

Emergent dilemma and periodic oscillation in behavioral epidemics with nonlinear feedback

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Human behaviors, particularly nonpharmaceutical interventions (NPIs), are dynamically coupled with epidemic spreading. However, most models assume a linear relationship between perceived risk and infection rates, failing to capture the nonlinear nature of real-world behavioral responses. Here, we propose a behavioral epidemic model by incorporating nonlinear feedback and social influence, and analytically derive its critical thresholds. Notably, once the infection rate exceeds a critical threshold, the fraction of NPI adopters abruptly drops to zero, in contrast to classic findings in linear models. This paradoxical decline indicates a social dilemma: While abandoning NPIs is individually optimal at high infection rates, it collectively triggers an explosive epidemic surge. Furthermore, as the intensity of social influence increases, the system undergoes a Hopf bifurcation leading to periodic oscillations, which reflects a vicious cycle of recurrent epidemic waves. Finally, we confirm the robustness of this NPI-abandonment dilemma in networked populations. Our work illustrates rich emergent dynamics, challenging traditional perspectives on behavioral epidemiology.

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I. INTRODUCTION

Epidemic spreading remains a major threat to global public health, and its trajectory is crucially influenced by human social behaviors [1–5]. For instance, during the COVID-19 pandemic, nonpharmaceutical interventions (NPIs) effectively reduced disease prevalence and saved countless lives [6–8]. However, in the real world, individual compliance with such preventive measures is rarely guaranteed. Instead, it manifests as a dynamic process shaped by multiple factors, such as risk perception and social influence, which continuously co-evolves with epidemic dynamics itself [9–11]. Consequently, unraveling the complex interplay between behavioral responses and epidemic spreading is critical for controlling infectious diseases.

Extensive research has explored the coevolution of epidemic and behaviors, yielding important insights such as the

dilemma of vaccination uptakes and the emergence of multiple thresholds [12–17]. In particular, the COVID-19 pandemic demonstrates that NPIs may serve as the only effective countermeasures during early stages of newly emerging infectious diseases, thereby garnering increasing attention [18–20]. A key characteristic of NPIs, such as mask wearing and social distancing, is their transient nature: They offer protection only during active compliance, with their efficacy ending immediately upon cessation [21,22]. This implies that the dynamics of NPI usage and epidemic transmission interact on a comparable timescale [23]. To model their interplay, researchers typically utilize contagion frameworks and evolutionary game theory [24,25]. Specifically, the latter frames NPI adoption as a strategic decision where individuals weigh socioeconomic costs of compliance against the benefits of mitigating infection risks [26,27]. These coupled models have revealed nontrivial phenomenon absent in isolated epidemic dynamics. For instance, there emerges the oscillating tragedy where multiple infection peaks appear [28–31]. Moreover, a higher infection rate counterintuitively leads to a smaller final epidemic size, as it elevates public risk perception, driving broader NPI adoption [32–34]. This insight suggests that amplifying perceived risk of being infected, through information dissemination, is beneficial for epidemic control.

More broadly, a related line of research has studied evolutionary games with environmental feedbacks, where population strategies reshape an environmental state that in

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turn modifies the payoff structure [35,36]. Such feedbacks and related frameworks involving game transitions and noisy environments can generate rich dynamical patterns including oscillatory dynamics [37–40]. Epidemic-behavior system is related to them but differs in mechanisms and interpretation. Specifically, the feedback variable is not a generic environmental state but epidemic prevalence itself, which is shaped by protective behavior and in turn determines incentives to adopt protection.

Despite recent advances in modeling such feedback, most existing works assume that perceived infection risk scales linearly with epidemic infection rate, overlooking the inherently nonlinear nature of risk perception that drives behavioral evolution. Furthermore, individual behavior is not driven by risk assessment alone. External social influences, including social norms and peer pressure, serve as fundamental drivers of behavioral evolution [32,41–44]. Yet, how the integration of nonlinear feedback and social influences drives macroscopic emergent phenomena remains an unresolved challenge.

In this work, we propose a behavioral epidemic model that incorporates both nonlinear risk perception and social influences, and theoretically analyze critical behaviors. The results demonstrate that the nonlinearity induces rich emergent phenomena. Specifically, as the infection rate increases, NPI adoption initially rises but then abruptly drops to zero. The decline highlights a social dilemma: At high infection rates, abandoning NPIs is individually optimal but collectively exacerbates the epidemic spreading. This is absent in linear models. In particular, socially induced overestimation of infection rate may facilitate the emergence of the dilemma. Furthermore, as the intensity of social influence increases, the system behavior can transit from a stationary prevalence to periodic oscillations. Moreover, we show that the dilemma robustly occurs on structured populations.

