

Modeling the Psychosocial Effects of Terror or Natural Disasters for Response Preparation

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Protecting human health provides the crux of any response to a catastrophic event, such as a bio-attack or an influenza pandemic. In the event of a disaster, people's anxiety and perceptions of risk influence the amount of response efforts required to mitigate the threat. Their behavior may accumulate and become collective social anxiety. In this study, we use differential dynamics to model the potential psychosocial impacts of a disaster on the affected population. In particular, we model how individuals respond to a disaster event, as well as the population-level collective psychosocial impacts. We then explore how such psychosocial impacts may challenge the effectiveness of response efforts or social productivity. Applying the proposed model to examine the potential psychosocial effects of SARS on health care professionals demonstrates the great effectiveness of the proposed model. The proposed model therefore can be used as a framework for quick assessments of the psychological effects of a terror or natural disaster and provides a guide for response preparation, such as targeting interventions and resource allocations.

Keywords: collective anxiety, disaster response, psychosocial effects, public health, modeling and simulation, social dynamics

1. Introduction

Without a doubt, a disaster—whether terrorism like the 9/11 attack in 2001 or a natural event like Hurricane Katrina in 2005—can cause tremendous damage to both physical entities (e.g., buildings, roads, factories) and humans (e.g., sickness, death). To suggest preparations in advance, many studies examine economic impacts by estimating possible losses and dollar costs to predict the potential effects of a disaster in advance [1, 2, 3, 4, 5]. For example, to help set emergency response plans, the U.S. Department of Homeland Security (DHS) has developed 15 detailed event scenarios relating to possible terrorist attacks, disease outbreaks, and natural disasters [6]. Assessments and predictions surrounding these 15 scenarios focus mainly on estimating potential damages to properties and their related economic costs. Similarly, the National Earthquake Hazard Research Program (NEHRP)—sponsored “Second Assessment of Research on Natural Hazards” concentrates on dollar losses from a wide array of natural hazards, severe weather-related events, and earthquakes. The physical impact of these large-scale traumatic events may last over hours or days or even years, and their mental health effects are large, diverse, and complex.

Researchers in public health study the possible long-term psychological consequences of a disaster using post-disaster analyses. Results from post-disaster analyses, such as those developed on the basis of post-traumatic stress disorder (PTSD) analysis in the past 15–20 years, show that in addition to physical property damages, a disaster event, whether natural or human-made, affects people's mindsets and results in different levels of psychological damage [7, 8, 9,

10]. For example, in a post-disaster analysis of survivors of the Oklahoma City bombing, North and colleagues [11, 12] uncover the profound psychological impacts of a terrorist attack or disaster event. A survey study conducted in the immediate aftermath of the September 11 terrorist attacks revealed that 44 percent of the national sample reported experiencing substantial psychological distress, both in the cities where the attacks occurred and across the country [13].

Disaster-induced negative psychological outcomes may take time to develop, whereas psychosocial effects start immediately when a disaster occurs. A typical phenomenon observed during a disaster is fear and collective anxiety, which represents common responses to imminent threats and actual disaster events [14, 15, 16, 17, 18]. Such collective anxiety also can involve somatic reactions; for example, the 1995 terrorist attack in which Sarin nerve gas was released in the Tokyo subway system produced a wave of mass sicknesses from the resulting anxiety. Psychological effects include not only collective social anxiety during and after the disaster but other psychopathologies as well, particularly PTSD and anxiety and depressive symptoms, such as decreased self-efficacy, impaired work performance, startle responses, and feelings of helplessness or sadness [19, 20].

Historical data from post-disaster analyses also show that disaster-induced negative outcomes influence the effectiveness of response efforts, such as disaster control procedures, rescue processes, and participation by health care workers and others. When a severe outbreak of a disease occurs, many measures get applied, some of which appear to be extreme and dictated by panic. The outbreak of severe acute respiratory syndrome (SARS) in 2003, for example, led to psychological distress for health care workers and the general public because of stigmatization of groups perceived to be at high risk and general fears about safety and health [21, 22, 23, 24]. In this sense, a disaster event's physical damages and associated acts resulting from mass anxiety represent a terrible reality with the potential to cause great public health and economic burdens. Yet no models of social response to catastrophic events evaluate the potential effects on human productivity [1, 25].

The affected populations, including both direct victims and those secondarily involved (e.g., first responders, care givers, care providers, educators), number in the millions. For these reasons, a recent report from the U.S. National Research Council of the National Academies [8] suggests that in addition to forecasting possible economic impacts before a disaster and examining the long-term effects after a disaster, there is an urgent need for real-time modeling that estimates the potential psychosocial impacts of a disaster during the disaster while also examining and recognizing how they affect response efforts. In this article, we target this research gap by building mathematical equations using differential dynamics to analyze the psychosocial effects of a disaster. This approach enables us to measure the individual-level psychosocial consequences of a disaster event and project the range and severity of the possible psychosocial consequences during the course of the disaster; in turn, we can (1) estimate the population-level social productivity and response efforts and (2) recommend appropriate and efficient response preparations in advance.

Because human behavior is not only determined by a person's own internal decisions but also influenced by others during a disaster, people affected to various degrees tend to intertwine over time. We therefore first examine how people perceive risk and quantify their behavioral reactions when facing a threat. We then model macro-level collective anxiety in the targeted population to analyze its effects on levels of social productivity. Psychosocial effects reduce social productivity and compromise response efforts. Moreover, these impacts can cause social disruption if no intervention occurs during the course of the disaster. By applying the proposed

model to explore the potential psychosocial effects of an infectious disease like SARS, we find that the model's prediction is highly consistent with the observed empirical data, in support of the effectiveness of the proposed model.

Although extensive research estimates economic impacts and long-term psychological effects (i.e., PSDT) of a disaster, to the best of our knowledge, no studies apply mathematical equations to study psychosocial effects or their impact on the effectiveness of the public response in terms of social productivity [25]. Fewer still published reports on the psychological impact of disease outbreaks adopt a survey approach or present summarized results at an aggregate level. Because individuals likely have different emotional and behavior reactions to the same threat, survey results suggest only the level of importance and cannot account for how individual-level psychosocial damage develops, nor understand its underlying dynamic processes. Therefore, the modeling effort proposed herein offers a first step in developing mathematical models of the impacts of a threat on people's minds during the disaster, which in turn may cause widespread anxiety or distress throughout society. The completed modeling at both individual- and population-levels, as well as the shift from a statistical to a dynamic mathematical model to evaluate psychosocial concerns, enable us to understand how people dynamically respond to a disaster event; suggest more effective responses, including resource allocations and intervention targets; and help reduce the occurrence of long-term psychological damages.

Furthermore, this study suggests ways to understand psychosocial effects during rather than after the disaster. In particular, the dynamic mathematical model can provide rapid assessments of early psychological and behavioral reactions to the loss of social and economic resources.

The remainder of this article is organized as follows: In Section 2, we provide a brief review of the literature on impacts of a disaster event. Section 3 describes the proposed model formulation and solution development. We offer an analytic analysis of the model in Section 4 and discuss its applicability for assessing potential psychosocial consequences of SARS in Section 5. We conclude with some discussions of possible managerial applications in Section 6.

2. Brief Literature Review

Studies examining the impacts of a potential disaster may be categorized into two tracks. One focuses on economic analyses and attempts to estimate or predict the potential property losses and dollar costs of a disaster in advance [1, 2, 3, 4, 5] by applying complex statistical methodology with predefined probability distributions of the key input variables to forecast the possible death toll, property damage, and dollar costs [8]. The other track examines the possible long-term psychological consequences of a disaster, specifically PTSD, by conducting post-disaster follow-up case analyses. That is, after a disaster, researchers survey the affected population to examine their post-impact reactions, then keep tracking them for several years after the disaster in an attempt to understand the factors related to increased or decreased risk for PTSD, which ideally helps affected people recover from their disaster distress [9, 10, 16, 17, 18, 26, 27, 28]. Researchers in the second track show that a disaster can impair people's physical bodies and minds, that people exposed to the disaster often display different reaction and symptom levels during or after the disaster, and that some people will be more affected than others, depending on the nature of the event and the characteristics of the persons who have experienced or witnessed it [26, 29]. Whereas some people experience significant subjective discomfort, others display conspicuous impairment in their day-to-day functioning, such as

sleeplessness, and still others indicate clear impairment in one or more functional aspects, such as work productivity or the ability to engage in and enjoy leisure activities.

Possible psychological impacts usually initiate immediately rather than long after the actual disaster, as during the 2004 water scare, which reflects how anxiety and fear about an event affects the public's response. In early 2004, reports indicated that drinking water in Washington D.C. contained high concentrations of lead, so the District of Columbia's Water and Sewer Authority recommended that residents let water run for 1–2 minutes prior to using it for drinking or cooking. However, many residents let their water run for approximately 10 minutes, which represents an inappropriate response [30]. In the months following the 9/11 terrorist attacks, anthrax cases caused not only widespread fears about bioterrorism but also collective anxiety nationwide. Similarly, between October 2001 and June 2002, a mysterious skin rash affecting thousands of students in mainly primary schools was reported in widely separated locations in 27 U.S. states and parts of Canada. In the wake of extensive bioterrorism fears, students paid excessive attention to their skin after hearing media reports of coetaneous anthrax. The Centers for Disease Control and Prevention (CDC) concluded that with 53 million young people attending 117,000 schools each school day in the United States, rashes from wide range of causes simply will exist [31, 32].

Such disaster-/threat-induced reactions may be classified into four categories on the basis of human mental functions [26, p. 28]: emotional (e.g., feelings of shock and helplessness, loss of pleasure, sadness), cognitive (e.g., impaired concentration and decision-making ability, disbelief, decreased self-efficacy), physical (e.g., fatigue, exhaustion, startled response, reduced immune response, insomnia, vulnerability to illness), and interpersonal (e.g., social withdrawal, impaired work or school performance, distrust, externalization of vulnerability) effects. If they occur, these effects weaken people's response capabilities during the course of a disaster.

