



Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Elsevier hereby grants permission to make all its COVID-19-related research that is available on the COVID-19 resource centre - including this research content - immediately available in PubMed Central and other publicly funded repositories, such as the WHO COVID database with rights for unrestricted research re-use and analyses in any form or by any means with acknowledgement of the original source. These permissions are granted for free by Elsevier for as long as the COVID-19 resource centre remains active.

## Journal Pre-proofs

Waves of infection emerging from coupled social and epidemiological dynamics

Yoh Iwasa, Rena Hayashi

PII: S0022-5193(22)00357-5  
DOI: <https://doi.org/10.1016/j.jtbi.2022.111366>  
Reference: YJTBI 111366

To appear in: *Journal of Theoretical Biology*

Received Date: 5 September 2022  
Revised Date: 16 November 2022  
Accepted Date: 18 November 2022



Please cite this article as: Y. Iwasa, R. Hayashi, Waves of infection emerging from coupled social and epidemiological dynamics, *Journal of Theoretical Biology* (2022), doi: <https://doi.org/10.1016/j.jtbi.2022.111366>

This is a PDF file of an article that has undergone enhancements after acceptance, such as the addition of a cover page and metadata, and formatting for readability, but it is not yet the definitive version of record. This version will undergo additional copyediting, typesetting and review before it is published in its final form, but we are providing this version to give early visibility of the article. Please note that, during the production process, errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

© 2022 The Author(s). Published by Elsevier Ltd.

## **Waves of infection emerging from coupled social and epidemiological dynamics**

by

<sup>1,2</sup>Yoh Iwasa and <sup>1</sup>Rena Hayashi

<sup>1</sup> *Department of Biology, Faculty of Science, Kyushu University, 744 Motoooka, Nishi-ku, Fukuoka 819-0395, Japan*

<sup>2</sup> *Institute of Freshwater Biology, Nagano University, 1088 Komaki, Ueda, Nagano 386-0031, Japan*

corresponding author:

Yoh Iwasa  
Department of Biology,  
Faculty of Science,  
Kyushu University,  
744 Motoooka, Nishi-ku,  
Fukuoka 809-0395, Japan

email: [yohiwasa@kyudai.jp](mailto:yohiwasa@kyudai.jp) cell phone: +81-80-5268-2641

(submitted to Journal of Theoretical Biology; September 5, 2022  
revised and resubmitted, November 17)

**Abstract**

The coronavirus (SARS-CoV-2) exhibited waves of infection in 2020 and 2021 in Japan. The number of infected had multiple distinct peaks at intervals of several months. One possible process causing these waves of infection is people switching their activities in response to the prevalence of infection. In this paper, we present a simple model for the coupling of social and epidemiological dynamics. The assumptions are as follows. Each person switches between active and restrained states. Active people move more often to crowded areas, interact with each other, and suffer a higher rate of infection than people in the restrained state. The rate of transition from restrained to active states is enhanced by the fraction of currently active people (conformity), whereas the rate of backward transition is enhanced by the abundance of infected people (risk avoidance). The model may show transient or sustained oscillations, initial-condition dependence, and various bifurcations. The infection is maintained at a low level if the recovery rate is between the maximum and minimum levels of the force of infection. In addition, waves of infection may emerge instead of converging to the stationary abundance of infected people if both conformity and risk avoidance of people are strong. (197 words)

*key words:* coupled behavioral-epidemiological dynamics; homoclinic bifurcation; Hopf bifurcation; conformity; risk avoidance.

## 1. Introduction

Human infectious diseases provide the best examples for studying population dynamics. Many key concepts and novel mathematical techniques for analyzing wild populations have been developed in the study of infectious disease dynamics, this is not surprising, considering the limited availability of time series for of wild animals and plants (Grenfell et al. 2002, 2004).

Some infectious diseases exhibit multiple waves of spreading, as exemplified by the COVID-19 infection in Japan (Fig. 1). In particular, from the beginning of 2020 to the end of 2021, oscillatory behavior was observed in the number of infected patients with five peaks occurring in April and August 2020, and January, May, and August 2021.

In the standard mathematical theory of infectious disease dynamics, the decline in the number of infected individuals after a peak abundance is often explained by a decrease in the number of susceptible individuals (Kermack and McKendrick 1927). However, this mechanism does not necessarily explain the waves of infection in COVID-19 in Japan because the cumulative number of patients infected by the end of 2021 was very low (1.37%; see caption in Fig. 1).

Rather, the multiple waves of COVID-19 infection from 2020 to 2021 are most likely explained by changes in people's behavior in response to the spread of infectious diseases. In Japan, as infection prevailed, the government urged people to adopt the behavior to reduce the infection risk, such as wearing masks, washing hands, and refraining from loud conversations. The government even introduced various legal regulations such as limiting the number of customers in restaurants, restricting large gatherings, and requesting the closure of sports events or theaters. In addition, people

voluntarily changed their behavior by staying at home. These behavioral responses often included time delays, which may have caused instabilities in the equilibrium abundance, and oscillation. Together, these factors may have led to the transient and sustained waves of infection.

One class of models for managing infectious disease dynamics considering human behavioral choices assumes that individuals perform a cost-benefit analysis when choosing their behavior (Funk et al. 2010). Travelers choose locations to visit by avoiding areas with a high disease prevalence (Meloni et al. 2011). Reluga (2010) discussed a differential game model for social distancing in response to an epidemic, in which each individual maximizes his/her own present value considering future events. Fenichel et al. (2011) considered the effect of adaptive human behavior on epidemiological dynamics, in which each person chooses the behavior that maximizes their utility given the current level of prevalence of infection. Wang et al. (2015) thoroughly reviewed the literature. More recently, Arthur et al. (2021) studied a case in which an individual maximizes their own utility function based on delayed information, and discussed the interactions between individual voluntary behavioral choices and governmental control (see also Dönges et al. 2022). These models emphasized the effect of infection dynamics on people's behavior.

The second group of theoretical works on the coupled dynamics of human behavior and epidemiology emphasized that people's behavior of people may be affected by the behavior of other members in the same community. One way of modeling this is to treat "the fear of the disease" as another form of contagion. In this model, a person without fear of the disease may acquire fear after making contact with another person. As the disease itself is also contagious, the model considers double-

contagion dynamics (Epstein et al. 2008). Similar models were analyzed to consider the prosocial awareness of COVID-19 dynamics by fitting the model to the data obtained from Columbia and India (Ghosh and Martcheva 2021). Perra et al. (2011) also treated fear of the disease as a contagion. They discussed several alternative models for behavioral choices. For example, fear may spread based on the current level of prevalence as well as on beliefs. Moreover, the values may be based on local or the global populations. Johnston and Pell (2020) studied the dynamics of the fear of infection and frustration with social distancing during the COVID-19 spread. The fear of contagion may reflect diverse social processes, such as prosociality, social norms, people's opinions and sentiments (Bauch 2005; Fu et al. 2010; Oraby et al. 2014; Jentsch et al. 2021; Kabir et al. 2021) and the information acquisition (d'Onfrio et al. 2007; d'Onfrio and Manfredi 2009; Poletti et al. 2009, 2012).

Many of these theoretical studies employed models with a large number of variables and parameters. Some of them even adopted individual-based modeling in an explicit space or in a network of individuals. Hence, these models were analyzed almost entirely numerically or by computer simulations (except for the stability of the disease-free state).

In this study, we construct a very simplified model for coupled social and epidemiological dynamics. To obtain a clear understanding of the behavior of the model, we deliberately choose a simple model. We assume that each person switches between active and restrained states: active people travel more often to crowded areas, interact with others, and suffer a higher rate of infection than people in the restrained state. The key assumptions of the model are that the rate of transition from restrained to active states is enhanced by the fraction of currently active people (conformity),

whereas the rate of backward transition is enhanced by the abundance of infected people (risk avoidance). We identify all equilibria and their local stability, perform a bifurcation analysis, and obtain insights into the conditions for the different behaviors of the dynamics. The model can show temporary or sustained oscillations in the number of infected people, initial-condition dependence, and various bifurcations. Multiple waves of infection (indicating repeated resurgence of the infection) may appear if (1) the recovery rate is between the maximum and minimum values of the force of infection and (2) both the risk avoidance and conformity of people are strong.

## 2. Model

Let  $Y$  be the number of individuals infected with the virus. Assume people with a fraction of  $z$  are actively spending their time in densely crowded areas of the city, engaging in loud conversations, and in risky behaviors: these people are referred to as "active." Other people (fraction  $1 - z$ ) tend to stay at home and adopt recommended behavior (e.g., universal masking). We refer to this group as "restrained." We denote the infection rates of active and restrained people by  $b_1$  and  $b_0$ , respectively. Active individuals have a higher rate of infection than restrained individuals ( $b_1 > b_0$ ). We consider a situation in which the prevalence of infection is not very high, as was the case of COVID-19 in Japan in year 2020-2021, when the fraction of cumulative fraction of patients was 1.37%. For simplicity, we assume that the number of susceptible hosts is a constant  $x_0$ . We consider the dynamics for  $Y$  as follows:

$$\frac{dY}{dt} = (b_1z + b_0(1 - z))x_0Y - cY \quad (1a)$$

The last term of Eq. (1a) indicates the recovery rate for infected people with a per day recovery rate  $c$ , indicating that the mean length of infection is  $1/c$ .