II. BEHAVIORAL EPIDEMIC MODEL

We consider the joint dynamics of disease spreading and behavioral evolution, as shown in Fig. 1. The corresponding parameters are summarized in Table I. The disease dynamics are based on susceptible-infected-susceptible (SIS) model. Specifically, infected individuals transmit the disease to unprotected susceptible ones at a rate of β , whereas this rate is reduced to $(1 - \alpha)\beta$ if susceptible individuals adopt NPIs. Here, α measures the effectiveness of NPIs: $\alpha = 1$ implies perfect protection, while $0 < \alpha < 1$ corresponds to more realistic imperfect protection. Moreover, infected individuals become susceptible again at a rate μ , and simultaneously revert to their preinfection behavioral status due to inertia.

Regarding behavioral dynamics, we assume that susceptible individuals face a binary strategic choice: whether or not to adopt NPIs. We model this decision-making process using an evolutionary game framework, where individuals weigh the payoffs associated with these two choices. Specifically, the total payoff consists of the following three components. First, adopting NPIs incurs personal discomfort and economic losses, denoted by C_0 . Second, individuals assign a perceived cost C_I to potential infection, which captures health and social

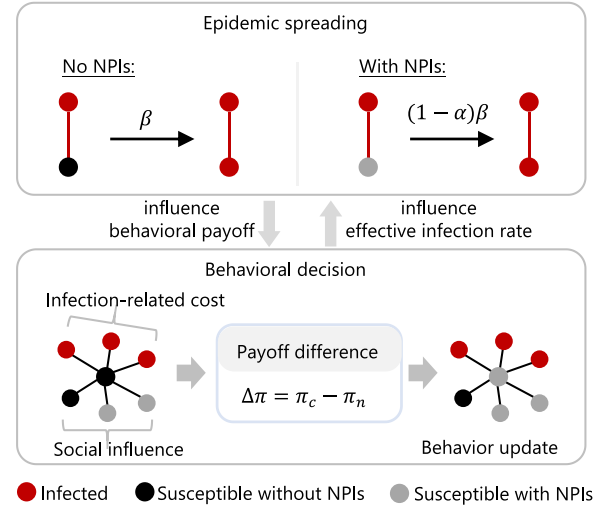


FIG. 1. Schematic illustration of behavioral epidemic dynamics. The epidemic process and behavioral decision interact through a bidirectional feedback loop. Disease prevalence affects individuals' perceived infection risk and thus behavioral payoff, thereby influencing their decisions to adopt NPIs. In turn, behavioral responses feed back into epidemic spreading by altering the effective infection rate.

burdens that individuals expect to suffer if infection occurs [28]. This cost is therefore an *ex ante* perceived loss considered during decision making, rather than an *ex post* realized cost incurred only after becoming infected. Inspired by previous works [23,28], we estimate the infection probability over a unit decision interval by converting instantaneous rate to a probability under Poisson contact process assumption. Specifically, let $I(t)$ denote the density of infected individuals at time t . For NPI nonadopters, the instantaneous infection hazard is given by

$$\lambda_n(t) = \beta k I(t), \quad (1)$$

where k represents the expected number of contacts and β is the infection rate. We divide the unit decision interval $[t, t + 1)$ into m subintervals of length $\Delta t = 1/m$. Assuming that $\lambda_n(t)$ remains approximately constant over this interval, the probability of avoiding infection in one subinterval is approximately $1 - \lambda_n(t)\Delta t$. Thus, the probability of avoiding

TABLE I. Descriptions for model parameters.