Nickell and colleagues [23], in their survey to investigate the psychological impacts of SARS on health care workers involved with SARS-affected patients/hospitals during and after the 2003 outbreak of SARS, find that of 1,988 health care workers, 40.7 percent of them showed some degree of concern about personal health (i.e., fear of contagion) during the outbreak, 24 percent were extremely concerned about their personal health, and another 29 percent indicated the presence of emotional distress. Before the virus was identified, SARS was an infection of unknown cause, unknown mode of transmission, global spread, and high mortality [33]. Such characteristics increase a person's perceived risk and heighten the level of fear and anxiety associated with being contaminated; even the psychological well-being of medical professionals could not avoid such impacts because such a disaster can threaten their lives as well [34]. These studies demonstrate that at the time of a disaster event, many people experience fear and anxiety.

Although little is known about how an emerging threat would affect people's minds and reactions, much existing literature provides a conceptual discussion of the psychological consequences of disasters or terrorism with a heavy emphasis on describing symptoms of PTSD in various victim populations [25]. Along with mathematical modeling, most existing models focus on the biology of an infectious disease [35, 36]. Unfortunately however, little research adopts mathematical modeling to evaluate the psychosocial effects of a disaster event and how such psychosocial impacts may challenge the effectiveness of response efforts or social productivity. Such mathematical modeling could advance our knowledge of individual-level emotional and behavioral responses, as well as the social dynamics of collective behavior. The novelty of our work thus lies in our mathematical approach to dynamically modeling psychosocial effects at both individual and population levels. Moreover, our proposed model can

provide real-time assessments of the physical and mental consequences of a disaster and suggest steps that enable decision makers to establish effective response preparations in advance.

3. The Model

A disaster might be a severe weather-related event, earthquake, severe disease outbreak, or large-scale attack on civilian populations. If a human-made or natural disaster occurs in a targeted area, it can be treated as external force acting on people in that area. Analogous to an information wave field or magnetic field, a disaster field forms, such that people in that area may be affected to various degrees (see Figure 1).

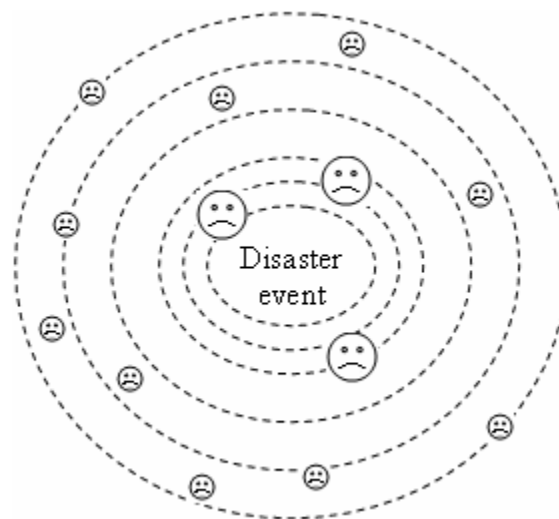


Figure 1. A Disaster Field

When a disaster occurs, people in the affected area incur different levels of influence, which may result in different levels of psychosocial damage: low, moderate, and high.

For modeling purpose, some simplifying assumptions are necessary. First, we assume that people are randomly affected by the disaster event, either as a witness or direct victim, and that each affected person has a different psychological reaction to the disaster event due to his or her personal experiences, perceived risk about the disaster event, and self-immune systems. Second, we assume people are risk-neutral with rational conduct. Looking back on the history, human survival depends on the ability to mount a successful response to threat. When a disaster occurs, the amygdala in humans' midbrains receives input on a conscious level from the cortex and sets off a chain of events that result in rapid release of oxytocin, vasopressin, and a corticotropin-releasing hormone, which in turn activates the sympathetic nervous system [37, 38]. In response, the adrenal glands produce adrenaline and, through a different pathway, cortisol, both of which prepare the body for fight or flight. Thus, without weighing pros and cons, the amygdala can make a quick decision to prompt immediate actions, such as fight or flight, when the person faces a threat. Meanwhile, the hippocampus, a seahorse-shaped area in the midbrain, receives information from the cortex and combines the encountered event, such as the source or context surrounding the encounter, with separate features of past experiences into an integrated memory picture in the brain to help the person make rational decisions. In a typical brain, the hippocampus works in conjunction with the amygdala to connect memory with emotion and

make rational decisions. Therefore, a person's reaction to a threat is influenced by his or her experiences in memory.

Historical data also suggest that people may be influenced by neighbors, community members, schoolmates, or social connections. Thus, fear and subjective risk perceptions get transmitted from one person to another through social communication networks. In addition, media reports may prompt broad transmissions. For example, the famous radio show "The War of The Worlds," broadcast in 1938, led radio listeners to panic and rush out of their homes to escape the so-called "Gas Raid from Mars" [39]. The psychological effects of a disaster clearly have social aspects and are transitive among different people. We therefore use the term "psychosocial effects" to describe the psychological effects of a disaster and its associated social characteristics.

Next we model the individual-level anxiety that a person may incur when exposed to an unexpected disaster. We then quantify the collective anxiety of the targeted population.

3.1 Modeling individual-level psychological damage

When a person perceives a threat, his or her brain, particularly the cortex, the amygdala, and the hippocampus, biologically and cognitively interpret that threat (see Figure 2). This information also gets processed in the limbic system, which manages emotional behavioral responses, such as anger, anxiety, sadness, or fear. Previous post-disaster analyses suggest that affected people may display decreased self-efficacy, reduced working hours, startled responses, or reduced work performance, including burnout and emotional exhaustion. Suppose, for example, a disaster occurs at time π , and people can generate normal productivity before π . After time π , if affected, people's performance on a task may not exhibit the same productivity. Let θ_i be the average frequency of performance error or startled response for person i per unit of time. The possible impact of a disaster on person i 's behavior (i.e., Impact_i) can be evaluated by examining how often affected person i shows maladaptive coping and reduced work performance. That is,

$$\text{Impact}_i = 1 - e^{-\int_{\pi}^t \theta_i(t) dt} \quad (1)$$

Now we need to find θ_i , which reflects an individual-specific measure corresponding to a specific disaster event.

As we mentioned previously, people have different psychological reactions to the same disaster event due to their personal experiences, perceptions of risk about the disaster event, and self-immune systems.

Let R_{ij} denote the reaction of person i when exposed to disaster j , where i refers to the i^{th} person exposed to the disaster. Also let pr_{ij} represent person i 's perceived risk about disaster j . In turn, $D_{ij}(t)$ denotes the information set that person i possesses at time t when determining the perceived risk pr_{ij} and the subsequent reaction. Then, person i 's expected reaction when facing disaster j is

$$E(R_{ij} | D_{ij}(t)) = F_{ij}(D_{ij}(t), PEXP_i)\alpha + \beta E(pr_{ij} | D_{ij}(t)) + \varepsilon_{ij}, \quad (2)$$

where $F_{ij}(D_{ij}(t), PEXP_i)$ contains information about the features of disaster j and the past experience and personal characteristics of person i (i.e., $PEXP_i$), ε_{ij} denotes the idiosyncratic

shock encountered by person i when evaluating perceived risk about disaster j , and α and β are constants.

The features of a disaster also affect people's behavior or reactions. For example, the characteristics of a bio-terror attack differ from those of a severe weather event such as a tornado. In the former, people in all areas, including those not in the affected site, may worry whether the bio-agent in the attack can spread through air; a tornado event only directly affects those who live in the touchdown area, and people living elsewhere may not sense fear about it.

If there is no disaster and people behave normally, person i exhibits normal behavior or action R_{i0} :

$$R_{i0} = PEXP_i \eta + \varepsilon_{i0}, \quad (3)$$

where η is a constant, ε_{i0} denotes an unobservable idiosyncrasy person i has.

Therefore, the difference in person i 's behavior or reaction due to disaster j can be captured on average by

$$\theta_{ij}(t) = F_{ij}[D_{ij}(t), PEXP_i] \alpha + \beta E(pr_{ij} | D_{ij}(t)) + \varepsilon_{ij} - PEXP_i \eta - \varepsilon_{i0}. \quad (4)$$

If we substitute Equation (4) into Equation (1), we can quantify the individual-level psychological damage that a person may incur at time t when exposed to an unexpected disaster, as follows.

$$\text{Impact}_i = 1 - e^{-\int_0^t \{F_{ij}[D_{ij}(t), PEXP_i] \alpha + \beta E(pr_{ij} | D_{ij}(t)) - PEXP_i \eta + \varepsilon_{ij} - \varepsilon_{i0}\} dt} \quad (4')$$

If this impact continues, the psychological damage may alter the neurons in a person's hippocampus, resulting in short-term memory impairment [40]. If this impairment occurs without any interruption, the amygdala may not work with the hippocampus properly, and a disconnection from the real world can occur, which prevents the association of flashback memories with real events. In this case, PTSD results.

Because some people are more affected by a disaster event than others, depending on the nature of the event and individual characteristics, and because studies show no single pattern of psychological consequences of disasters exists [41], many post-disaster analyses adopt a measure to quantify negative psychological impacts into three levels on the basis of their severity: low, moderate, and high [8, 10]. Following a similar measurement scale, we also use low, moderate, and high to indicate the level of psychological damages a person experiences at time t . According to this classification, the "high" level indicates that a person may incur a severe impairment that results in long-term distress.

Figure 2, a graphic illustration of Equation (4'), displays how such individual-level psychological damage can be produced.