Each individual switches between the two behavioral states (active and restrained) at random times. We consider the dynamics satisfied by the fraction of active people as follows:

$$\frac{dz}{dt} = (1 - z)B(1 + \gamma z^2) - z(A_0 + A_1 Y) \quad (1b)$$

The first term on the right-hand side of Eq. (1b) indicates the rate of transition from the restrained to active state. The rate is proportional to the current fraction of the restrained people  $1 - z$  with a basic rate  $B$ . In addition, owing to the conformity of people, they become more willing to engage in active behavior when there are more active people. Thus, we assume that this conformity effect occurs at an accelerated rate indicated by the factor  $1 + \gamma z^2$ , where  $\gamma$  is the strength of the conformity.

The second term on the right-hand side of Eq (1b) indicates the rate of transition from restrained to active. It increases with the prevalence of viral infection  $Y$ .  $A_0$  denotes the basic rate of return to the restrained state. In the absence of infection, people stay in the active state for  $1/A_0$  on average. The presence of infection at  $Y > 0$  would make this period  $1/(A_0 + A_1 Y)$  shorter than in the absence of infection.

Eq. (1a) indicates that the number of infected individuals  $Y$  continues increasing with time if  $b_0 x_0 > c$ , leading to the indefinite growth of  $Y$ . This is not consistent with a situation in which the infection level remains small relative to the total number of susceptible individuals. In contrast,  $Y$  continues decreasing with time if  $c > b_1 x_0$ , indicating that the infection is eventually eradicated from the system. This also does not correspond to the situation being considered. Hence, in the following, we focus our analysis by considering  $c$  as between  $b_0 x_0$  and  $b_1 x_0$  ( $b_0 x_0 < c < b_1 x_0$ ). This inequality indicates that the recovery rate is higher than the minimum force of

infection (when all individuals are restrained) and lower than the maximum force of infection (when all individuals are active).

To reduce the number of parameters, we introduce rescaled variables  $y = \frac{A_1}{B\gamma}Y$  and  $\tau = (b_1 - b_0)x_0t$ . We also introduce three quantities:  $z_0 = \frac{c - b_0x_0}{(b_1 - b_0)x_0}$ ,  $\sigma = \frac{B\gamma}{(b_1 - b_0)x_0}$ , and  $\lambda = \frac{A_0}{B\gamma}$ . Then Eq. (1) can be rewritten as follows:

$$\frac{dy}{d\tau} = (z - z_0)y \quad (2a)$$

$$\frac{dz}{d\tau} = \sigma z(f(z) - \lambda - y) \quad (2b)$$

where  $f(z)$  is defined as follows:

$$f(z) = \left(\frac{1-z}{z}\right)\left(z^2 + \frac{1}{\gamma}\right) \quad (2c)$$

See Appendix A for the derivation. We have  $0 < z_0 < 1$  from  $b_0x_0 < c < b_1x_0$ .

## 2.1 Equilibria

Let  $\hat{y}$  and  $\hat{z}$  be the levels of the two variables in the equilibrium of Eq. (2).

According to the calculations in Appendix B, the dynamics may have multiple equilibria. We distinguish these into the two types as follows.

[Type 1] Equilibrium with some infected individuals ( $\hat{y} > 0$ ).

$$\hat{z} = z_0 \text{ and } \hat{y} = f(z_0) - \lambda. \quad (3a)$$

At the equilibrium,  $0 < z_0 < 1$  and  $f(z_0) > \lambda$  hold.

[Type 2] Equilibrium without infectious people ( $\hat{y} = 0$ ).

$$\hat{y} = 0 \text{ and } f(\hat{z}) = 0 \quad (3b)$$

The dynamics of Eq. (2) have at most one equilibrium of type 1. They have multiple type-2 equilibria: these are calculated as the solutions of  $f(\hat{z}) = 0$  satisfying  $0 < \hat{z} < 1$ . In general, a type-2 equilibrium has  $\hat{z}$ , which is different from  $z_0$ .

The distinction between type-1 and type-2 equilibria is important from an epidemiological perspective. A type-1 equilibrium refers to a population that maintains an infectious disease. In contrast, a type-2 equilibrium corresponds to a population in which the infection is eradicated.

We can draw trajectories of the dynamics on the phase plane (or  $(z,y)$ -plane), as illustrated in Fig. 2. The horizontal axis indicates  $z$  and the vertical axis indicates  $y$ . Several parts of the figure show the dynamics given in Eq. (2) for different parameters. The solid circles and open circles represent the locally stable and unstable equilibria of the dynamics, respectively. In Fig. 2a, there is a stable limit cycle showing a perpetual oscillation of people's activity and infection abundance. The number, location, and stability of the equilibria can be calculated mathematically. However, the sizes and shapes of the limit cycles can be known only numerically. By changing the parameters, the number of equilibria and their stability may change. Additionally, the limit cycle for perpetual oscillation may appear or disappear. These represent bifurcation phenomena of several types common to nonlinear dynamics (Strogatz 1994).

## ***2.2 Stability of the equilibria***

In Appendix B, we derive the local stability of the equilibria for the dynamics given in Eq. (2).

### *2.3.1. Local stability of the type-1 equilibrium.*

An equilibrium with a positive abundance of infected ( $\hat{y} > 0$ ) corresponds to a type-1 equilibrium. It is  $(z_0, f(z_0) - \lambda)$ . The local stability is determined from the slope of the isocline  $y = f(z) - \lambda$  at equilibrium as follows:

$$(z_0, f(z_0) - \lambda) \text{ is locally stable if } \frac{df}{dz}(z_0) < 0. \quad (4a)$$

$$(z_0, f(z_0) - \lambda) \text{ is unstable if } \frac{df}{dz}(z_0) > 0. \quad (4b)$$

See Appendix B for derivation.

If  $\gamma < 27$ ,  $y = f(z) - \lambda$  is a monotonically decreasing function of  $z$  and the equilibrium is stable. This implies that the dynamics may converge to an endemic population with some infected hosts.

In contrast, if  $\gamma > 27$ , the function  $f(z)$  has a local minimum and a local maximum denoted by  $z_1$  and  $z_2$ , respectively. These satisfy  $0 < z_1 < z_2 < \frac{1}{2}$ . In addition, if  $z_0$  is between these two values ( $z_1 < z_0 < z_2$ ), the function  $y = f(z)$  has a positive slope at  $z = z_0$ . If  $f(z_0) > \lambda$ , point  $(z_0, f(z_0) - \lambda)$  is an unstable type-1 equilibrium and an unstable focus. This equilibrium may or may not be accompanied by a stable limit cycle, as is known from numerical analyses. Even if  $\gamma > 27$ , if  $z_0 < z_1$  or  $z_0 > z_2$ , the equilibrium is stable and the dynamics converge to the stationary abundance of infected hosts.

### 2.3.2 Local stability of a type-2 equilibrium

The boundary equilibrium (type 2) is  $(\hat{z}, 0)$  with  $f(\hat{z}) = 0$ . The stability of the equilibrium needs to be examined as the deviation from the equilibrium is constrained to the half plane  $y > 0$ . According to the calculations in Appendix B, the local stability of the boundary equilibrium is as follows:

$$(\hat{z}, 0) \text{ is locally stable if both } \frac{df}{dz}(\hat{z}) < 0 \text{ and } \hat{z} < z_0 \text{ hold.} \quad (5a)$$

$$(\hat{z}, 0) \text{ is unstable if } \frac{df}{dz}(\hat{z}) > 0 \text{ holds or if } \hat{z} > z_0 \text{ holds.} \quad (5b)$$

The local stability of the type-2 equilibrium ( $\hat{y} = 0$ ) depends on the sign of the slope of the isocline  $y = f(z) - \lambda$ , or on the sign of  $\frac{df}{dz}(\hat{z})$ . However, it also depends on the sign of  $\hat{z} - z_0$ , unlike the type-1 equilibrium.

### 3. Bifurcations and other nonlinear behavior of the dynamics

As the parameters change continuously, the dynamics may exhibit a transition in qualitative behavior called a "bifurcation" (Strogatz 1994).

Below, we discuss how the number of equilibria and their local stability change as the parameters change. In Appendix C, we explain the dependence on two key parameters  $\lambda$  and  $z_0$ . To analyze the appearance and disappearance of a limit cycle, we need to adopt numerical analyses. Then, we can determine bifurcations of several types.

In this section, we illustrate the several types of bifurcation shown by the model given in Eq. (2). We also show that the dynamics may exhibit a relaxation-oscillation.

#### 3.1 Homoclinic bifurcation

If the type-1 equilibrium ( $\hat{y} > 0$ ) is unstable, based on numerical analysis, it is an unstable focus. Both sustained and transient oscillations are observed around equilibrium. In this case, there are two qualitatively different situations. There can be a stable limit cycle surrounding the unstable focus showing perpetual oscillation of the

dynamics, as illustrated in Fig. 2(a). Alternatively, there may be no limit cycle, and trajectories starting near the unstable focus may exhibit oscillation for a finite number of times, and then converge to an equilibrium at the  $z$ -axis ( $\hat{y} = 0$ ). In this case, the infection is eradicated, as illustrated in Fig. 2(c). Distinguishing between these two requires numerical analyses.