Parameters	Descriptions
β	Infection rate
μ	Recovery rate
α	Effectiveness of NPIs
γ	Perception error that rescales objective infection rate in decision process
C_0	Economic cost of adopting NPIs
C_I	Perceived cost associated with potential infection
η	Relative weight of social influence
ω	Parameter adjusting the relative timescale

infection over the whole interval can be approximated by

$$\lim_{m \rightarrow \infty} \left(1 - \frac{\lambda_n(t)}{m}\right)^m = e^{-\lambda_n(t)}. \quad (2)$$

Therefore, the probability of being infected during $[t, t + 1)$ is

$$x_n = 1 - e^{-\lambda_n(t)} = 1 - e^{-\beta k I(t)}. \quad (3)$$

However, the real infection rate may not be accurately perceived by individuals. Their perception is often influenced by social factors such as media exposure and public information [3,45]. Thus, we further incorporate socially induced perception error through a parameter γ , which rescales the objective infection hazard before it is converted into a perceived probability. This yields the perceived infection probability:

$$y_n = 1 - e^{-\gamma \beta k I(t)}. \quad (4)$$

Here, γ characterizes the direction and magnitude of socially induced perception bias. Specifically, $\gamma > 1$ means socially induced overestimation, while $\gamma < 1$ implies an underestimation. Moreover, in the low-risk regime where $\gamma \beta k I(t) \ll 1$, a first-order Taylor expansion gives $y_n \approx \gamma \beta k I(t)$, indicating that the nonlinear perception reduces approximately to the linear perception model (see Sec. X of the Supplemental Material [46]).

Thus, the perceived expected infection-related cost, denoted by $\pi_{n,I}$, is

$$\pi_{n,I} = -C_I y_n = -C_I (1 - e^{-\gamma \beta k I(t)}). \quad (5)$$

Similarly, for susceptible individuals who adopt NPIs, the perceived expected cost associate with infection can be written as

$$\pi_{c,I} = -C_I (1 - e^{-(1-\alpha)\gamma \beta k I(t)}). \quad (6)$$

Third, we incorporate social influence and quantify its payoff based on the principle that it is positively associated with the fraction of individuals adopting similar behaviors. Without loss of generality, we use the simplest functional form: The social payoff for NPI compliance is given by $\pi_{c,s} = \eta(p(t) - (1 - p(t)))$, while the social payoff for noncompliance is $\pi_{n,s} = \eta((1 - p(t)) - p(t))$. Here, $p(t)$ denotes the fraction of NPI compliance in the susceptible population, and η measures the relative weight of social influence.

Therefore, the total payoffs of NPI compliance and noncompliance can be, respectively, written as

$$\begin{aligned} \pi_c &= -C_0 - C_I (1 - e^{-(1-\alpha)\gamma \beta k I(t)}) + \eta(2p(t) - 1), \\ \pi_n &= -C_I (1 - e^{-\gamma \beta k I(t)}) + \eta(1 - 2p(t)). \end{aligned} \quad (7)$$

Finally, using the contagion framework and replicator dynamics, we derive the governing equations of this behavioral epidemic model:

$$\begin{aligned} \frac{dI}{dt} &= -\mu I + \beta k (1 - \alpha p) I (1 - I), \\ \frac{dp}{dt} &= \omega p(1 - p) \{-C_0 - C_I (e^{-\gamma \beta k I} - e^{-(1-\alpha)\gamma \beta k I}) \\ &\quad + 2\eta(2p - 1)\}, \end{aligned} \quad (8)$$

where ω adjusts the relative timescale of disease spreading and behavioral evolution. Moreover, considering that

epidemiological parameters are difficult for individuals to estimate, we also analyze a formulation of the perceived infection probability, where β and k are removed from the exponential term as a robustness check (see Sec. IX of the Supplemental Material [46]).

III. RESULTS

A. Emergent dilemma and bistability

We perform both theoretical analysis and model simulations in well-mixed populations (see details in Sec. I of the Supplemental Material [46]). First, we examine the dynamics driven solely by nonlinear feedback in the absence of social influence ($\eta = 0$). Figure 2(a) shows the complex changes in epidemic prevalence and NPI compliance level, exhibiting multiple phase transitions. Specifically, as infection rate β grows, NPI compliance would increase, thereby reducing epidemic prevalence, which confirms the classic finding from prior works [33,34]. Yet, as β grows further beyond a critical value, the fraction of NPI compliance surprisingly abruptly drops to zero, thereby triggering an explosive increase in epidemic prevalence. This finding challenges conventional wisdom that greater infection risks enhances the willingness to adopt NPIs. More critically, this finding unveils an emergent dilemma: When β is high, abandoning NPIs is individually optimal, while it collectively exacerbates epidemic prevalence across the population.