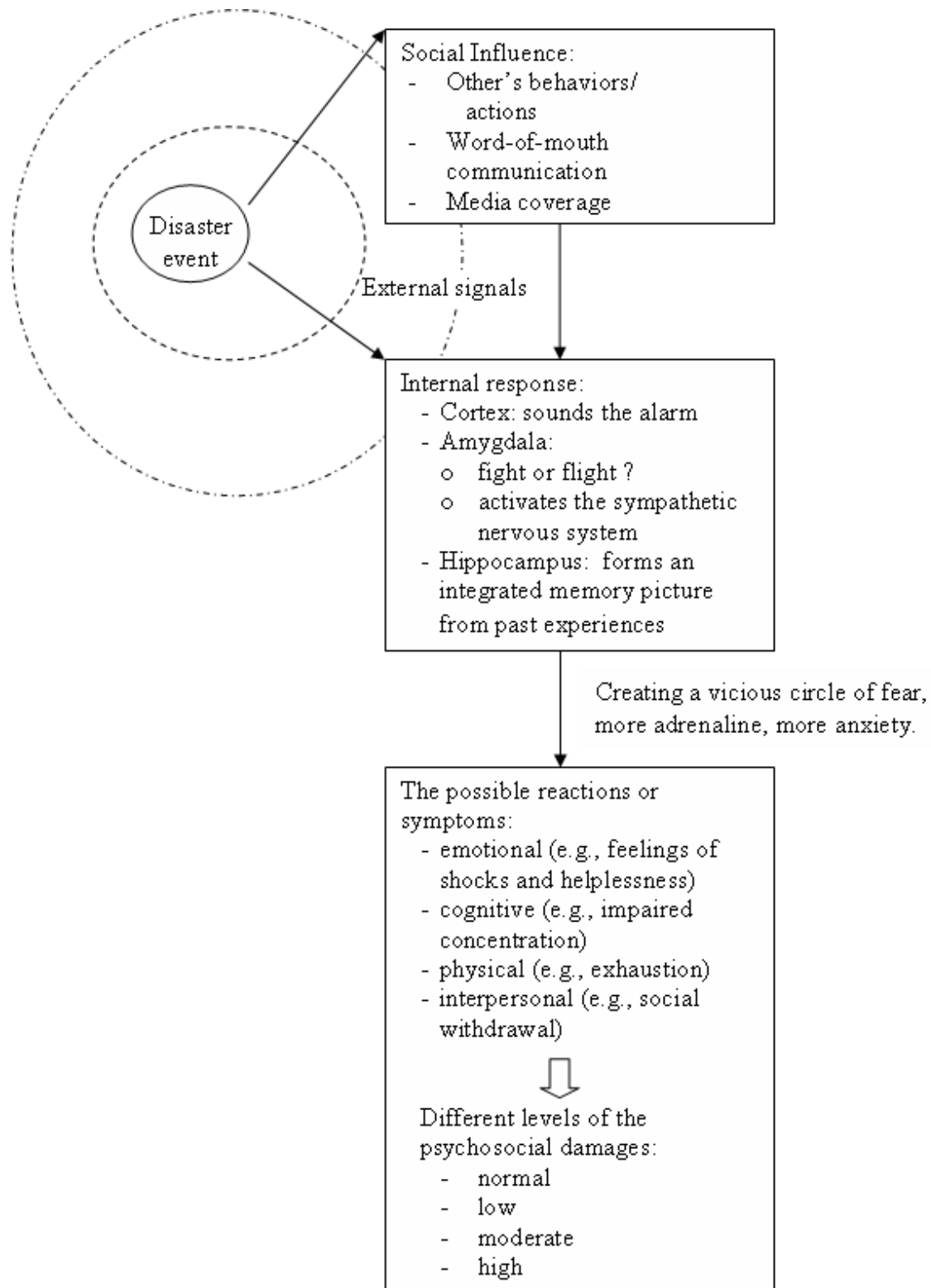


Figure 2. Individual Mental-level Risk Perception and Reaction

3.2 Macro-level collective anxiety when facing a disaster event

Let C be the total population size in the targeted area (i.e., country, region, or state). At any time t during the course of the disaster, a person in C may be affected or not. If affected, the psychological damages the person experiences coincide with one of three levels: low, moderate, or high at time t . Therefore, the aggregate behavior of the population at each point in time t can be summarized by a “state” labeled $L(t)$, $M(t)$, $H(t)$, or $N(t)$, where $N(t)$ denotes a state in which people have not been affected or have recovered by time t . That is, at any time t of the disaster, any member of the population C will reside in one of four states: $N(t)$, $L(t)$, $M(t)$, or $H(t)$, depending on his or her degree of exposure (i.e., the value of Impact_i in Equation (4')).

Research indicates that people with prior psychological distress histories (e.g., disaster, threat) suffer a much greater risk of becoming retraumatized by an experience. Thus, the impact of disaster-/threat-induced psychological damage is cumulative (i.e., the more often a person experiences a disaster event, the greater the impact); additive, in that exposure to different types of impairments correlates with greater impact; and summative, because the combination of events and their impacts are what people carry forward through time, inscribed in their memory, their sense of self, and their behavior [42]. The saying, “Once bitten by a snake, one shies at a coiled rope for the next ten years,” reflects such psychological impacts.

Lonigan and colleagues [43] find that greater anxiety leads to more PTSD symptomatology in children who have experienced more severe exposure to a disaster event. Therefore, as time passes, people’s status in the population may change from one state to another, on the basis of whether (1) the situation becomes more severe, such that collective social anxiety behavior deteriorates over time if ignored, or (2) the individual human body can make self-adjustments to recover through resilience. Since the duration of a disaster is short compared to the life of an individual, the size of the target population is assumed to be constant and the death toll due to the disaster itself is counted in the severe state H . Because the social influence (i.e. a person in one state may be influenced by another person in the same or other state) has been captured at the individual level in equation (4'), in the aggregate level model, we assume the rate of change per unit time from one state such as $N(t)$ to another state (e.g. $L(t)$) is proportional to the number of people in state $N(t)$ at time t times the state transition rate. For example, to model the quantity $N(t + \Delta t)$, we check the number of people in state N at time t plus those converted to state $N(t)$ over Δt , but minus the number of people who are affected and transferred to another state such as $L(t)$, $M(t)$ or $H(t)$ during the same time interval Δt . Thus

$$N(t + \Delta t) = N(t) + [\beta_\phi L(t) - (\gamma_\phi + \gamma_\psi + \gamma_\xi) N(t)] \Delta t$$

If we allow the time interval becomes very small, then in the limit the above equation becomes

$$\frac{dN(t)}{dt} = \beta_\phi L(t) - (\gamma_\phi + \gamma_\psi + \gamma_\xi) N(t)$$

For practical purposes, we assume that the impact of the disaster takes place immediately and continuously when the disaster event occurs. Thus, the affected population can be modeled as a four-dimensional dynamic system, as shown in Figure 3, in which the four states intertwine over time. The arrows in the graph indicate the changing directions of transitions in state.

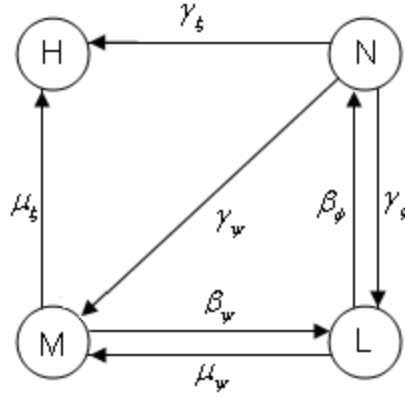


Figure 3. Changing Status over Time

As time passes, people's status in the population may change from one state to another, on the basis of whether (1) the situation becomes more severe such that collective anxiety behavior deteriorates over time if ignored, or (2) the individual human body can make self-adjustments to recover through resilience.

This leads to the following differential equation system:

$$\begin{aligned}
 \frac{dL(t)}{dt} &= -(\beta_\phi + \mu_\psi)L(t) + \beta_\psi M(t) + \gamma_\phi N(t), \\
 \frac{dM(t)}{dt} &= \mu_\psi L(t) - (\beta_\psi + \mu_\xi)M(t) + \gamma_\psi N(t), \\
 \frac{dH(t)}{dt} &= \mu_\xi M(t) + \gamma_\xi N(t), \quad \text{and} \\
 \frac{dN(t)}{dt} &= \beta_\phi L(t) - (\gamma_\phi + \gamma_\psi + \gamma_\xi)N(t)
 \end{aligned} \tag{5}$$

where the initial conditions are $N(0) = C$, $L(0) = 0$, $M(0) = 0$, and $H(0) = 0$ (i.e., $t = 0$); and the equation $N(t) + M(t) + L(t) + H(t) = C$ holds at any time t . The unit for t is any convenient unit of time such as a second, an hour, day, week, and so on. The constants β_ϕ and β_ψ represent the average recovery rates as persons move from low to normal states or recover from moderate to low states, respectively. Similarly, μ_ξ and μ_ψ denote the average deterioration rates as people move from moderate to severe or low to moderate states, respectively. Furthermore, γ_ϕ , γ_ψ , and γ_ξ are the transition rates by which people move from normal to low, moderate, or high states, respectively. Note that γ_ξ denotes the transition rate at which people immediately enter a severe state and incur long-term stress when exposed to the disaster. In line with [8] and [26], $H(t)$ represents the average number of people who suffer extreme fear and may have developed more severe levels of distress psychologically at time t , whereas $L(t)$ refers to the number of people who show mild distress or anxiety behavior but not clinical somatic symptoms. The moderate level $M(t)$ denotes that the person expresses anxiety behaviors, together with obvious somatic symptoms (i.e., clinically significant morbidity as opposed to mild symptoms in level $L(t)$).