In Fig. 2, the transition of the dynamics is caused by an increase in  $\lambda$ . If  $z_1 < z_0 < z_2$  is satisfied, the type-2 equilibrium is an unstable focus. If  $\lambda$  is small, the unstable focus is surrounded by a stable limit cycle (Fig. 2(a)). As  $\lambda$  increases, the limit cycle approaches an unstable type-2 equilibrium ( $\hat{y} = 0$ ). The oscillation period along the limit cycle becomes longer because the movement of the state point near an equilibrium is slow. When they merge, a trajectory starting from the unstable equilibrium shows a round trip returning to the same equilibrium: this is called a "homoclinic orbit" (Guckenheimer and Holmes 1983; Strogatz 1994) (Fig. 2(b)). As  $\lambda$  increases further, the trajectory converges to a stable type-2 equilibrium where there is no infection (Fig. 2(c)). The transition of dynamics of this type is called "homoclinic bifurcation."

The top and bottom parts of Fig. 3(a) illustrate the amplitudes of oscillation of  $y$  and  $z$  in the limit cycle with the parameter  $\lambda$ . As  $\lambda$  increases, the difference between the maximum and minimum of  $y$  in the limit cycle does not change significantly and the oscillation of  $y$  and  $z$  stops when  $\lambda = 0.1276$ . For  $\lambda > 0.1276$ , the final outcome is the equilibrium without infection ( $\hat{y} = 0$ ). Fig. 3(b) indicates the time change of  $y$  for different values of  $\lambda$ . As  $\lambda$  approaches the critical value 0.1276, the period of oscillation becomes longer, and it becomes infinitely long at the critical value (Strogatz 1994).

### 3.2 Hopf bifurcation

In Fig. 4(a), the top and bottom parts indicate  $y$  and  $z$ , respectively. The horizontal axis is for  $\gamma$ . As mentioned before, the strength of conformity  $\gamma$  needs to be sufficiently large for the system to exhibit persistent oscillation. In particular, for  $\gamma < 27$ , the system does not have an unstable type-1 focus, suggesting that the limit cycle may be possible if  $\gamma$  exceeds 27. Fig. 4(a) illustrates the dependence on  $\gamma$ . A supercritical Hopf bifurcation takes place at approximately  $\gamma \approx 27.2$ .

Another example of Hopf bifurcation is illustrated in Fig. 4(b). The top and bottom parts illustrate  $y$  and  $z$ , respectively. The horizontal axis indicates the parameter  $z_0$ . For  $z_0 > z_2$  and  $z_0 < z_1$ , the system shows convergence to the stable type-1 equilibrium. For  $z_0$  between  $z_1$  and  $z_2$ , the equilibrium  $z = z_0$  is unstable. At  $z_0 = z_1$  and  $z_0 = z_2$ , a supercritical Hopf-bifurcation occurs (Guckenheimer and Holmes 1983; Strogatz 1994). In Fig. 4(b), the unstable focus is surrounded by the stable limit cycle for all values of  $z_0$  within  $z_1 < z_0 < z_2$ .

### 3.3 Transcritical bifurcation

As a parameter changes, a type-1 equilibrium decreases the abundance of infected hosts  $y$ , and ultimately becomes a type-2 equilibrium with  $y = 0$ . When this transition occurs, the type-1 equilibrium merges with an unstable type-2 equilibrium, leaving a single stable type-2 equilibrium. This transition is called a "transcritical" bifurcation (Guckenheimer and Holmes 1983; Strogatz 1994). Further details are provided in Appendix D.

### 3.4. Relaxation oscillation

The dynamics of Eq. (2) have another aspect typical of nonlinear dynamics. Here, we consider the behavior when parameter  $\sigma$  is very large ( $\sigma \gg 1$ ), i.e., the speed of the change in  $z$  is much faster than that in  $y$ . This is the case when people adjust their activity quickly to the current level of infection: correspondingly, the change in the number of infected hosts is slower.

We choose  $z_1 < z_0 < z_2$ , and the type-1 equilibrium is an unstable focus. The oscillation illustrated in Fig. 5(a) is an example of the "relaxation oscillation" which appears in many nonlinear dynamics in engineering and biology (Strogatz 1994; Murray 1989; Iwasa and Pomiankowski 1995). We can imagine a quasi-equilibrium level of activity  $z$  for each level of infected  $y$ . For any value of  $y$  between the local maximum and local minimum of the curve ( $f(z_1) < y < f(z_2)$ ), the fast dynamics of  $z$  have two stable equilibria. As  $y$  changes slowly,  $z$  also changes slowly, remaining in the high or low branches of the quasi-equilibrium: that is called "slow dynamics." While  $z$  remains at the branch of the higher quasi-equilibrium,  $y$  increases slowly and  $z$  declines with time. When  $z$  becomes as low as  $z_2$ ,  $z$  quickly moves to the lower branch of the quasi-equilibrium. Subsequently, slow dynamics occur along the lower branch, in which  $y$  decreases and  $z$  increases. When  $z$  reaches  $z_1$ ,  $y$  quickly moves to the higher branch of the two quasi-stable equilibria. Taken together,  $y$  moves between the maximum value  $f(z_2)$  and minimum value  $f(z_1)$ .

In contrast, Fig. 5(b) illustrates the oscillation when  $\sigma$  is smaller. The length of period is longer and the amplitude is larger than in Fig. 5(a).

## 4. Persistence of infection and perpetual or transient oscillation



The COVID-19 infection in 2020 and 2021 in Japan had the following three characteristics. First, the infection was maintained in the population, rather than being eradicated. Second, the number of infected individuals remained at a low level instead of an indefinite expansion. Third, it exhibited multiple waves rather than a smooth convergence to a stationary state. The latter suggests a transient or sustained oscillation around an unstable equilibrium. In this section, we investigate whether the simple model in Eq. (2) can explain these qualitative behaviors.

Fig. 6 shows a phase plane indicating the parameter regions differing in the presence of an equilibrium, including some infected hosts (type-1) and their local stability. The horizontal axis indicates the recovery rate  $c$  for the infected hosts and the vertical axis indicates  $\lambda = A_0/B\gamma$ , i.e., the rate of active people returning to the restrained state in the absence of enhancement by infection risk ( $A_1y = 0$ ). In region labeled E, the infection level continues to decrease with time. In contrast, in the region labeled G, the number of infected individuals continues to increase with time. Neither of these results is consistent with the behavior observed in Fig. 1. In Fig. 6, the curve represents the graph of  $\lambda = f\left(\frac{c - b_0x_0}{b_1x_0 - b_0x_0}\right)$ . Only under this curve do the dynamics have a type-1 equilibrium with a finite and positive abundance of infected hosts. This occurs when the recovery rate  $c$  is larger than the minimum force of infection when all people are restrained: correspondingly, it is smaller than the maximum force of infection when all people are active ( $b_0x_0 < c < b_1x_0$ ).

In Fig. 6(a), the graph for  $\lambda = f\left(\frac{c - b_0x_0}{b_1x_0 - b_0x_0}\right)$  has one local minimum and one local maximum. The curve has a positive slope for  $c$  between the two values. When a type-1 equilibrium exists (under the curve), the parameter region under the curve  $0 <$

$\lambda < f\left(\frac{c - b_0x_0}{b_1x_0 - b_0x_0}\right)$  is separated into two areas according to the local stability of the type-1 equilibrium. It is locally stable when  $c$  is a value corresponding to a negative slope of the curve. In this case, the number of infected converges smoothly to the stationary state. In contrast, the type-1 equilibrium is an unstable focus when  $c$  is a value corresponding to a positive slope of the curve. Then the dynamics show an oscillation near the equilibrium (Fig. 2(a) and 2(b)). The parameter region corresponding to the latter situation is indicated by the shaded region in Fig. 6(a).

In Fig. 6(b), we illustrate the phase diagram with a smaller  $\gamma$ . The function  $\lambda = f\left(\frac{c - b_0x_0}{b_1x_0 - b_0x_0}\right)$  is monotonically decreasing with  $c$ , and there is no region for the type-1 equilibrium to be stable. Hence, the model does not show oscillation around the equilibrium. A comparison between Fig. 6(a) and 6(b) indicates that a sufficiently strong conformity (large  $\gamma$ ) is needed for either transient or sustained oscillation.

## 5. Discussion

In the present paper, we discussed a simple model for the coupling of disease spread and human behavioral changes. In ecological and environmental sciences, the coupled dynamics of human behavior and ecological systems are called "Social-Ecological Systems" or "Human Environmental Systems" (Folke et al. 2005; Walker et al. 2004; Manuel-Navarrete et al. 2007; Bailey et al. 2019). One example is a model for a lake-water pollution level, where the results are strongly affected by the cooperation levels of local residents (Iwasa et al. 2007, 2010; Suzuki and Iwasa 2009a, 2009b). Recently, mathematical bases for such models have been studied (Sun and Hilker 2020; Heggerud et al. 2022). Other examples include forest harvesting, where the quality of each site is affected by the land use of surrounding sites (Satake and Iwasa 2006; Satake

et al. 2007a, 2007b, 2008); illegal logging in tropical forests (Lee et al. 2015a, 2017, 2018, 2019), ecotourism in Jeju islands (Lee and Iwasa 2011, 2020), and the migration of herders in Mongolian rangeland (Lee et al. 2015b).