This counterintuitive phenomenon cannot arise from linear couplings (see Sec. X of the Supplemental Material [46]), but can be naturally explained by the nonlinear risk perception mechanism characterized by Eqs. (4)–(6). This nonlinearity induces nonmonotonic changes in payoff difference between NPI compliance and noncompliance (see Fig. S1 in the Supplemental Material [46]). Specifically, once the fraction of infected individuals exceeds a value, the payoff of NPI compliance falls below that of noncompliance. This drives individuals to abandon NPIs, thereby leading to higher infection prevalence, which in turn enlarge the relative advantage of noncompliance. Such a vicious feedback loop induces the explosive decrease in NPI compliance.

Moreover, our analysis reveals that the threshold for the dilemma is sensitive to initial conditions (see Fig. S2 in the Supplemental Material [46]). Specifically, a higher initial density of NPI compliance can postpone this critical threshold. This finding gives rise to another intriguing phenomenon, i.e., a bistable regime, where final state could be effectively controlled by modulating initial conditions.

To give a theoretical insight, we analytically derive threshold formulas for these phase transitions, through exploring stability of different equilibria (I^* , p^*) in Eq. (8) (see Sec. III of the Supplemental Material [46]). Specifically, $\beta_1^c = \mu/k$ represents the epidemic invasion threshold, derived from the stability condition of the disease-free equilibrium (0, 0). Furthermore, through analyzing the equilibrium $(1 - \mu/\beta k, 0)$, we obtain critical conditions for both the emergence and the abrupt disappearance of NPI compliance. These critical values, indicated by β_2^c and β_4^c in Fig. 2(a), are the two roots of the following equation:

$$e^{-(1-\alpha)\gamma(\beta k - \mu)} - e^{-\gamma(\beta k - \mu)} = \frac{2\eta + C_0}{C_I}. \quad (9)$$

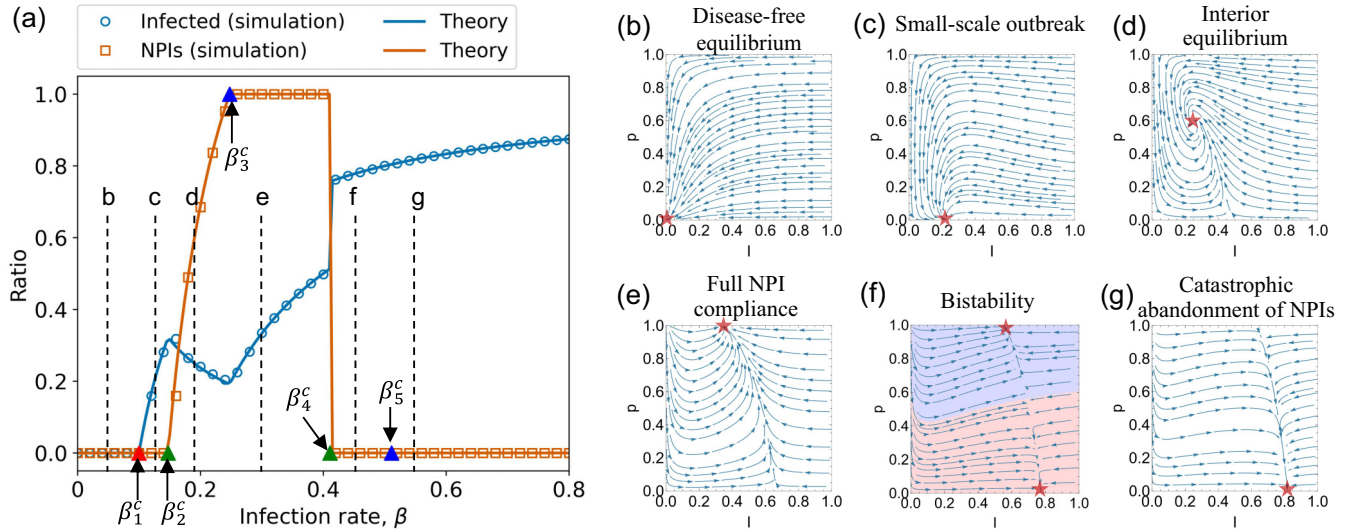


FIG. 2. Catastrophic abandonment of NPIs and bistability. (a) We present the fraction of infected individuals and NPI compliance as a function of β . They exhibit complex changes with multiple phase transitions. An intriguing transition is the collective abandonment of NPIs, which induces explosive growth in epidemic prevalence. The triangles represent the thresholds β_2^c – β_5^c , which are solved by Eqs. (9) and (12). (b)–(g) Vector fields are shown under six representative β . The red stars denote stable equilibria. The cases correspond to six distinct phases: disease-free equilibrium, small-scale outbreak without NPI compliance, stable interior equilibrium, full NPI compliance, bistability, and catastrophic abandonment of NPIs. In the bistability phase, two locally stable equilibria coexist, and the shaded regions in different colors denote their corresponding basins of attraction. Parameters: $N = 2000$, $k = 10$, $\alpha = 0.5$, $\mu = 1$, $\gamma = 1$, $C_0 = 1$, $C_I = 6$, $\eta = 0$, and $\omega = 1$.