We solve System (5) to obtain quantitative information about the changing behavior of the system and the social effects of psychological damages on the population. Applying the Laplace

transformation, we arrive at the following explicit analytical solutions (for a detailed derivation, see Appendix A):

$$\begin{aligned}
N(t) &= \frac{u_1 k^2 - v_1 k + w_1}{(g-k)(s-k)} e^{-kt} + \frac{u_1 s^2 - v_1 s + w_1}{(k-s)(g-s)} e^{-st} + \frac{u_1 g^2 - v_1 g + w_1}{(s-g)(k-g)} e^{-gt}, \\
M(t) &= \frac{u_2 k^2 - v_2 k + w_2}{(g-k)(s-k)} e^{-kt} + \frac{u_2 s^2 - v_2 s + w_2}{(k-s)(g-s)} e^{-st} + \frac{u_2 g^2 - v_2 g + w_2}{(s-g)(k-g)} e^{-gt}, \\
L(t) &= \frac{u_3 k^2 - v_3 k + w_3}{(g-k)(s-k)} e^{-kt} + \frac{u_3 s^2 - v_3 s + w_3}{(k-s)(g-s)} e^{-st} + \frac{u_3 g^2 - v_3 g + w_3}{(s-g)(k-g)} e^{-gt}, \text{ and } (6) \\
H(t) &= \frac{-Qk^3 + u_4 k^2 - v_4 k + w_4}{-k(g-k)(s-k)} e^{-kt} + \frac{-Qs^3 + u_4 s^2 - v_4 s + w_4}{-s(k-s)(g-s)} e^{-st} \\
&\quad + \frac{-Qg^3 + u_4 g^2 - v_4 g + w_4}{-g(s-g)(k-g)} e^{-gt} + \frac{w_4}{skg},
\end{aligned}$$

where:

$$\begin{aligned}
u_1 &= N(0), \\
v_1 &= \beta_\phi L(0) + (\beta_\phi + \beta_\psi + \mu_\psi + \mu_\xi) N(0), \\
w_1 &= (\beta_\phi \beta_\psi + \beta_\phi \mu_\xi) L(0) + \beta_\phi \beta_\psi M(0) + (\beta_\phi \beta_\psi + \beta_\phi \mu_\xi + \mu_\xi \mu_\psi) N(0), \\
u_0 &= \gamma_\phi + \gamma_\psi + \gamma_\xi + \beta_\phi + \beta_\psi + \mu_\psi + \mu_\xi, \\
v_0 &= \mu_\xi (\gamma_\phi + \gamma_\psi + \gamma_\xi + \beta_\phi + \mu_\psi) + \beta_\phi (\gamma_\xi + \beta_\psi + \gamma_\psi) + (\gamma_\phi + \gamma_\psi + \gamma_\xi) (\beta_\psi + \mu_\psi), \text{ and} \\
w_0 &= \mu_\xi \mu_\psi (\gamma_\phi + \gamma_\psi + \gamma_\xi) + \beta_\phi (\beta_\psi \gamma_\xi + \mu_\xi \gamma_\xi + \gamma_\psi \mu_\xi).
\end{aligned}$$

We obtain k from the following equation:

$$-k^3 + u_0 k^2 - v_0 k + w_0 = 0 \quad (7)$$

s can be solved from

$$s^2 - (u_0 - k)s + v_0 - k(u_0 - k) = 0, \quad (8)$$

$$g = u_0 - k - s, \quad (9)$$

here, k , s , and g are eigenvalues of the system (5). In addition, we have

$$\begin{aligned}
u_2 &= M(0), \\
v_2 &= \gamma_\psi N(0) + (\beta_\phi + \mu_\psi + \gamma_\phi + \gamma_\psi + \gamma_\xi) M(0) + \mu_\psi L(0), \\
w_2 &= (\beta_\phi \gamma_\psi + \gamma_\phi \mu_\psi + \mu_\psi \gamma_\psi + \mu_\psi \gamma_\xi) L(0) + (\gamma_\phi \mu_\psi + \beta_\phi \gamma_\psi + \gamma_\psi \mu_\psi) N(0) \\
&\quad + (\gamma_\phi \mu_\psi + \gamma_\psi \beta_\phi + \beta_\phi \gamma_\xi + \gamma_\xi \mu_\psi + \mu_\psi \gamma_\psi) M(0), \\
u_3 &= L(0), \\
v_3 &= \gamma_\phi N(0) + \beta_\psi M(0) + (\beta_\psi + \mu_\xi + \gamma_\phi + \gamma_\psi + \gamma_\xi) L(0), \\
w_3 &= (\gamma_\psi + \gamma_\phi + \gamma_\xi) [(\beta_\psi + \mu_\xi) L(0) + \beta_\psi M(0)] + (\gamma_\phi \beta_\psi + \mu_\xi \gamma_\phi + \beta_\psi \gamma_\psi) N(0), \\
Q &= H(0), \\
u_4 &= \gamma_\xi N(0) + \mu_\xi M(0) + (\beta_\psi + \beta_\phi + \mu_\xi + \mu_\psi + \gamma_\phi + \gamma_\psi + \gamma_\xi) H(0),
\end{aligned}$$

$$\begin{aligned}
v_4 &= \mu_\xi(\gamma_\psi + \gamma_\xi)[C - L(0)] + \gamma_\xi\beta_\phi[C - M(0)] + \mu_\xi\mu_\psi[C - N(0)] \\
&\quad + \gamma_\xi(\beta_\psi + \mu_\psi)[N(0) + H(0)] + \mu_\xi(\beta_\phi + \gamma_\phi)[M(0) + H(0)] \\
&\quad + [\beta_\phi(\gamma_\psi + \beta_\psi) + (\gamma_\phi + \gamma_\psi)(\beta_\psi + \mu_\psi)]H(0), \text{ and} \\
w_4 &= C[\beta_\phi(\gamma_\psi\mu_\xi + \gamma_\xi\mu_\xi + \gamma_\xi\beta_\psi) + \mu_\psi\mu_\xi(\gamma_\phi + \gamma_\psi + \gamma_\xi)].
\end{aligned}$$

Equation (6) offers a fundamental set of solutions to System (5) that measures the average social effects of psychological damages on the population as a result of a disaster.

4. Analysis of the Model

4.1 A possible long-term effect

From Equation (6), we find that when $t \rightarrow \infty$, $e^{-kt} \rightarrow 0$, $e^{-st} \rightarrow 0$, and $e^{-gt} \rightarrow 0$. Therefore, the following happens when $t \rightarrow \infty$:

$$N(t)|_{t \rightarrow \infty} \Rightarrow 0, \quad L(t)|_{t \rightarrow \infty} \Rightarrow 0, \quad M(t)|_{t \rightarrow \infty} \Rightarrow 0, \quad \text{and} \quad H(t)|_{t \rightarrow \infty} \Rightarrow \frac{w_4}{kgs}.$$

From Equation (9), we know that $g = u_0 - k - s$. Therefore, we have

$$kgs = ks(u_0 - k - s) = k[(u_0 - k)s - s^2]. \quad (10)$$

Then, from Equation (8), we can derive $(u_0 - k)s - s^2 = v_0 - k(u_0 - k)$. When we substitute it into Equation (10), we get

$$kgs = k[v_0 - k(u_0 - k)] = kv_0 - u_0k^2 + k^3.$$

Equation (7) also indicates that $kv_0 - u_0k^2 + k^3 = w_0$; thus, $kgs = w_0$. When we reexamine w_4 , we find that $w_4 = C[\beta_\phi(\gamma_\psi\mu_\xi + \gamma_\xi\mu_\xi + \gamma_\xi\beta_\psi) + \mu_\psi\mu_\xi(\gamma_\phi + \gamma_\psi + \gamma_\xi)] = Cw_0$. Therefore,

$$H(t)|_{t \rightarrow \infty} \Rightarrow \frac{w_4}{kgs} = \frac{Cw_0}{w_0} = C. \quad (11)$$

These derivations suggest that when people are exposed to a disaster without any intervention for an extended period, their tolerance of the disaster decreases, and they become psychologically exhausted. That is, when neither resistance nor escape is possible, the human system of self-defense becomes overwhelmed and disorganized. In the long run, disaster events produce profound and lasting changes in people's physical arousal, emotion, cognition, and memory, which result in long-term, severe damages. The Adverse Childhood Experiences study (<http://acestudy.org>) has examined the health and social impacts of disasters on 18,000 children and finds that negative consequences are far more prevalent than previously recognized and that the impacts are cumulative. Therefore, if left unaddressed, such damage can cause a wide range of health problems (e.g., heart disease, cancer, chronic lung disease, liver disease, skeletal fractures) and social problems such as homelessness and inability to hold a job [44, 45].

4.2 Macro-level social productivity

Moreover, at any time t during the course of a disaster event, social productivity also decreases. Suppose people in the normal state can produce at a normal level, such that the number of working units before the disaster is C . During the disaster, the number of people in the normal state (i.e., $N(t)$) may change over time because some people may be influenced and psychologically distressed. Thus, the working units available that can produce normal productivity at time t decrease from C to $N(t) + A_1L(t) + A_2M(t) + A_3H(t)$, where A_1, A_2 , and A_3 are weight parameters, such that $0 < A_1, A_2$, and $A_3 < 1$ for the proportions of people in states L, M, and H who can achieve normal productivity, respectively, at time t . People suffering moderate- or high-level damage may not be able to work normally during the disaster, so remaining social productivity at time t during the disaster can be calculated as

$$\text{work forces available} = \frac{(u_1 + Au_3)k^2 - (v_1 + Av_3)k + w_1 + Aw_3}{(g - k)(s - k)} e^{-kt} + \frac{(u_1 + Au_3)s^2 - (v_1 + Av_3)s + w_1 + Aw_3}{(k - s)(g - s)} e^{-st} + \frac{(u_1 + Au_3)g^2 - (v_1 + v_3)g + w_1 + Aw_3}{(s - g)(k - g)} e^{-gt}$$

4.3 Measuring the efficiency of policy interventions

Our analysis in Section 4.1 suggests the need for psychosocial interventions that may relieve anxiety and depression. These interventions should be designed to support four overlapping functions: emotional support (e.g., someone to confide in who provides comfort or emotional attachment), instrumental support (e.g., strong services, money), informational support (e.g., advice/guidance, help with problem solving), and companionship support (e.g., feeling connected to others).