### ***5.1 Coupled behavioral and epidemiological dynamics***

Many papers have been published on epidemiological dynamics considering people's behavioral responses. Some works have considered the contact rate chosen by each person to maximize his/her own utility dependent on the prevalence of infection. As the number of infected increases, susceptible individuals recognize the risk and reduce their activity (Fenichel et al. 2011; Arthur et al. 2021). Other studies have considered dynamic optimization, including the risk of harming the expected future payoff (Reluga 2010). Another approach to representing the behavioral change of people is to consider the "fear of infection" as a contagion itself. An individual without fear may encounter with another individual with fear and acquire said fear. In corresponding studies, the model considered both the infection and fear of infection as contagions transmitted between people (Epstein et al. 2008; Johnston and Pell 2020). There have also been studies considering how individual behavior depend both on the presence of other individuals with fear and the presence of infected individuals (Perra et al. 2011). There are also diverse choices for the functional forms (Perra et al. 2011). All of these models include many variables. Many have discussed individual-based models including explicit consideration of the space and network structures of individuals (Wang et al. 2015). The analyses were performed numerically (except for a few; e.g. Poletti et al. 2009).

In the present study, we considered a model with only two variables. A simple model has a merit of providing us explicit mathematical conditions for different behaviors of the system, such as the maintenance of infection and the oscillation. We obtained the conditions for the various bifurcations, such as the homoclinic bifurcation, transcritical bifurcation, Hopf bifurcation, and saddle-node bifurcation.

Fig. 6(a) illustrates the parameter regions for the different behaviors of the model. G indicates the parameter region where the number of infected individuals continues growing, and E indicates the region where the number of infected individuals continues decreasing. If  $b_0x_0 > c$ , the number of infected individuals continues increasing, and does not remain much smaller than the entire population. In contrast, if  $c > b_1x_0$ , the number of infected continues decreasing, and the infection is eventually eradicated. Neither of these is consistent with the situation being considered, in which the infection remained at a rather low level in the population. We focused our analysis for the case  $b_0x_0 < c < b_1x_0$ , i.e., where the recovery rate  $c$  was larger than the force of infection when all individuals are restrained ( $b_0x_0$ ) and smaller than the force of infection when all individuals are active ( $b_1x_0$ ).

Fig. 6(a) indicates that the model has an equilibrium with a positive abundance of infected individuals only when  $\lambda$  is below the curve ( $\lambda < f\left(\frac{c - b_0x_0}{b_1x_0 - b_0x_0}\right)$ ). The parameter range is further separated according to whether the equilibrium is locally stable or unstable. For the dynamics to exhibit an oscillatory abundance of the number of infected individuals, the equilibrium must be an unstable focus, which is possible in the region shaded in Fig. 6(a). The unstable equilibrium may be surrounded by a limit cycle implying a perpetual oscillation, or it may be accompanied by trajectories that lead to the eradication of the infection after transient oscillation.

Perpetual oscillation is possible only when equilibrium with some infected individuals exists, and it is unstable. For this to occur, the function  $f(z)$  must have a portion with a positive derivative. In addition, the balance of  $c - b_0x_0$  and  $b_1x_0 - c$  makes  $z_0$  be within this portion. As shown in Appendix B, the function  $f(z)$  has a portion with a positive derivative only when the conformity  $\gamma$  is sufficiently strong. In addition, if we adopt a linear function  $1 + \tilde{\gamma}z$ , instead of a quadratic function  $1 + \gamma z^2$ , the equilibrium with a positive abundance of infected individuals is always locally stable, and oscillation does not occur (Appendix B). We conclude that the oscillation requires accelerating conformity.

Second, for the presence of the equilibrium with a positive abundance of infected  $\lambda = A_0/B\gamma$  needs to be small.  $\lambda$  is the relative rate for newly active people returning to inactive, independent of the number of infected people. If  $\lambda$  is large, the fraction of active people stays low and an equilibrium with a positive abundance of infected becomes difficult to maintain. In contrast, if  $\lambda$  is small, the fraction of active people can become large in the absence of infected people and can be controlled by the risk avoidance processes owing to the infected people, thereby realizing the equilibrium of positive abundance of infected ( $\hat{y} > 0$ ). Thus, the smallness of  $\lambda$  is related to the need for the risk avoidance of people to be sufficiently strong.

Among the many theoretical papers for coupled behavioral and epidemiological dynamics, some have assumed that people's activity is reduced by the prevalence of infection in the population (Fenichel et al. 2011; Arthur et al. 2021; Reluga 2010), i.e., risk avoidance. Other works have considered the contagion of the fear of infection among people (Epstein 2008; Perra et al. 2011; Johnston and Pell 2020): this approach represents the conformity of the people. The model we employed in this study incorporates both effects in a

simplified manner. Interestingly, in the current study, both risk avoidance and conformity are needed for oscillation to occur.

## **5.2 Future studies**

Even if people's behavior does not change, infectious diseases can produce oscillations through several other mechanisms. The simplest would be an annual oscillation in the transmission rate or supply of susceptible people. Annual fluctuations may produce an oscillation with clear periodicity of two or three years in nonlinear epidemiological dynamics (Earn et al. 2000; Kamo and Sasaki 2005). A nonlinear transmission rate might produce a periodic oscillation in a constant environment (Capasso and Serio 1978; Liu et al. 1986, 1987), which can be interpreted as representing people's reactions to disease prevalence. The number of newly infected could be a nonlinear function of the current number of infected (Grenfell et al. 2002; Tkachenko et al. 2021). How these processes interact with the people's behavioral changes as analyzed herein, this behavior would be an interesting theme for future theoretical study.

The number of infected of COVID-19 in 2020-2021 in Japan stayed rather low and exhibited characteristic oscillation. According to the analysis herein, this behavior is possible only under limited conditions. Testing the predicted conditions is an interesting theme of future study. We need to examine both the nature of the virus (their infection rate is between two levels of the force of infection) and that of society (people respond sensitively to the spread of infection and they have strong conformity).

Many other processes can modify the model parameters. As the proportion of vaccinated individuals increases, the number of susceptible  $x_0$  declines. If a viral strain is

replaced with a mutant, some of the parameters may change. In addition, society and medical treatment systems might change to cope with the infection more effectively.

Novel mutants tend to start from a small number of infected individuals: hence, they suffer a high risk of stochastic extinction. This may have an important effect on viral evolution (e.g. Haraguchi and Sasaki 1997; Sasaki and Haraguchi 2000; Sasaki et al. 2012; see also literatures explained in Hayashi et al. 2022). Incorporating stochasticity owing to the small number of individuals is also an interesting issue for future theoretical studies.

### **Acknowledgements**

We thank C. Gokhale, A. Hara, K. Ito, L. Lehmann, O. Leimer, J.M. McNamara, A. Nagahama, A. Sasaki, A. Satake, H. Seno, and S. Tomimoto for their helpful comments.

Journal Pre-proofs



## Appendix A

### *Simplification of the dynamics*

We start with the dynamics of  $Y$  and  $z$ , as follows:

$$\frac{dY}{dt} = (b_0z + b_1(1 - z))x_0Y - cY \quad (\text{A.1a})$$

$$\frac{dz}{dt} = (1 - z)B(1 + \gamma z^2) - z(A_0 + A_1Y) \quad (\text{A.1b})$$

To reduce the number of parameters and simplify the behavior of the dynamics, we introduce rescaled variables and parameters. We rewrite Eq. (A.1.a) as follows:

$$\begin{aligned} \frac{dY}{dt} &= [(b_0z + b_1(1 - z))x_0 - c]Y \\ &= (b_1 - b_0)x_0 \left[ \frac{b_1x_0 - c}{(b_1 - b_0)x_0} - z \right] Y \end{aligned} \quad (\text{A.2a})$$

Eq. (A.1b) is rewritten as follows:

$$\frac{dz}{dt} = B\gamma z \left[ \frac{1 - z}{z} \left( \frac{1}{\gamma} + z^2 \right) - \frac{A_0}{B\gamma} - \frac{A_1}{B\gamma} Y \right] \quad (\text{A.2b})$$

We introduce the rescaled variables  $y = \frac{A_1}{B\gamma} Y$ ,  $\tau = (b_1 - b_0)x_0 t$ . Eqs. (A.2a) and (A.2b)

become as follows:

$$\frac{dy}{d\tau} = \left[ z - \frac{c - b_0x_0}{(b_1 - b_0)x_0} \right] y \quad (\text{A.3a})$$

$$\frac{dz}{d\tau} = \frac{B\gamma}{(b_1 - b_0)x_0} z \left[ \frac{1 - z}{z} \left( \frac{1}{\gamma} + z^2 \right) - \frac{A_0}{B\gamma} - y \right] \quad (\text{A.3b})$$

We also introduce three quantities calculated from the parameters:  $z_0 = \frac{c - b_0x_0}{(b_1 - b_0)x_0}$ ,  $\sigma =$

$\frac{B\gamma}{(b_1 - b_0)x_0}$ , and  $\lambda = \frac{A_0}{B\gamma}$ . Then Eqs. (A.3a) and (A.3b) can be rewritten as follows:

$$\frac{dy}{d\tau} = (z - z_0)y \quad (\text{A.4a})$$

$$\frac{dz}{d\tau} = \sigma z \left[ \frac{1 - z}{z} \left( \frac{1}{\gamma} + z^2 \right) - \lambda - y \right] \quad (\text{A.4b})$$

These are Eqs. (2a) and (2b) in the text.