The other nontrivial equilibrium is the interior equilibrium (I^*, p^*) , which exists if

$$\begin{aligned} I^* &= 1 - \frac{\mu}{\beta k(1 - \alpha p^*)}, \\ p^* &= \frac{1}{2} + \frac{C_0 + C_I(e^{-\gamma\beta k I^*} - e^{-(1-\alpha)\gamma\beta k I^*})}{4\eta}, \\ 0 &< I^* < 1, \\ 0 &< p^* < 1. \end{aligned} \quad (10)$$

The stability boundary of the interior equilibrium is

$$\begin{aligned} -\beta k(1 - \alpha p^*)I^* + 4\omega\eta p^*(1 - p^*) &< 0 \\ -4\eta(1 - \alpha p^*) - \alpha C_I\gamma\beta k(1 - I^*)\mathcal{E}(I^*) &> 0, \end{aligned} \quad (11)$$

where $\mathcal{E}(I^*) = (1 - \alpha)e^{-(1-\alpha)\beta k\gamma I^*} - e^{-\beta k\gamma I^*}$. The detailed derivation is provided in Sec. III of the Supplemental Material [46].

Then, we analyze the case under large initial density of NPI compliance, which is associated with the equilibrium $(1 - \mu/(1 - \alpha)\beta k, 1)$. Its stability condition corresponds to the lower and upper thresholds for full NPI compliance, indicated by β_3^c and β_5^c in Fig. 2(a), which can be computed by

$$e^{-(1-\alpha)\gamma(\beta k - \frac{\mu}{1-\alpha})} - e^{-\gamma(\beta k - \frac{\mu}{1-\alpha})} = \frac{C_0 - 2\eta}{C_I}. \quad (12)$$

The region enclosed by β_4^c and β_5^c constitutes the bistable regime. Figure 2(a) demonstrates that our theory accurately predicts the final prevalence and critical behaviors observed in large-scale simulations.

These critical thresholds divide the system into six phases, with their representative vector fields presented in Figs. 2(b)–2(g). Notably, Fig. 2(f) shows critical initial conditions within

the bistable regime, which determine whether the entire susceptible population adopts or abandons NPIs. Figure 2(g) illustrates the collective abandonment of NPIs at large β .

We further examine the conditions for the emergent dilemma, i.e., catastrophic abandonment of NPIs. For a small initial fraction of NPI compliance, we note that this phenomenon occurs if and only if Eq. (9) yields two solutions for β . When $\alpha = 1$, the equation has at most one solution for β , demonstrating that perfect protection could not induce the catastrophic abandonment of NPIs. When $0 < \alpha < 1$, by comparing the maximum of the left-hand side with the right-hand side of Eq. (9), we analytically derive the condition for the existence of two solutions, which is

$$C_I > \frac{(1 - \alpha)(2\eta + C_0)}{\alpha} e^{-\frac{\ln(1-\alpha)}{\alpha}}. \quad (13)$$

Thus, Eq. (13) serves as the necessary condition for the dilemma to emerge. This condition is represented by the critical line in Fig. 3(a). More specifically, Figs. 3(b) and 3(c) present phase diagrams illustrating the fraction of NPI compliance under joint effect of C_I and β as well as joint effect of α and β , where the white lines represent the thresholds. Phase diagrams of epidemic prevalence are shown in Sec. V of the Supplemental Material [46]. As illustrated by solid lines in Figs. 3(b) and 3(c), the results validate the predictive accuracy of Eq. (13) and demonstrate that the emergent dilemma exists within a wide range of parameters. Furthermore, we find that the threshold for NPI abandonment increases with C_I and α .