If appropriate intervention efforts occur during time $t = \tau_0$, the transition rates γ_ϕ , γ_ψ , and γ_ξ gradually decrease after τ_0 , as do the deteriorating rates μ_ξ and μ_ψ . From Equation (6), we know that the effects of psychosocial damages at time τ_{0-} can be calculated as $L(\tau_0)$, $M(\tau_0)$, and $H(\tau_0)$. After time τ_0 , the influence of the disaster on the public decreases. If we let $\bar{\gamma}(t)$ be the average influence rate of the disaster on the public's mind after time τ_0 , the average change of the decrease in $\bar{\gamma}$ per unit of time is proportional to the time spent implementing intervention efforts, the types of intervention efforts (e.g., policy designs for mitigating the disaster, supporting service), and the transition rates before the intervention efforts (i.e., before time τ_0). Because types of intervention efforts may differ according to policy designs, for simplicity, we use a constant $P > 0$ to represent the average efficiency of the intervention efforts. In turn,

$$\frac{\Delta \bar{\gamma}}{\Delta t} = -P \times t \times \bar{\gamma}.$$

When Δt becomes arbitrarily small, we can solve this equation as

$$\bar{\gamma}(t) = \bar{\gamma}(\tau_0) e^{-P(t-\tau_0)^2/2}. \quad (12)$$

When $t = \tau_0$, $\bar{\gamma}(t) = \bar{\gamma}(\tau_0)$, which is the average influence rate before time τ_0 . To get $\bar{\gamma}(\tau_0)$, we may require statistical sample data about the target population. Alternatively, we can

calculate it by letting $\bar{\gamma}(\tau_0) = (\gamma_\phi + \gamma_\psi + \gamma_\xi)/3$, with the assumption that γ_ϕ , γ_ψ , and γ_ξ remain unchanged until τ_0 , at which time the intervention efforts occur. In section 3.1 we have presented a measurement for the individual-level psychosocial damage (i.e., equation (4')). We can use this measurement to compute an average influence rate in collective level before time τ_0 . Suppose out of the population of size C there are n people affected by disaster j before time τ_0 . Then the average influence rate of disaster j on the targeted population per unit time before time τ_0 can be obtained using (12').

$$\bar{\gamma}(\tau_0) = \frac{\frac{n}{C} \times \frac{\sum_{i=1}^n Imapct_{ij}}{n}}{\Delta t} = \frac{\sum_{i=1}^n Imapct_{ij}}{C\Delta t}. \quad (12')$$

If we rewrite Equation (8) and let ε represent a minuscule number, which indicates that the impact rate of the disaster is very small, we get

$$P = \frac{\ln \bar{\gamma}(\tau_0) - \ln \varepsilon}{t^2 / 2}. \quad (13)$$

Equation (13) thus shows the efficiency of policy interventions that may be achieved given a minuscule number ε . For instance, if $\varepsilon = 10^{-3}$, the intervention or policy designed would decrease the influence of the disaster such that only 1 out of 1000 people may develop psychosocial damages, given the uncontrolled incursions of the disaster.

4.4 On targeting interventions and resource allocations

The individual-level Equation (4') and the population level's four different levels of psychosocial measurements—normal (N), low (L), moderate (M), and high (H)—can be used to suggest appropriate targeting interventions and resource allocations at time t . To reduce the probability of people becoming affected and moving from normal to low, moderate, or high states, Equation (4) targeted interventions that reducing people's perceived risk. As illustrated in Figure 2, the characteristics of the threat event itself and social influence play important roles in determining perceived risk. Sometimes the uncertainty in the disaster event may increase the level of psychosocial morbidity. When the perceived threat is greater than the tangible exposure (e.g., biological or chemical threat), those who are not in the affected area may still perceive themselves as being at risk and seek medical screening. Understanding the characteristics of a disaster and providing rapid and accurate information to the public thus may reduce uncertainty and avoid unnecessary anxiety.

The analytic solutions to the system (i.e., Equation (6)) projects the proportion of persons affected in each state during the course of a disaster. That is, the solutions project the range and severity of the expected psychosocial consequences. These numbers indicate that the segments of the affected population (i.e., people in the four states) respond differently because they are affected to different degrees. Thus, these numbers suggest the type of interventions and resources needed in each corresponding state. For example, the value of $H(t)$ suggests the scope and volume of supplies needed at time t to care for those who have developed more severe levels of psychological distress. For those people who show anxiety and fear with obvious clinical somatic symptoms, the value of $M(t)$ reflects the required amount of medical care and mental health services.

In next section, we use an example to illustrate the application of our proposed model.

5. An Empirical Application

The outbreak of SARS in 2003 in multiple countries, especially those in Asia and Canada, caused not only extraordinary public health concerns but also tremendous psychological distress, particularly among health care workers. Several studies have examined the psychological impacts of SARS on health care workers in Hong Kong, Singapore, and Toronto [21, 22, 23, 24] and reveal that health care workers suffer much more psychological distress, including anxiety, fear of contagion, feelings of stigmatization, loneliness, boredom, anger, burnout, emotional exhaustion, and a sense of uncertainty, compared with the general population. To illustrate the proposed model, we use the survey results reported in Maunder and colleagues' [22, p. 1931] study. In their study, 769 health care workers at 9 Toronto hospitals that treated SARS patients during the 2003 SARS outbreak completed a survey related to several adverse outcomes. The proportion of health care workers who reported more than two negative consequences during the period from 13 to 26 months after the SARS outbreak appears as the scattered dots in Figure 4.

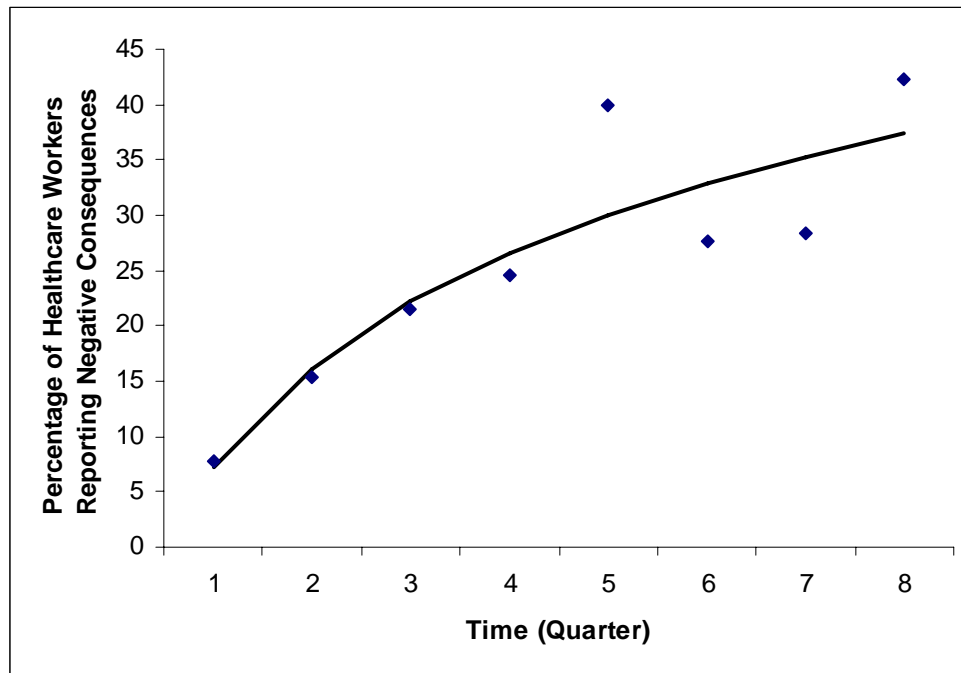


Figure 4. The Prolonged Psychosocial Effects after the SARS Outbreak

Notes: This figure is based on a survey data reported by Maunder and colleagues [25]. The scattered dots in the figure are the percentage of health care workers who reported more than two adverse outcomes during the period from 13 to 26 months after the SARS outbreak. The bold line (predicted psychosocial effects) is based on Equation (6), which measures multiple negative outcomes by summing the low, moderate, and high levels of psychosocial damages (i.e., $L(t) + M(t) + H(t)$). This equation gives the following formula, according to the Mathematica 6 software:

$$\text{Psychosocial effects} = 1 - 0.276e^{-0.52t} - 0.402e^{-0.029t} - 0.384e^{-0.03t}$$

This survey result provides only a summary measure of the negative psychological consequences, whereas our proposed model includes four different levels of psychosocial

measurements: normal (N), low (L), moderate (M), and high (H). To make use of the data and capture multiple negative psychosocial effects at the same time, we add L, M, and H in Equation (6) together to represent the total number of people who displayed psychological distress. Note that the unit measurement that Maunder and colleagues [22] use is the percentage of the population instead of the absolute population number. To reflect this characteristic, we also adopt the percentage as a unit measurement.

Using Mathematica 6 (i.e., non-linear fit function) software and applying Equation (6) (in particular, $L(t) + M(t) + H(t)$) to the survey results, we obtain the following formula:

$$\text{Psychosocial effects} = 1 - 0.276 e^{-0.52t} - 0.402 e^{-0.029t} - 0.384 e^{-0.03t} . \quad (14)$$

The predicted psychosocial effects obtained using Equation (14) appear in Figure 4, represented by the bold line. Comparing the predicted psychosocial effects (bold line) to the actual data (scattered dots), we find that the prediction derived from Equation (14) fits the observed data very well. Meanwhile, the mean absolute prediction error for the proposal model is 3.83 (percentage), which is much smaller than that (5.40, also percentage) of the benchmark exponential model, which relies on the observed data's trend line using Excel. This result demonstrates the significant effectiveness of the proposal model. As we mentioned previously, the proposed model consists of four equations that quantify different levels of psychosocial effects. If such detailed data are available, similar empirical analyses may be performed.