## Appendix B

### *Equilibria and their local stability*

We consider the following dynamics as follows:

$$\frac{dy}{d\tau} = (z - z_0)y \quad (\text{B.1a})$$

$$\frac{dz}{d\tau} = \sigma(f(z) - \lambda - y) \quad (\text{B.1b})$$

At the equilibria of these dynamics,  $(z - z_0)y = 0$  and  $f(z) - \lambda - y = 0$  hold. The equilibria are of the following two types:

[type 1] Equilibrium with positive abundance of infected individuals:

$$\hat{z} = z_0 \text{ and } \hat{y} = f(z_0) - \lambda > 0 \quad (\text{B.2})$$

[type 2] Equilibria with no infected patients:

$$\hat{y} = 0 \text{ and } f(\hat{z}) - \lambda = 0 \quad (\text{B.3})$$

If  $f(z_0) \leq \lambda$ , there is no type-1 equilibrium.

The model has either a single type-1 equilibrium or no type-1 equilibrium. If there is one type-1 equilibrium, it can be locally stable or unstable. If it is unstable, it can be accompanied by a stable limit cycle, indicating perpetual oscillation and repeated resurgence of infections. Otherwise, after oscillation of a finite number of times, the trajectories proceed to the equilibrium without infection (type 2).

In contrast, there is always one or more type-2 equilibrium, because the function  $f(z) = \lambda$  has one or more roots satisfying  $0 < z \leq 1$ . If there are multiple type-2 equilibria, they can be locally stable or unstable.

#### ***B.1 Stability of the equilibrium with infection (type 1)***

According to numerical analyses, if there is a stable limit cycle indicating a perpetual oscillation of infection abundance, there exists an unstable equilibrium

surrounded by a limit cycle. Hence, we first consider the condition in which there is an unstable type-1 equilibrium. The type-1 equilibrium indicates a population containing some infected hosts. The local stability of this equilibrium can be determined from the eigenvalue of the linearized dynamics. We calculate the Jacobian matrix at the equilibrium  $(\hat{z}, \hat{y})$ , the four elements of which are as follows:

$$\frac{\partial dy}{\partial y d\tau} = (\hat{z} - z_0) = 0; \quad \frac{\partial dy}{\partial z d\tau} = \hat{y} > 0; \quad \frac{\partial dz}{\partial y d\tau} = -\sigma < 0, \quad \text{and} \quad \frac{\partial dz}{\partial z d\tau} = f'(z_0). \quad (\text{B.4})$$

The characteristic equation for the eigenvalues  $\xi$  of the linearized matrix is as follows:

$$\det \begin{vmatrix} -\xi & \hat{y} \\ -\sigma & \sigma f'(z_0) - \xi \end{vmatrix} = 0$$

This equation becomes as follows:

$$\xi^2 - \sigma f'(z_0)\xi + \sigma\hat{y} = 0. \quad (\text{B.5})$$

Eq. (B.3) has two eigenvalues both with negative real parts if  $f'(z_0) < 0$ ; this is the condition for the local stability of the equilibrium. In contrast, if  $f'(z_0) > 0$ , the equilibrium is unstable. Note that  $f'(z_0)$  is the slope of isocline  $y = f(z) - \lambda$  on the  $(z, y)$ -plane. Hence, we conclude that the positive equilibrium is locally stable if the isocline has a negative slope at the equilibrium, and it is unstable if the isocline has a positive slope at the equilibrium.

The shape of  $y = f(z)$  is examined by considering its derivative.  $f'(z) = 0$  is rewritten as follows:

$$f'(z) = \frac{1}{z^2} \left( z^2 - 2z^3 - \frac{1}{\gamma} \right) \quad (\text{B.6})$$

From this, we have the following:

If  $0 < \gamma < 27$ ,  $f'(z) < 0$  for all  $0 < z < 1$

If  $\gamma > 27$ , there are two solutions of  $f'(z) = 0$ ,  $z_1$  and  $z_2$ , satisfying  $0 < z_1 < z_2 < 0.5$ . Then, the derivative  $f'(z)$  has a sign as follows:

$$f'(z) < 0 \text{ for } 0 < z < z_1 \text{ and } z_2 < z < 1, \quad (\text{B.7a})$$

$$f'(z) > 0 \text{ for } z_1 < z < z_2. \quad (\text{B.7b})$$

Hence, we can conclude as follows:

If  $0 < \gamma < 27$ ,  $f(z)$  is a monotonically decreasing function.

If  $\gamma > 27$ ,  $f(z)$  has a local minimum at  $z_1$  and a local maximum at  $z_2$ , where  $0 < z_1 < z_2 < 0.5$ . For the interval between  $z_1$  and  $z_2$ , there is a portion for which  $y = f(z)$  has a positive slope.

By combining these results with the local stability of the equilibrium with some infected (type 1), we can conclude that the dynamics have an unstable equilibrium if the following three conditions are met: [1]  $\gamma > 27$ ; [2]  $z_0$  is between  $z_1$  and  $z_2$  ( $z_1 < z_0 < z_2$ ); and [3]  $f(z_0) > \lambda$ . Please note that, even if these conditions are met, there may not be a stable limit cycle surrounding the unstable equilibrium. The existence of unstable type-1 equilibrium is a necessary but not sufficient condition for a limit cycle.

## ***B.2 Stability of the equilibrium without infection (type 2)***

Next, we consider the stability of the equilibrium with  $\hat{y} = 0$ . It satisfies  $f(\hat{z}) = \lambda$  and  $0 < \hat{z} < 1$ . We examine the dynamics starting near the equilibrium. Let  $z(t) = \hat{z} + \varepsilon(t)$  and  $Y(t) = \kappa(t)$ , where both  $\varepsilon(t)$  and  $\kappa(t)$  are small and  $\varepsilon(t)$  can be positive or negative, but  $\kappa(t)$  is nonnegative. Using these, Eqs. (B.1) become as follows:

$$\frac{d\varepsilon}{dt} = \sigma \hat{z} f'(\hat{z}) \varepsilon(t) - \sigma \hat{z} \kappa(t) + O(\dots) \quad (\text{B.8a})$$

$$\frac{d\kappa}{dt} = (\hat{z} - z_0) \kappa(t) + O(\dots) \quad (\text{B.8b})$$

In the above,  $O(\dots)$  indicate small terms of second or higher order with respect to  $\varepsilon$  and  $\kappa$ . Because we should consider only a small deviation with  $Y \geq 0$  only, the deviation  $\kappa(\tau)$  is constrained as  $\kappa(\tau) \geq 0$ , and we have the following results:

This boundary equilibrium is locally stable if  $f'(\hat{z}) < 0$  and  $\hat{z} < z_0$ .

It is unstable if either inequality with the reversed direction holds.

(B.9)

This is consistent with the numerical analyses shown in Fig. 4.

### **B.3 Bifurcation diagram**

Here, we consider the bifurcation diagram where the horizontal axis is the parameter  $\lambda$  and the vertical axis is  $z$ . Suppose  $z_0$  is chosen with  $Y = f(z)$  having a positive slope, implying  $z_1 < z_0 < z_2$ . Let  $\lambda_1 = f(z_1)$  which is the value of  $Y = f(z)$  at the local minimum  $z_1$ . We also define  $\lambda_0 = f(z_0)$ , i.e., which is the value of  $Y = f(z)$  when  $z = z_0$ .

Concerning the equilibria of the dynamics, we have the following results:

If  $0 < \lambda < \lambda_1 = f(z_1)$ , the system has one type-1 equilibrium (with  $z = z_0$  and  $y = f(z_0) = \lambda$ ). This is an unstable focus. There is a single type-2 equilibrium satisfying  $\frac{1}{2} < z < 1$ , which is locally unstable.

At  $\lambda = \lambda_1$ , a saddle-node bifurcation takes place. For  $\lambda_1 < \lambda < \lambda_0$ , there is one type-1 equilibrium ( $z = z_0$ ), and three type-2 equilibria (with  $y = 0$ ). Among the type-2 equilibria, the one with smallest  $z$  is locally stable, but the other two are unstable.

At  $\lambda = \lambda_0$ , a bifurcation takes place and the type-1 equilibrium satisfying  $z = z_0$  is merged with an unstable boundary equilibrium (with  $z_1 < z < z_0$ ). However, this is not very important, because both of them are unstable.

Somewhere between  $\lambda_1$  and  $\lambda_0$ , there occurs a homoclinic bifurcation at  $\lambda = \lambda_{hc}$ . For  $\lambda_1 < \lambda < \lambda_{hc}$ , there is a stable limit cycle surrounding the unstable equilibrium. In contrast, for  $\lambda > \lambda_{hc}$ , there is no limit cycle and the trajectories converge to a type-2 equilibrium without infection. At  $\lambda = \lambda_{hc}$ , the stable limit cycle merges with the unstable type-2 equilibrium. The transition of the dynamics is called a homoclinic bifurcation. Fig. 2 illustrates these changes. The location of the homoclinic bifurcation is important, but it can only be identified numerically.

