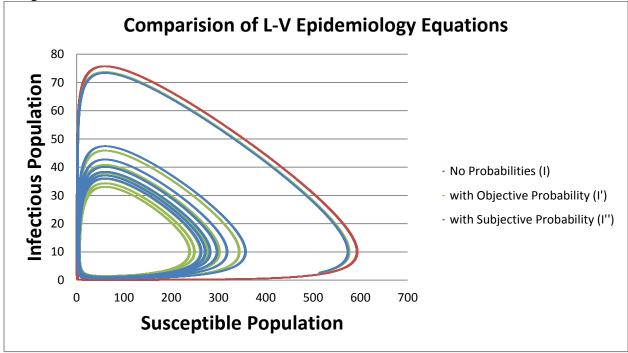


Figure 7: Trajectory comparisons of No, Objective and Subjective Perception of Probability using Runge-Kutta method.

In the graph illustrated, I indicate the trajectory of modified L-V epidemiology equation with the initial values of S=0.43 and I=0.57. I' represents the trajectory of the epidemiology Lotka-Volterra equations with the addition of the Objective Probability Perception and Prevention term with the initial values of S=512.5 and I=2.5, where objective probability helps to increase preventive measures, resulting in an increase of the uninfected population. I" represents the trajectory of the epidemiology Lotka-Volterra equations with the addition of the Subjective Probability Perception and Prevention term with the initial values of S=512.5 and I=2.6, where the subjective probability helps to increase awareness thereby decreasing the infectious population.

5.2. Hyper-Careful and Lazy Sections of Subjective Probability

It is interesting to contemplate the modeling of two distinctly different modes of subjective probability perception, a hyper-careful mode, and a lazy mode. People in the hyper-careful mode are proposed to adopt only subjective probability assessment when it occurs above the objective probability, and will thus only overestimate low objective probabilities, as shown in Figure 8.

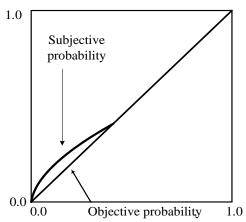


Figure 8: Hyper-careful subjective probability perception

People operating in Lazy mode have their subjective probability perception matched with objective probabilities in low objective probabilities, and they underestimate high objective probabilities, as shown in the Figure 9.

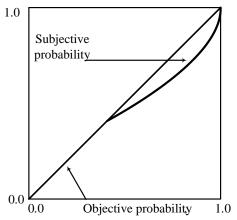


Figure 9: Lazy subjective probability perception

6. Conclusions and Future Work

The Lotka-Volterra predator-prey equations are a good starting point for epidemiology modeling. A crucial addition in epidemiology models is a term that accounts for human perception of the objective probability of contagion and subsequent preventive measures. A further refinement of the probability perception is the translation of objective probability of contagion to an emphatically human subjective probability of contagion, which generally works to heighten prevention measures for small but significant objective probabilities of contagion. The utility of this approach was explored with a numerical study.

Suggested future investigatory directions also include the construction and evaluation of traditional epidemiology equations modified for inclusion of subjective perception. Models available for modification include the SIR, SEIS, SEIR, SEIR, MSIR, MSEIR, and MSEIRS models, for example. This work will involve simulation of disease spread with software capable

of discrete and continuous simulation software, and the fitting of coefficients based on statistical data from different diseases in different time periods.

Future work will include researching and suggesting amelioration of non-useful subjective perceptions and reactions. Future work in this area should prove useful for advances in epidemiology and public health.

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