6. Discussion and Conclusions

Whether or not SARS outbreaks recur, other new emerging pathogens, such as a pandemic caused by a deadly avian influenza virus (H5N1), offer ongoing threats. Even though disaster-induced negative psychological outcomes take time, the resultant psychosocial effects start immediately after a disaster occurs. For example, widespread emotional reactions such as fear and anxiety are common responses to imminent threats and actual disaster events. These effects have important impacts on the effectiveness of the response efforts used to mitigate disasters and may decrease social productivity.

We introduce differential dynamics to analyze and evaluate both individual-level and population-level psychosocial impacts during a disaster event. Since the duration of a disaster is short compared to the life of an individual, the size of the target population is assumed to remain constant and the death toll due to the disaster itself is counted in the severe state H. Because previous research has found that the impact of disaster-/threat-induced psychological damage is cumulative, additive, and summative, a continuous system approach is used to capture such psychological damage. The resulting equation (6) can be applied to different time frames such as hours, several days or weeks without changing any parameters. Our modeling efforts shed greater light on how people respond to a threat and the dynamics of psychological effects. The analytic solutions project the range and severity of the possible psychosocial consequences of a disaster and suggest the extent to which these consequences influence effective behavioral responses.

In applying our proposed model to examine the potential psychosocial effects of an infectious disease like SARS, we demonstrate that the model's predictions are highly consistent with the observed empirical data. Thus, we consider this work a first step in developing mathematical models to understand the psychosocial effects of a disaster and how such impacts can affect social productivity. We also show that psychosocial effects reduce social productivity

and compromise response efforts; they may even cause social disruption if no intervention gets deployed during the early stages of the disaster. Therefore, planning for behavioral health responses in advance is urgent and important. In our view, dynamic systems theory and mathematical modeling can lead to additional important advances in planning for such responses.

Appendix A: A Detailed Procedure for Solution Development

In this appendix, we show how we derive Equation (6), the analytical solution to the model system. For easy reference, we use new labels (a1)–(a4) to denote each equation in our model System (5). That is,

$$\frac{dL(t)}{dt} = -(\beta_\phi + \mu_\psi)L(t) + \beta_\psi M(t) + \gamma_\phi N(t), \quad (\text{a1})$$

$$\frac{dM(t)}{dt} = \mu_\psi L(t) - (\beta_\psi + \mu_\xi)M(t) + \gamma_\psi N(t), \quad (\text{a2})$$

$$\frac{dH(t)}{dt} = \mu_\xi M(t) + \gamma_\xi N(t), \text{ and} \quad (\text{a3})$$

$$\frac{dN(t)}{dt} = \beta_\phi L(t) - (\gamma_\phi + \gamma_\psi + \gamma_\xi)N(t), \quad (\text{a4})$$

where the initial conditions are as follows: $N(0) = C$, $L(0) = 0$, $M(0) = 0$, and $H(0) = 0$ (i.e., $t = 0$); and the equation $N(t) + M(t) + L(t) + H(t) = C$ holds at any time t . We use Laplace transform technique to solve (a1)–(a4).

Step 1. Transform (a1) – (a4) into four algebraic equations.

Let $a(p) = \mathbf{Lap}[L(t)]$, $b(p) = \mathbf{Lap}[M(t)]$, $c(p) = \mathbf{Lap}[H(t)]$, and $d(p) = \mathbf{Lap}[N(t)]$.

The Laplace transform of a variable $x(t)$ can be written as:

$$\mathbf{Lap}[x(t)] = X(p), \mathbf{Lap}[\dot{x}(t)] = p \times X(p) - x(0), \text{ and } \mathbf{Lap}[K] = K \times \frac{1}{p}.$$

On the basis of this formula, we apply the Laplace transform to (a1)–(a4) and obtain the following four algebraic equations, respectively:

$$\begin{aligned} a(p) [p + \beta_\phi + \mu_\psi] - \beta_\psi b(p) - \gamma_\phi d(p) &= L(0), \\ -\mu_\psi a(p) + (p + \beta_\psi + \mu_\xi) b(p) - \gamma_\psi d(p) &= M(0), \\ -\mu_\xi b(p) + p c(p) - \gamma_\xi d(p) &= H(0), \text{ and} \\ -\beta_\phi a(p) + (p + \gamma_\phi + \gamma_\psi + \gamma_\xi) d(p) &= N(0). \end{aligned} \quad (\text{a5})$$

Rewriting (a5) in a matrix format yields

$$\begin{bmatrix} p + \beta_\phi + \mu_\psi & -\beta_\psi & 0 & -\gamma_\phi \\ -\mu_\psi & p + \beta_\psi + \mu_\xi & 0 & -\gamma_\psi \\ 0 & -\mu_\xi & p & -\gamma_\xi \\ -\beta_\phi & 0 & 0 & p + \gamma_\phi + \gamma_\psi + \gamma_\xi \end{bmatrix} * \begin{bmatrix} a(p) \\ b(p) \\ c(p) \\ d(p) \end{bmatrix} = \begin{bmatrix} L(0) \\ M(0) \\ H(0) \\ N(0) \end{bmatrix}. \quad (\text{a6})$$

Step 2. Solve (a6) for $a(p)$, $b(p)$, $c(p)$, and $d(p)$.

Obviously,

$$a(p) = \frac{\Delta_a(p)}{\Delta(p)}, b(p) = \frac{\Delta_b(p)}{\Delta(p)}, c(p) = \frac{\Delta_c(p)}{\Delta(p)}, \text{ and } d(p) = \frac{\Delta_d(p)}{\Delta(p)}, \quad (\text{a7})$$

where

$$\Delta(p) = \begin{vmatrix} p + \beta_\phi + \mu_\psi & -\beta_\psi & 0 & -\gamma_\phi \\ -\mu_\psi & p + \beta_\psi + \mu_\xi & 0 & -\gamma_\psi \\ 0 & -\mu_\xi & p & -\gamma_\xi \\ -\beta_\phi & 0 & 0 & p + \gamma_\phi + \gamma_\psi + \gamma_\xi \end{vmatrix},$$

$$\Delta_a(p) = \begin{vmatrix} L(0) & -\beta_\psi & 0 & -\gamma_\phi \\ M(0) & p + \beta_\psi + \mu_\xi & 0 & -\gamma_\psi \\ H(0) & -\mu_\xi & p & -\gamma_\xi \\ N(0) & 0 & 0 & p + \gamma_\phi + \gamma_\psi + \gamma_\xi \end{vmatrix},$$

$$\Delta_b(p) = \begin{vmatrix} p + \beta_\phi + \mu_\psi & L(0) & 0 & -\gamma_\phi \\ -\mu_\psi & M(0) & 0 & -\gamma_\psi \\ 0 & H(0) & p & -\gamma_\xi \\ -\beta_\phi & N(0) & 0 & p + \gamma_\phi + \gamma_\psi + \gamma_\xi \end{vmatrix},$$

$$\Delta_c(p) = \begin{vmatrix} p + \beta_\phi + \mu_\psi & -\beta_\psi & L(0) & -\gamma_\phi \\ -\mu_\psi & p + \beta_\psi + \mu_\xi & M(0) & -\gamma_\psi \\ 0 & -\mu_\xi & H(0) & -\gamma_\xi \\ -\beta_\phi & 0 & N(0) & p + \gamma_\phi + \gamma_\psi + \gamma_\xi \end{vmatrix}, \text{ and}$$

$$\Delta_d(p) = \begin{vmatrix} p + \beta_\phi + \mu_\psi & -\beta_\psi & 0 & L(0) \\ -\mu_\psi & p + \beta_\psi + \mu_\xi & 0 & M(0) \\ 0 & -\mu_\xi & p & H(0) \\ -\beta_\phi & 0 & 0 & N(0) \end{vmatrix}.$$

Step 3. Evaluate $\Delta(p)$.

Expanding the matrix $\Delta(p)$, we have

$$\begin{aligned} \Delta(p) &= (-1)^{4+1} (-\beta_\phi) \begin{vmatrix} -\beta_\psi & 0 & -\gamma_\phi \\ p + \beta_\psi + \mu_\xi & 0 & -\gamma_\psi \\ -\mu_\xi & p & -\gamma_\xi \end{vmatrix} \\ &\quad + (p + \gamma_\phi + \gamma_\psi + \gamma_\xi) \begin{vmatrix} p + \beta_\phi + \mu_\psi & -\beta_\psi & 0 \\ -\mu_\psi & p + \beta_\psi + \mu_\xi & 0 \\ 0 & -\mu_\xi & p \end{vmatrix} \\ &= \beta_\phi [-\gamma_\phi (p + \beta_\psi + \mu_\xi) p - (-\beta_\psi) p (-\gamma_\psi)] + (p + \gamma_\phi + \gamma_\psi + \gamma_\xi) \\ &\quad \times [p (p + \beta_\psi + \mu_\xi) (p + \beta_\phi + \mu_\psi) - p (-\mu_\psi) (-\beta_\psi)] \\ &= p \{ (p + \gamma_\phi + \gamma_\psi + \gamma_\xi) [p (p + \beta_\psi + \mu_\xi) + \mu_\psi p + \mu_\psi \mu_\xi] \\ &\quad + \beta_\phi [p (p + \beta_\psi + \mu_\xi + \gamma_\psi + \gamma_\xi) + \gamma_\psi \mu_\xi + \mu_\xi \gamma_\xi + \gamma_\xi \beta_\psi] \} \end{aligned}$$

$$= p [p^3 + u_0 p^2 + v_0 p + w_0]. \quad (\text{a8}')$$

In addition,

$$u_0 = \gamma_\phi + \gamma_\psi + \gamma_\xi + \beta_\phi + \beta_\psi + \mu_\psi + \mu_\xi,$$

$$v_0 = \mu_\xi(\gamma_\phi + \gamma_\psi + \gamma_\xi + \beta_\phi + \mu_\psi) + \beta_\phi(\gamma_\xi + \beta_\psi + \gamma_\psi) + (\gamma_\phi + \gamma_\psi + \gamma_\xi)(\beta_\psi + \mu_\psi), \text{ and}$$

$$w_0 = \mu_\xi \mu_\psi (\gamma_\phi + \gamma_\psi + \gamma_\xi) + \beta_\phi (\beta_\psi \gamma_\xi + \mu_\xi \gamma_\xi + \gamma_\psi \mu_\xi).$$

Rewriting (a8'), we obtain

$$\begin{aligned} \Delta(p) &= p [p^3 + u_0 p^2 + v_0 p + w_0] \\ &= p (p+k)(p+s)(p+g), \end{aligned} \quad (\text{a8})$$

where k, s, and q are the three roots of the equation $p^3 + u_0 p^2 + v_0 p + w_0 = 0$.