The amplitude of oscillation of the stable limit cycle for  $\lambda < \lambda_{hc}$  depends on  $\sigma$ , the relative speed of the two variables.  $\sigma$  is the magnitude of the speed of variable  $y$  relative to  $z$ . As  $\sigma$  becomes larger, the amplitude of the oscillation is larger, but this causes the homoclinic bifurcation to occur earlier.

#### ***B.4 When conformity factor is of a linear function***

In the model shown in Eq. (1), the conformity factor is  $1 + \gamma z^2$ , a quadratic function of  $z$ . This implies that the conformist tendency becomes increasingly stronger as  $z$  increases in an accelerating manner. If instead, the conformity factor is a linear function  $1 + \tilde{\gamma}z$ , the equilibrium with some infected is locally stable.

We start from a calculation as follows:

$$\frac{dz}{dt} = (1 - z)B(1 + \tilde{\gamma}z) - z(A_0 + A_1Y) \quad (\text{B.10})$$

We consider the dynamics by combining Eq. (1a) in the text and Eq. (B.10). Then, we

introduce  $y = \frac{A_1}{B\tilde{\gamma}}Y$ ,  $\sigma = \frac{B\tilde{\gamma}}{(b_1 - b_0)x_0}$ , and  $\lambda = \frac{A_0}{B\tilde{\gamma}}$ , which have a modified symbol  $\tilde{\gamma}$

instead of  $\gamma$ ; as well as  $\tau = (b_1 - b_0)x_0 t$  and  $z_0 = \frac{c - b_0 x_0}{(b_1 - b_0)x_0}$ . We then obtain Eq. (2)

in the text with  $f(z) = \frac{1-z}{z}\left(z + \frac{1}{\bar{\gamma}}\right)$ , which is different from that in the text. The

equilibria are obtained in the same way as in Appendix A, as far as adopting this

modified function  $f(z)$ . We have a type-1 equilibrium with some infection in the

population as  $z = z_0$  and  $y = f(z_0)$ . The equilibrium is locally stable if  $f'(z_0) < 0$ ,

and is unstable if  $f'(z_0) > 0$ . As  $f(z) = \frac{1-z}{z}\left(z + \frac{1}{\bar{\gamma}}\right)$  is a monotonically decreasing

function of  $z$  for  $0 < z < 1$ , it is always stable.

## Appendix C

### *Value of $z$ at equilibria for different $\lambda$*

Fig. C.1 illustrates the value of  $z$  at the equilibria. The horizontal axis is for  $\lambda$ . The different parts of the figure indicate cases with different values for  $z_0$ . A type-1 equilibrium ( $\hat{y} > 0$ ) appears as a horizontal line ( $z = z_0$ ). Stable and unstable type-1 equilibria are indicated by solid and dotted lines, respectively. Type-2 equilibria ( $\hat{y} = 0$ ) appear as a curve  $f(z) = \lambda$  on these figures. They may be stable or unstable, as shown by the solid portion and dotted portions of the curve, respectively. Fig. C.1(a) and C.1(c) are bifurcation diagrams, because the trajectories converge to one of the stable branches appearing as solid line or solid portion of a curve. However, Fig. C.1(b) cannot be regarded as a bifurcation diagram, because the trajectories may converge to a limit cycle. To avoid the complicated figure, we did not show the limit cycles on Fig. C.1(b).

In Fig. C.1(a), the type-1 equilibrium ( $\hat{y} > 0$ ) is locally stable because  $f'(z_0) < 0$  holds. In addition, there are type-2 equilibria which appear as points on the curve  $f(z) = \lambda$ , which may be stable or unstable. (n.b.  $\lambda_1 = f(z_1)$ ,  $\lambda_0 = f(z_0)$ , and  $\lambda_2 = f(z_2)$  are adopted in Fig. C.1(a).) For  $\lambda < f(z_1)$ , there is a single type-1 equilibrium, which is globally stable according to numerical analysis. When  $\lambda$  passes  $f(z_1)$ , a saddle-node bifurcation occurs, and a pair of stable and unstable type-2 equilibria appear ( $y = 0$ ). For  $f(z_1) < \lambda < f(z_0)$ , the dynamics are bistable, with two attractors: one at a type-1 equilibrium ( $y > 0$ ) and the other at a type-2 equilibrium ( $\hat{y} = 0$ ). As  $\lambda$  increases, the value of  $\hat{y}$  at the type-1 equilibrium decreases and  $\lambda = f(z_0)$  holds. Then, the type-1 equilibrium and unstable type-2 equilibrium merge. For  $\lambda$  larger than  $f(z_0)$ , there is no type-1 equilibrium with a positive  $\hat{y}$ . The type-2 equilibrium which



was unstable for  $\lambda < f(z_0)$  becomes a stable equilibrium for  $\lambda > f(z_0)$ . This transition is named a "transcritical bifurcation" or "alternation of stability" (Guckenheimer and Holmes 1983; Strogatz 1994) (see Appendix D and Fig. D.1). The dynamics are bistable (having two attractors) for  $f(z_0) < \lambda < f(z_2)$ . At  $\lambda = f(z_2)$ , the stable type-2 equilibrium merges with the unstable equilibrium and they disappear, indicating a saddle-node bifurcation. For  $\lambda > f(z_2)$ , the system only has a globally stable type-2 equilibrium.

Fig. C.1(b) show the case for  $z_1 < z_0 < z_2$ . The type-1 equilibrium is locally unstable. The dynamics may have a limit cycle or the trajectories may converge to a type-2 equilibrium. For  $\lambda < \lambda_{hc}$ , the type-1 equilibrium is surrounded by a stable limit cycle, as illustrated in Fig. 2(a). The limit cycle is not shown in Fig. C.1(b). At  $\lambda = \lambda_{hc}$ , a homoclinic bifurcation takes place (see Fig. 2(b)). For  $\lambda$  slightly larger than  $\lambda_{hc}$ , no limit cycle exists. As  $\lambda$  changes, the dynamics show various transitions with bifurcations of different types, which can be traced by numerical analyses.

In Fig. C.1(c) in which  $0 < z_0 < z_1$  holds, the type-1 equilibrium is stable for  $0 < \lambda < f(z_0)$ , because  $f'(z_0) < 0$  holds. At  $\lambda = f(z_0)$ , the dynamics exhibit transcritical bifurcation (see Appendix D). For  $\lambda > f(z_0)$ , the type 2 equilibrium becomes globally stable.

## Appendix D

Fig. D.1 illustrates the dynamics on the  $(z,y)$ -plane for different values of  $\lambda$ . The type-1 equilibrium is locally stable because  $z_0 > z_2$  holds. As  $\lambda$  increases, the stable equilibrium including some infected hosts (type 1) becomes merges with an unstable equilibrium without infection (type 2). When  $\lambda$  increases further, the type-1 equilibrium disappears, and a stable type-2 equilibrium without infection remains. This transition is called a "transcritical bifurcation" or an alternation of stability (Guckenheimer and Holmes, 1983; Strogatz 1994).

**Literature Cited**

- Arthur RF, Jones JH, Bonda MH, Ram Y, Feldman MW. 2021. Adaptive social contact rates induces complex dynamics during epidemics. *PLOS Computational Biology* 17, art 1008639 doi: 10.1371/journal.pcbi.1008639
- Bailey RM, Carrella, E, Axtell R, Burgess MG, Cabral RB, Drexler M, Dorsett C, Madsen JK, Merkl A, Saul S. 2019. A computational approach to managing coupled human–environmental systems: the POSEIDON model of ocean fisheries. *Sustainability Science* (2019) 14, 259–275. doi: 10.1007/s11625-018-0579-9
- Bauch CT. 2005. Imitation dynamics predict vaccinating behaviour. *Proc Biol Sci.* 2005 Aug 22;272(1573):1669-1675. doi: 10.1098/rspb.2005.3153.
- Capasso, V, Serio G. 1978. A Generalization of the Kermack-Mckendrick Deterministic Epidemic Model. *Mathematical Biosciences* 42, 41-61. doi: 10.1016/0025-5564(78)90006-8
- Dönges P, Wanger J, Contreras S, Iftekhar EM, Bauer S, Mohr SB, Dehning J, Valdez AC, Kretzschmar M, Maes M, Nagel K, Priesemann V. 2022. Interplay between risk perception, behavior, and COVID-19 spread. 2022, *Frontiers in Physics* 10 art 842180. doi: 10.3389/fphy.2022.842180
- d’Onofrio A, Manfredi P. 2009. Information-related changes in contact patterns may trigger oscillations in the endemic prevalence of infectious diseases. *Journal of Theoretical Biology* 256 (2009) 473–478. doi:10.1016/j.jtbi.2008.10.00
- d’Onofrio A, Manfredi P, Salinelli E. 2007. Vaccinating behaviour, information, and the dynamics of SIR vaccine preventable diseases. *Theoretical Population Biology* 71 (2007) 301–317. doi: 10.1016/j.tpb.2007.01.001

- Epstein JM, Parker J, Cummings D, Hammond RA. 2008. Coupled contagion dynamics of fear and disease: mathematical and computational explorations. *PLOS ONE* 12, e3955. doi: 10.1371/journal.pone.0003955
- Earn DJD, Rohani P, Bolker BM, Grenfell BT. 2000. A Simple model for complex dynamical transitions in epidemics. *Science* 287, 667-670. doi: 10.1126/science.287.5453.667.
- Fenichel EP, Castillo-Chavez C, Ceddia MG, Chowell G, Parra PAG, IHicking GJ, Holloway G, Horan R, Morin B, Perrings C, Springborn M, Valazquez L, Villalobos C. 2011. Adaptive human behavior in epidemiological models. *PNAS* 108, 6306-6311. doi: 10.1073/pnas.1011250108
- Folke C, Hahn T, Olsson P, Norberg J. 2005. Adaptive governance of social-ecological systems. *Annu. Rev. Environ. Resour.* 30, 441-473. doi: 10.1146/annurev.energy.30.050504.144511
- Fu F, Rosenbloom DI, Wang L, Nowak MA. 2010 Imitation dynamics of vaccination behavior on social networks *Proceedings Royal Soc. B* 278, 42-49. doi: 10.1098/rspb.2010.1107.
- Funk S, Salathe M, Jansen VA. 2010. Modelling the influence of human behaviour on the spread of infectious diseases: a review. *J Royal Soc Interface* 7, 1247-1256. doi: 10.1098/rsif.2010.0142
- Grenfell BT, Bjornstad ON, Finkenstadt BR. 2002. Dynamics of measles epidemics: scaling noise, determinism, and predictability with the TSIR model. *Ecological Monographs* 72, 2002, pp. 185–202. doi: 10.1890/0012-9615(2002)072.

- Grenfell BT, Pybus OG, Gog JR, Wood JL, Daly JM, Munford JA, Holmes EC, 2004. Unifying the Epidemiological and Evolutionary Dynamics of Pathogens. *Science* 303, 327-332z. doi: 10.1126/science.1090727.
- Ghosh I, Martcheva M. 2021. Modeling the effects of prosocial awareness on COVID-19 dynamics: case on colombia and India. *Nonlinear Dynamics* 104, 4681-4700. doi: 10.1007/s11071-021-06489-x
- Guckenheimer J, Holmes P. 1983. "Nonlinear oscillations, dynamical systems and bifurcations of vector fields." Springer-Nature pp. 462.
- Haraguchi Y, Sasaki A. 1997. Evolutionary pattern of intra-host pathogen antigenic drift: effect of cross-reactivity in immune response. *Philos. Trans. R. Soc. B* 352, 11-20. doi: 10.1098/rstb.1997.0002.
- Hayashi, R., Iwami, S., Iwasa, Y. 2022. Escaping stochastic extinction of mutant virus: temporal pattern of emergence of drug resistance within a host. *Journal of Theoretical Biology* 537, 111029. doi: 10.1016/j.jtbi.2022.111029
- Heggerud CM, Wang H, Lewis MA. 2022. Coupling the socio-economic and ecological dynamics of cyanobacteria: Single lake and network dynamics. *Ecological Economics* 194 107324. doi: 10.1016/j.ecolecon.2021.107324
- Iwasa Y, Pomiankowski A. 1995. Continual change in mate preferences. *Nature* 377, 420-422. doi: 10.1038/377420a0
- Iwasa Y, Uchida T, Yokomizo H. 2007. Nonlinear behavior of the socio-economic dynamics for lake water pollution control. *Ecological Economics* 63:219-229. doi: 10.1016/j.ecolecon.2006.11.003

- Iwasa Y, Suzuki-Ohno Y, Yokomizo H. 2010. Paradox of nutrient removal in coupled socio-economic and ecological dynamics for lake water pollution. *Theoretical Ecology* **3**:113-122. doi: 10.1007/s12080-009-0061-5
- Jentsch PC, Scab B, Anand M, Bauch CT. 2021. Prioritising COVID-19 vaccination in changing social and epidemiological landscapes: a mathematical modelling study. *Lancet Infectious Diseases* **21**(8) August 2021 Pages 1097-1106.
- Johnston MD, Pell B 2020. A dynamical framework for modeling fear of infection and frustration with social distancing in COVID-19 spread. *Mathematical Biosciences and engineering* **17**, 7892-7915. doi: 10.3934/mbe.2020401
- Kabir KMA, Risa T, Tanimoto, J. Prosocial behavior of wearing a mask during an epidemic: an evolutionary explanation. *Sci Rep* **11**, 12621 (2021). doi: 10.1038/s41598-021-92094-2
- Kamo M, Sasaki A. 2005. Evolution toward multi-year periodicity in epidemics. *Ecology Letters* **8**, 378-385. doi:10.1111/j.1461-0248.2005.00734.x
- Kermack WO, McKendrick AG. 1927. A contribution to the mathematical theory of epidemics. *Proc Royal Soc Lond A* **115**, 700-721. doi: 10.1098/rspa.1927.0118
- Lee J-H, Iwasa Y. 2011. Tourists and traditional divers in a common fishing ground. *Ecological Economics* **70**, 2350-2360. doi: 10.1016/j.ecolecon.2011.07.013
- Lee J-H, Iwasa Y. 2020. Ecotourism development and the heterogeneity of tourists. *Theoretical Ecology* **13**, 371-383. doi:10.1007/s12080-020-00458-7
- Lee, J-H, Sigmund K, Dieckmann U, Iwasa Y. 2015a. Games of corruption: how to suppress illegal logging *Journal of Theoretical Biology* **367**:1-13. doi: 10.1016/j.jtbi.2014.10.037

- Lee J-H, Kakinuma K, Okuro T, Iwasa Y. 2015b. Coupled social and ecological dynamics for herders in Mongolian rangeland. *Ecological Economics* 114, 208-217. doi: 10.1016/j.ecolecon.2015.03.003
- Lee, J-H, Jusup M, Iwasa Y. 2017. Games of corruption in controlling the overuse of common pool resources. *Journal of Theoretical Biology* 428:76-86. doi: 10.1016/j.jtbi.2017.06.001
- Lee, J-H, Kubo Y, Fujiwara T, Septiana RM, Riyanto S, Iwasa Y. 2018. Optimal management of state teak plantation with high risk of illegal logging: role of agroforestry and profit sharing. *Ecological Economics* 149:140-148.
- Lee, J-H, Iwasa Y, Dieckmann U, Sigmund K. 2019. Social learning leads to the persistent corruption. *Proceedings of the National Academy of Sciences* 116(27):13276-13281. doi: 10.1073/pnas.1900078116
- Liu W-M, Levin SA, Iwasa Y. 1986. Influence of nonlinear incidence transmission rates upon the behavior of SIRS epidemic models. *Journal of Mathematical Biology* 23,187-204. doi: 10.1007/BF00276956
- Liu W-M, Hethcote HW, Levin SA. 1987. Dynamical behavior of epidemiological models with nonlinear incidence rates. *Journal of Mathematical Biology* 25, 359– 380.
- Manuel-Navarrete D, Gomez JJ, Gallopin G. 2007. Syndromes of sustainability of development for assessing the vulnerability of coupled human–environmental systems. The case of hydrometeorological disasters in Central America and the Caribbean. *Global Environmental Change* 17, 207–217. doi: 10.1016/j.gloenvcha.2006.07.002

- Meloni S, Perra N, Arenos A, Gomez S, Moreno Y, Vespignoni A. 2011. Modeling human mobility responses to the large-scale spreading of infectious diseases. *Scientific Reports* 1, e62. doi: 10.1038/srep00062
- Murray JD. 1989. "Mathematical biology" Springer. Biomathematics series. vol. 19.
- Oraby T, Thampi V, Bauch CT. 2014. The influence of social norms on the dynamics of vaccinating behaviour for paediatric infectious diseases. *Proc Biol Sci.* 281(1780):20133172. doi: 10.1098/rspb.2013.3172.  
Erratum in: *Proc Biol Sci.* 2016 Oct 12;283(1840):
- Perra N, Balcan D, Gonsalves B, Vespignani 2011. Towards a characterization of behavior-disease models. *PLOS ONE* 6, e23084. doi: 10.1371/journal.pone.0023084
- Poletti P, Caprile B, Ajelli A, Pugliese A, Merler S. 2009. Spontaneous behavioural changes in response to epidemics. *Journal of Theoretical Biology* 260 (2009), 31–40. doi: 10.1016/j.jtbi.2009.04.029
- Poletti P, Ajelli M, Merler S. 2012. Risk perception and effectiveness of uncoordinated behavioral responses in an emerging epidemic. *Mathematical Biosciences* 238(2), 80-89. doi: 10.1016/j.mbs.2012.04.003
- Reluga T. 2010. Game theory of social distancing in response to an epidemic. *PLOS Comput Biol* 6, e1000793. doi: 10.1371/journal.pcbi.1000793
- Sasaki A, Haraguchi Y. 2000. Antigenic drift of viruses within a host: a finite site model with demographic stochasticity. *J. Math. Biol.* 51, 245-255. doi: 10.1007/s002390010086
- Sasaki A, Haraguchi Y, Yoshida H. 2012. Estimating the risk of re-emergence after stopping polio vaccination. *Front. Microbiol.* 3, art 178. doi: 10.3389/fmicb.2012.00178. doi: 10.3389/fmicb.2012.00178