Step 4. Solve $\Delta_d(p)$ for $N(t)$.

$$\begin{aligned} \Delta_d(p) &= \beta_\phi \begin{vmatrix} -\beta_\psi & 0 & L(0) \\ p + \beta_\psi + \mu_\xi & 0 & M(0) \\ -\mu_\xi & p & H(0) \end{vmatrix} + N(0) \begin{vmatrix} p + \beta_\phi + \mu_\psi & -\beta_\psi & 0 \\ -\mu_\psi & p + \beta_\psi + \mu_\xi & 0 \\ 0 & -\mu_\xi & p \end{vmatrix} \\ &= \beta_\phi [p\beta_\psi M(0) + L(0)p(\mu_\xi + p + \beta_\psi)] \\ &\quad + N(0) [(p + \beta_\phi + \mu_\xi)(p + \beta_\psi + \mu_\psi)p - (-\beta_\psi)(-\mu_\psi)p] \\ &= p \{ p^2 N(0) + p [\beta_\phi L(0) + (\beta_\phi + \mu_\psi + \beta_\psi + \mu_\xi) N(0)] \\ &\quad + L(0) (\beta_\phi \beta_\psi + \mu_\xi \beta_\phi) + M(0) \beta_\psi \beta_\phi \\ &\quad + N(0) (\beta_\phi \beta_\psi + \mu_\xi \beta_\phi + \mu_\psi \gamma_\psi) \} \\ &= p (u_1 p^2 + v_1 p + w_1), \end{aligned} \quad (\text{a9})$$

where $u_1 = N(0)$,

$$v_1 = \beta_\phi L(0) + (\beta_\phi + \beta_\psi + \mu_\psi + \mu_\xi) N(0), \text{ and}$$

$$w_1 = (\beta_\phi \beta_\psi + \beta_\phi \mu_\xi) L(0) + \beta_\phi \beta_\psi M(0) + (\beta_\phi \beta_\psi + \beta_\phi \mu_\xi + \mu_\xi \mu_\psi) N(0).$$

Substituting (a8) and (a9) into (a7) yields

$$d(p) = \frac{\Delta_d(p)}{\Delta(p)} = \frac{p(u_1 p^2 + v_1 p + w_1)}{p(p+k)(p+s)(p+g)} = \frac{X_1}{p-(-k)} + \frac{Y_1}{p-(-s)} + \frac{Z_1}{p-(-g)}. \quad (\text{a10})$$

Solving for (a10), we have

$$X_1 = \frac{u_1 k^2 - v_1 k + w_1}{(g-k)(s-k)}, \quad Y_1 = \frac{u_1 s^2 - v_1 s + w_1}{(k-s)(k-g)}, \text{ and } Z_1 = \frac{u_1 g^2 - v_1 g + w_1}{(k-g)(s-g)}.$$

After we take the inverse Laplace transform of equation (a10), the solution is

$$\begin{aligned} N(t) &= \text{Lap}^{-1}[d(p)] \\ &= \text{Lap}^{-1}\left(\frac{X_1}{p-(-k)}\right) + \text{Lap}^{-1}\left(\frac{Y_1}{p-(-s)}\right) + \text{Lap}^{-1}\left(\frac{Z_1}{p-(-g)}\right) \\ &= X_1 e^{-kt} + Y_1 e^{-st} + Z_1 e^{-gt}. \end{aligned}$$

Step 5. Solve $\Delta_b(p)$ for $M(t)$.

$$\begin{aligned}
\Delta_b(p) &= (-1)^{4+1}(-\beta_\phi) \begin{vmatrix} L(0) & 0 & -\gamma_\phi \\ M(0) & 0 & -\gamma_\psi \\ H(0) & p & -\gamma_\xi \end{vmatrix} + N(0) \begin{vmatrix} p + \beta_\phi + \mu_\psi & 0 & -\gamma_\phi \\ -\mu_\psi & 0 & -\gamma_\psi \\ 0 & p & -\gamma_\xi \end{vmatrix} \\
&\quad + (p + \gamma_\phi + \gamma_\psi + \gamma_\xi) \begin{vmatrix} p + \beta_\phi + \mu_\psi & L(0) & 0 \\ -\mu_\psi & M(0) & 0 \\ 0 & H(0) & p \end{vmatrix} \\
&= \beta_\phi [p(-\gamma_\phi)M(0) - L(0)p(-\gamma_\psi)] + N(0) [\gamma_\phi\mu_\psi p + (p + \beta_\phi + \mu_\psi)\gamma_\psi p] \\
&\quad + (p + \gamma_\phi + \gamma_\psi + \gamma_\xi) [(p + \beta_\phi + \mu_\psi)M(0)p - p(-\mu_\psi)L(0)] \\
&= p[(\beta_\phi\gamma_\psi L(0) - \beta_\phi\gamma_\phi M(0)) \\
&\quad + (\gamma_\phi\mu_\psi N(0) + \beta_\phi\gamma_\psi N(0) + \mu_\psi\gamma_\psi N(0) + p\gamma_\psi N(0)) + p^2 M(0) \\
&\quad + p(\beta_\phi M(0) + \mu_\psi M(0) + \mu_\psi L(0) + \gamma_\psi M(0) + \gamma_\phi L(0) + \gamma_\xi L(0) \\
&\quad + (\gamma_\phi\beta_\phi + \gamma_\phi\mu_\psi)M(0) + (\gamma_\xi + \gamma_\psi)(\beta_\phi + \mu_\psi)M(0) + \gamma_\phi\mu_\psi L(0) \\
&\quad + (\gamma_\psi + \gamma_\xi)L(0)\mu_\psi] \\
&= p[u_2 p^2 + v_2 p + w_2], \tag{a11}
\end{aligned}$$

where, $u_2 = M(0)$,

$$v_2 = \gamma_\psi N(0) + (\beta_\phi + \mu_\psi + \gamma_\phi + \gamma_\psi + \gamma_\xi) M(0) + \mu_\psi L(0), \text{ and}$$

$$w_2 = (\beta_\phi\gamma_\psi + \gamma_\phi\mu_\psi + \mu_\psi\gamma_\psi + \mu_\psi\gamma_\xi) L(0) + (\gamma_\phi\mu_\psi + \beta_\phi\gamma_\psi + \gamma_\psi\mu_\psi) N(0) \\ + (\gamma_\phi\mu_\psi + \gamma_\psi\beta_\phi + \beta_\phi\gamma_\xi + \gamma_\xi\mu_\psi + \mu_\psi\gamma_\psi) M(0).$$

Substituting (a8) and (a11) into (a7) yields

$$b(p) = \frac{\Delta_b(p)}{\Delta(p)} = \frac{p(u_2 p^2 + v_2 p + w_2)}{p(p+k)(p+s)(p+g)} = \frac{X_2}{p-(-k)} + \frac{Y_2}{p-(-s)} + \frac{Z_2}{p-(-g)}. \tag{a12}$$

Solving for (a12), we have

$$X_2 = \frac{u_2 k^2 - v_2 k + w_2}{(g-k)(s-k)}, \quad Y_2 = \frac{u_2 s^2 - v_2 s + w_2}{(k-s)(k-g)}, \quad \text{and} \quad Z_2 = \frac{u_2 g^2 - v_2 g + w_2}{(k-g)(s-g)}.$$

After we take the inverse Laplace transform of (a12), we get

$$\begin{aligned}
M(t) &= Lap^{-1}[b(p)] \\
&= Lap^{-1}\left(\frac{X_2}{p-(-k)}\right) + Lap^{-1}\left(\frac{Y_2}{p-(-s)}\right) + Lap^{-1}\left(\frac{Z_2}{p-(-g)}\right) \\
&= X_2 e^{-kt} + Y_2 e^{-st} + Z_2 e^{-gt}.
\end{aligned}$$

Step 6. Solve $\Delta_a(p)$ for $L(t)$.