- Satake S., Iwasa Y. 2006. Coupled ecological and social dynamics in a forested landscape: the deviation of the individual decisions from the social optimum. *Ecological Research* 21, 370-379. doi: 10.1007/s11284-006-0167-9
- Satake A, Janssen M, Levin SA, Iwasa Y. 2007. Synchronized deforestation induced by social learning under uncertainty of forest-use value *Ecological Economics* 63, 452-462. doi: 10.1016/j.ecolecon.2006.11.018
- Satake A, Leslie HM, Iwasa Y, Levin SA. 2007. Coupled ecological-social dynamics in a forested landscape: spatial interactions and information flow. *Journal of Theoretical Biology* 246, 695-707. doi: 10.1016/j.jtbi.2007.01.014
- Satake A, Levin SA, Iwasa Y. 2008. Comparison between perfect information and passive-adaptive social learning models of forest harvesting. *Theoretical Ecology* 1:189-197. doi: 10.1007/s12080-008-0019-z
- Strogatz SH. 1994. "Nonlinear dynamics and chaos." Perseus Books Publ., Boston. pp.498
- Sun RT, Hilker FM. 2020. Analyzing the mutual feedbacks between lake pollution and human behaviour in a mathematical social-ecological model. *Ecological Complexity* 43, 100834. doi: 10.1016/j.ecocom.2020.100834
- Suzuki Y, Iwasa Y. 2009a. The coupled dynamics of human socio-economic choice and lake water system: the interaction of two sources of nonlinearity. *Ecological Research* 24: 479-489. doi:10.1007/s11284-008-0548-3
- Suzuki Y, Iwasa Y. 2009b. Conflict between groups of players in coupled socio-economic and ecological dynamics. *Ecological Economics* 68, 1006-1115. doi:10.1016/j.ecolecon.2008.07.024

Tkachenko A, Maslow S, Wang T, Elbana A, Wong GN, Goldenfeld N. 2021

Stochastic social behavior coupled to COVID-19 dynamics leads to wanes ,  
plateaus, and an endemic state. *eLife* 10, e68341. doi: 10.7554/eLife.68341

Walker B, Holling CS, Carpenter SR, Kinzig A. 2004. Resilience, adaptability and

transformability in social-ecological systems. *Ecology and Society* 9, art 5.  
doi: 10.5751/ES-00650-090205

Wang Z, Andrews MA, Wu ZX, Wang L, Bauch CT. 2015. Coupled diseases-behavior

dynamics on complex networks: a review. *Physics of life reviews* 15, 1-29.  
doi: 10.1016/j.plrev.2015.07.006

**Figure captions**

Fig. 1 COVID-19 cases daily report of the number of infected patients in Japan from January 16, 2020 to December 31, 2021. The average over 7 days is shown. The cumulative number of infected by the end of this period was 1.37 % of the total population. This figure was made based on the data from <https://www3.nhk.or.jp/news/special/coronavirus/data/> downloaded on June 30, 2022.

Fig. 2 Disappearance of perpetual oscillations as parameter  $\lambda$  changes. Trajectories of the dynamics given in Eq. (2) are illustrated on  $(z,y)$ -plane. Solid and open circles are locally stable and unstable equilibria, respectively. Isoclines are shown in broken curves. (a) The dynamics have a stable limit cycle surrounding the equilibrium, indicating perpetual oscillation in the number of infected hosts  $y$ . The dynamics also have a locally stable equilibrium without infected exist ( $y = 0$ ).  $z_0 = 0.47$ , and  $\sigma = 1.76$ . (b) A trajectory started from the unstable equilibrium without infection leads to a single bout of explosive increase in infection and then returned to the same equilibrium. This is called a "homoclinic orbit."  $z_0 = 0.46$ , and  $\sigma = 1.72$ . (c) A stable equilibrium without infected hosts exists.  $z_0 = 0.44$ , and  $\sigma = 1.67$ . Other parameters are:  $\gamma = 150$ , and  $\lambda = 0.2$ .

Fig. 3 Homoclinic bifurcation. (a) Top and bottom parts illustrate the amplitudes of the oscillations of  $y$  and  $z$  in the limit cycle, respectively. The two curves represent the maximum (red) and minimum (black) values of the variables. The horizontal axis represents  $\lambda$ . (b) Time course of  $y$  for different  $\lambda$ . The horizontal axis represents time

$t$ . The top, middle, and bottom parts indicate the results for  $\lambda = 5 \times 10^{-5}$ ,  $\lambda = 0.1$ , and  $\lambda = 0.1275$ , respectively. The period of oscillation increases as  $\lambda$  approaches to the critical value  $\lambda = 0.1276$ , where the period becomes infinitely long. For  $\lambda > 0.1276$ , there is no limit cycle. Other parameters are:  $z_0 = 0.3$ ,  $\gamma = 200$ , and  $\sigma = 20$ .

Fig. 4 Hopf bifurcation. Top and bottom portions indicate values of  $y$  and  $z$ , respectively. The two curves indicate the maximum (black) and minimum (red) values on the stable limit cycle. (a) The horizontal axis is  $\gamma$ . A Hopf bifurcation occurs at approximately  $\gamma \approx 27.2$ . For  $\gamma < 27.2$ , the equilibrium is stable. For  $\gamma > 27.2$ , the system oscillates with the amplitude increasing with  $\gamma$ . The parameters are:  $z_0 = 0.3$ ,  $\lambda = 0.1275$ , and  $\sigma = 20$ . (b) The horizontal axis indicates a parameter  $z_0$ . (b) The horizontal axis is  $z_0$ . For  $z_0 < 0.08$  and  $z_0 > 0.49$ , the equilibrium  $z = z_0$  is stable. For  $0.08 < z_0 < 0.49$ , the equilibrium is unstable and there is a stable limit cycle. Parameters are:  $\gamma = 200$ ,  $\lambda = 5 \times 10^{-5}$ ,  $\sigma = 20$ .

Fig. 5 Shape of oscillation when  $\sigma$  is very large or very small. Top and bottom graphs are  $y(t)$  and  $z(t)$  in the limit cycle, respectively. The horizontal axis indicates time  $t$ . (a) The relative speed of the changes in  $z$  is much faster than that of the changes in  $y$ . The oscillation is a typical relaxation-oscillation.  $\sigma = 20$ . (b) The speed of  $z$  is slower than that of  $y$ . The amplitude of the limit cycle is larger and the period of oscillation is longer than in (a).  $\sigma = 2$ . Other parameters are:  $\gamma = 200$ ,  $z_0 = 0.3$ , and  $\lambda = 0.1$ .

Fig. 6 Phase diagram. The horizontal axis represents recovery rate  $c$ , and the vertical axis represents  $\lambda (= A_0/B\gamma)$ . (a) In the area labeled E, the infection will be eradicated eventually. In the area labeled G, the number of infected continues increasing. In the area labeled S, there is a stable equilibrium with some infected hosts. In the area labeled U, the equilibrium with some infected hosts exists but is unstable. In the last case, there can be either a stable limit cycle (perpetual oscillation), or the trajectory may oscillate a finite number of times and then converge to an equilibrium without infection (transient oscillation).  $\gamma = 200$ . (b) The region labeled as U disappears. The trajectories converge to the equilibrium with some infected hosts.  $\gamma = 10$ . Other parameters are:  $b_1x_0 = 20$ , and  $b_0x_0 = 10$ .

Fig. C.1 Value of  $z$  at equilibria for different parameter  $\lambda$ . Curves indicate  $\lambda = f(z)$ . The solid line and solid portions of the curve represent the stable equilibrium. The dotted line and dotted portions of the curve indicate the unstable equilibrium. (a)  $z_0 > 0.490$ . The equilibrium  $z = z_0$  for  $0 < \lambda < f(z_0)$  is stable. (b)  $0.077 < z_0 < 0.490$ . The equilibrium  $z = z_0$  for  $0 < \lambda < f(z_0)$  is an unstable focus. (c)  $z_0 < 0.077$ . The equilibrium  $z = z_0$  for  $0 < \lambda < f(z_0)$  is stable.  $z_1$  and  $z_2$  are the local minimum and the local maximum of  $f(z)$ .  $\lambda_1 = f(z_1)$ ,  $\lambda_2 = f(z_2)$ , and  $\lambda_0 = f(z_0)$ . Other parameter is  $\gamma = 200$ .

Fig. D.1 Transcritical bifurcation. The trajectories of the dynamics of Eq. (2) are shown on  $(z,y)$ -plane. Solid and open circles are locally stable and unstable equilibria, respectively. Isoclines are shown in broken curves. (a) The dynamics are bistable: a stable equilibrium with some infected and stable equilibrium without infected exist.  $z_0$

= 0.6. (b) A stable equilibrium and an unstable equilibrium exist, both without infected hosts.  $z_0 = 0.8$ . Other parameters are:  $\gamma = 150$ ,  $\lambda = 0.2$ , and  $\sigma = 1.5$ .

Highlights:

The infected of COVID-19 showed multiple distinct peaks in 2020 and 2021 in Japan.

We study a simple model for the coupling of social and epidemiological dynamics.

People switch between active and restrained states differing in the infection rate.

Transition rate to active state increases with the number currently active people.

Backward transition rate increases with the abundance of infected people.

The model showed a transient or sustained oscillation and various bifurcations.

Credit Author statement

Yoh Iwasa: Conceptualization, Methodology, Formal analysis, Visualization, Writing - original draft.

Rena Hayashi: Conceptualization, Methodology, Formal analysis, Visualization, Writing - original draft.