$$\Delta_a(p) = -N(0) \begin{vmatrix} -\beta_\psi & 0 & -\gamma_\phi \\ p + \beta_\psi + \mu_\xi & 0 & -\gamma_\psi \\ -\mu_\xi & p & -\gamma_\xi \end{vmatrix} +$$

$$\begin{aligned}
& (p + \gamma_\phi + \gamma_\psi + \gamma_\xi) \begin{vmatrix} L(0) & -\beta_\psi & 0 \\ M(0) & p + \beta_\psi + \mu_\xi & 0 \\ H(0) & -\mu_\xi & p \end{vmatrix} \\
&= -N(0) [-\gamma_\phi(p + \beta_\psi + \mu_\xi)p - (-\beta_\psi)p(-\gamma_\psi)] \\
&\quad + (p + \gamma_\phi + \gamma_\psi + \gamma_\xi) [L(0)p(p + \beta_\psi + \mu_\xi) - (-\beta_\psi)M(0)p] \\
&= p\{ p\gamma_\phi N(0) + N(0)[\gamma_\phi\beta_\psi + \gamma_\phi\mu_\xi + \beta_\psi\gamma_\psi] \\
&\quad + (p + \gamma_\phi + \gamma_\psi + \gamma_\xi) [L(0)p + L(0)(\beta_\psi + \mu_\xi) + \beta_\psi M(0)] \} \\
&= p\{ p^2 L(0) + p[\gamma_\phi N(0) + \beta_\psi M(0) + (\gamma_\phi + \gamma_\psi + \gamma_\xi + \beta_\psi + \mu_\xi)L(0)] \\
&\quad + N(0)[\gamma_\phi(\beta_\psi + \mu_\xi) + \beta_\psi\gamma_\psi] \\
&\quad + (\gamma_\phi + \gamma_\psi + \gamma_\xi)[L(0)(\beta_\psi + \mu_\xi) + M(0)\beta_\psi] \} \\
&= p[u_3 p^2 + v_3 p + w_3], \tag{a13}
\end{aligned}$$

where $u_3 = L(0)$,

$$v_3 = \gamma_\phi N(0) + \beta_\psi M(0) + (\beta_\psi + \mu_\xi + \gamma_\phi + \gamma_\psi + \gamma_\xi) L(0), \text{ and}$$

$$w_3 = (\gamma_\psi + \gamma_\phi + \gamma_\xi)[(\beta_\psi + \mu_\xi)L(0) + \beta_\psi M(0)] + (\gamma_\phi\beta_\psi + \mu_\xi\gamma_\phi + \beta_\psi\gamma_\psi)N(0).$$

Substituting (a8) and (a13) back into (a7), we get

$$a(p) = \frac{\Delta_a(p)}{\Delta(p)} = \frac{p(u_3 p^2 + v_3 p + w_3)}{p(p+k)(p+s)(p+g)} = \frac{X_3}{p-(-k)} + \frac{Y_3}{p-(-s)} + \frac{Z_3}{p-(-g)}. \tag{a14}$$

Solving for (a14) then yields

$$X_3 = \frac{u_3 k^2 - v_3 k + w_3}{(g-k)(s-k)}, \quad Y_3 = \frac{u_3 s^2 - v_3 s + w_3}{(k-s)(k-g)}, \quad \text{and} \quad Z_3 = \frac{u_3 g^2 - v_3 g + w_3}{(k-g)(s-g)}.$$

After taking the inverse Laplace transform of equation (a14), we obtain

$$\begin{aligned}
L(t) &= Lap^{-1}[a(p)] \\
&= Lap^{-1}\left(\frac{X_3}{p-(-k)}\right) + Lap^{-1}\left(\frac{Y_3}{p-(-s)}\right) + Lap^{-1}\left(\frac{Z_3}{p-(-g)}\right) \\
&= X_3 e^{-kt} + Y_3 e^{-st} + Z_3 e^{-gt}.
\end{aligned}$$

Step 7. Solve $\Delta_c(p)$ for $H(t)$.

$$\begin{aligned}
\Delta_c(p) &= \gamma_\phi \begin{vmatrix} -\mu_\psi & p + \beta_\psi + \mu_\xi & M(0) \\ 0 & -\mu_\xi & H(0) \\ -\beta_\phi & 0 & N(0) \end{vmatrix} - \gamma_\psi \begin{vmatrix} p + \beta_\phi + \mu_\psi & -\beta_\psi & L(0) \\ 0 & -\mu_\xi & H(0) \\ -\beta_\phi & 0 & N(0) \end{vmatrix} \\
&\quad + \gamma_\xi \begin{vmatrix} p + \beta_\phi + \mu_\psi & -\beta_\psi & L(0) \\ -\mu_\psi & p + \beta_\psi + \mu_\xi & M(0) \\ -\beta_\phi & 0 & N(0) \end{vmatrix} \\
&\quad + (p + \gamma_\phi + \gamma_\psi + \gamma_\xi) \begin{vmatrix} p + \beta_\phi + \mu_\psi & -\beta_\psi & L(0) \\ -\mu_\psi & p + \beta_\psi + \mu_\xi & M(0) \\ 0 & -\mu_\xi & H(0) \end{vmatrix}
\end{aligned}$$

$$\begin{aligned}
&= p [(-\beta_\phi)\gamma_\phi H(0)] + \beta_\phi\gamma_\phi [-H(0)(\beta_\psi + \mu_\xi) - \mu_\xi M(0)] + \gamma_\phi\mu_\psi\mu_\xi N(0) \\
&\quad + p(\gamma_\psi\mu_\xi N(0) + (\beta_\phi + \mu_\psi)\mu_\xi\gamma_\psi N(0) + \gamma_\psi\mu_\xi\beta_\phi L(0) - \gamma_\psi\beta_\psi\beta_\phi H(0)) \\
&\quad + p^2\gamma_\xi N(0) + p[\gamma_\xi\beta_\phi(N(0) + L(0)) + (\mu_\psi + \beta_\psi + \mu_\xi)\gamma_\xi N(0)] \\
&\quad + \gamma_\xi N(0)(\beta_\phi\beta_\psi + \beta_\phi\mu_\xi + \mu_\xi\mu_\psi) + \gamma_\xi\beta_\phi\beta_\psi(M(0) + L(0)) + \gamma_\xi L(0)\beta_\phi\mu_\xi \\
&\quad + (p + \gamma_\phi + \gamma_\psi + \gamma_\xi)\{(p + \beta_\phi + \mu_\psi)(p + \beta_\psi + \mu_\xi)H(0) + (-\mu_\psi)(-\mu_\xi)L(0) \\
&\quad\quad - (p + \beta_\phi + \mu_\psi)M(0)(-\mu_\xi) - H(0)(-\mu_\psi)(-\beta_\psi)\} \\
&= p^3 H(0) + p^2[\gamma_\xi N(0) + \mu_\xi M(0) + H(0)(\mu_\psi + \beta_\psi + \beta_\phi + \mu_\xi + \gamma_\xi + \gamma_\phi + \gamma_\psi)] \\
&\quad + p\{(\gamma_\psi + \gamma_\xi)\mu_\xi[C - L(0)] + \gamma_\xi\beta_\phi[C - M(0)] + \mu_\psi\mu_\xi[C - N(0)] \\
&\quad\quad + \gamma_\xi(\mu_\psi + \beta_\psi)[N(0) + H(0)] + \mu_\xi(\beta_\phi + \gamma_\phi)[M(0) + H(0)] \\
&\quad\quad + H(0)[\beta_\phi(\gamma_\psi + \beta_\psi) + (\mu_\psi + \beta_\psi)(\gamma_\psi + \gamma_\phi)]\} \\
&\quad + [N(0) + L(0) + M(0) + H(0)] \times \\
&\quad\quad [\beta_\phi(\gamma_\psi\mu_\xi + \mu_\xi\gamma_\xi + \gamma_\xi\beta_\psi) + \mu_\xi\mu_\psi(\gamma_\phi + \gamma_\psi + \gamma_\xi)] \\
&= Qp^3 + u_4p^2 + v_4p + w_4, \tag{a15}
\end{aligned}$$

where $Q = H(0)$,

$$u_4 = \gamma_\xi N(0) + \mu_\xi M(0) + (\beta_\psi + \beta_\phi + \mu_\xi + \mu_\psi + \gamma_\phi + \gamma_\psi + \gamma_\xi) H(0),$$

$$\begin{aligned}
v_4 &= \mu_\xi(\gamma_\psi + \gamma_\xi)[C - L(0)] + \gamma_\xi\beta_\phi[C - M(0)] + \mu_\xi\mu_\psi[C - N(0)] \\
&\quad + \gamma_\xi(\beta_\psi + \mu_\psi)[N(0) + H(0)] + \mu_\xi(\beta_\phi + \gamma_\phi)[M(0) + H(0)] \\
&\quad + [\beta_\phi(\gamma_\psi + \beta_\psi) + (\gamma_\phi + \gamma_\psi)(\beta_\psi + \mu_\psi)]H(0), \text{ and}
\end{aligned}$$

$$w_4 = C[\beta_\phi(\gamma_\psi\mu_\xi + \mu_\xi\gamma_\xi + \gamma_\xi\beta_\psi) + \mu_\psi\mu_\xi(\gamma_\phi + \gamma_\psi + \gamma_\xi)].$$

Substituting (a8) and (a15) back into (a7), we get

$$c(p) = \frac{\Delta_c(p)}{\Delta(p)} = \frac{Qp^3 + u_4p^2 + v_4p + w_4}{p(p+k)(p+s)(p+g)} = \frac{l}{p} + \frac{X_4}{p-(-k)} + \frac{Y_4}{p-(-s)} + \frac{Z_4}{p-(-g)}. \tag{a16}$$

Then, by expanding (a16), we can get the following four algebraic equations:

$$Q = l + X_4 + Y_4 + Z_4,$$

$$u_4 = -l(s+k+g) - X_4(s+g) - Y_4(k+g) - Z_4(s+k),$$

$$v_4 = l(sk + kg + gs) + X_4sg + Y_4kg + Z_4sk, \text{ and}$$

$$w_4 = -lskg.$$

Solving these four equations yields

$$X_4 = \frac{-Qk^3 + u_4k^2 - v_4k + w_4}{-k(g-k)(s-k)}, \quad Y_4 = \frac{-Qs^3 + u_4s^2 - v_4s + w_4}{-s(k-s)(k-g)},$$

$$Z_4 = \frac{-Qg^3 + u_4g^2 - v_4g + w_4}{-g(k-g)(s-g)}, \text{ and } l = \frac{w_4}{kgs}.$$

After taking the inverse Laplace transform of equation (a16), we realize

$$\begin{aligned}
H(t) &= Lap^{-1}[c(p)] = Lap^{-1}\left(\frac{l}{p} + \frac{X_4}{p-(-k)} + \frac{Y_4}{p-(-s)} + \frac{Z_4}{p-(-g)}\right) \\
&= l + X_4 e^{-kt} + Y_4 e^{-st} + Z_4 e^{-gt}.
\end{aligned}$$

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