In Fig. 4, we explore the perception error regarding the infection rate, characterized by γ , which is sensitive to social factors including information dissemination and public awareness campaigns. For instance, intensive media coverage may lead individuals to overestimate the infection rate, which is captured by $\gamma > 1$. Figures 4(a) and 4(b) demonstrate that

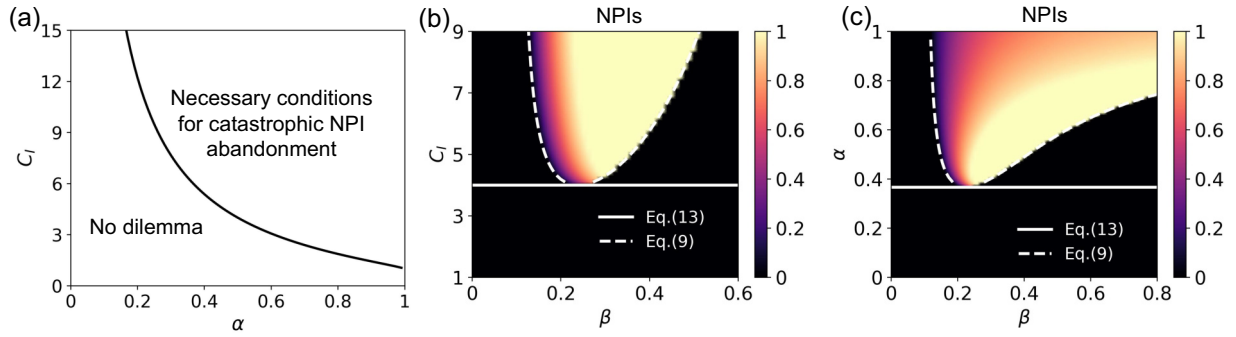


FIG. 3. Critical conditions for the catastrophic NPI abandonment. (a) The black line is computed by Eq. (13), which represents the necessary conditions for the dilemma to emerge. Phase diagrams illustrating the ratio of NPI compliance are shown under (b) $\alpha = 0.5$ and (c) $C_I = 6$. The solid line is obtained by Eq. (13) and represents the necessary condition for abrupt NPI abandonment, while the dashed lines, solved by Eq. (9), represent the thresholds for NPI adoption and abrupt NPI abandonment. The results reveal that the dilemma about catastrophic NPI abandonment stably exists in a wide parameter space. Parameters: $N = 2000$, $k = 10$, $\mu = 1$, $\gamma = 1$, $C_0 = 1$, $\eta = 0$, and $\omega = 1$.

socially induced overperception exerts a twofold impact. On the positive side, it reduces the emergence threshold for NPI compliance, thereby inhibiting epidemic spreading. Conversely, it lowers the threshold for NPI abandonment, which means that the explosive growth of the disease occurs at smaller β . Moreover, Fig. 4(c) shows that γ has a nonmonotonic impact on system outcomes. Moderate value of γ enhances NPI compliance and reduces epidemic size. However, once γ exceeds a critical value γ_c , collective NPI abandonment emerges, leading to explosive growth of diseases. This critical value is determined by the larger positive root of Eq. (9). Equivalently, with $R_0 = \beta k / \mu$, γ_c corresponds to the larger root of the following equation:

$$e^{-(1-\alpha)\gamma\mu(R_0-1)} - e^{-\gamma\mu(R_0-1)} = \frac{2\eta + C_0}{C_I}. \quad (14)$$

In addition, γ_c decreases with β [see Fig. 4(a)]. These findings imply that overly aggressive campaigns are not universally beneficial and can even be counterproductive, particularly when the actual infection rate is high.

B. Periodic oscillations upon incorporating social influence

We extend our analysis by incorporating social influence, where its relative weight is denoted by η . Figure 5(a) shows β - η phase diagram, where region colors denote steady states and lines mark boundaries solved by Eqs. (9)–(12). Here, the right branch of the dashed line separates the green full-compliance region from the purple bistable region, thereby representing β threshold for abrupt NPI abandonment under low initial NPI adoption. This threshold decreases as η increases. Conversely, the right dash-dotted curve gives the corresponding threshold under high initial NPI adoption, showing that this critical β increases with η . These results suggest that the social dilemma of NPI abandonment persists after social influence is incorporated, whereas the abandonment threshold shifts in opposite directions depending on the initial level of NPI adoption.

Crucially, when η is moderate, we note a gray region where all equilibria are unstable. By the Poincaré-Bendixson theorem, this implies the existence of at least one stable limit cycle in this region. The dark red line, computed from Eqs. (10) and (11), marks the boundary of this region and corresponds to the transition of the interior equilibrium from stable to unstable.

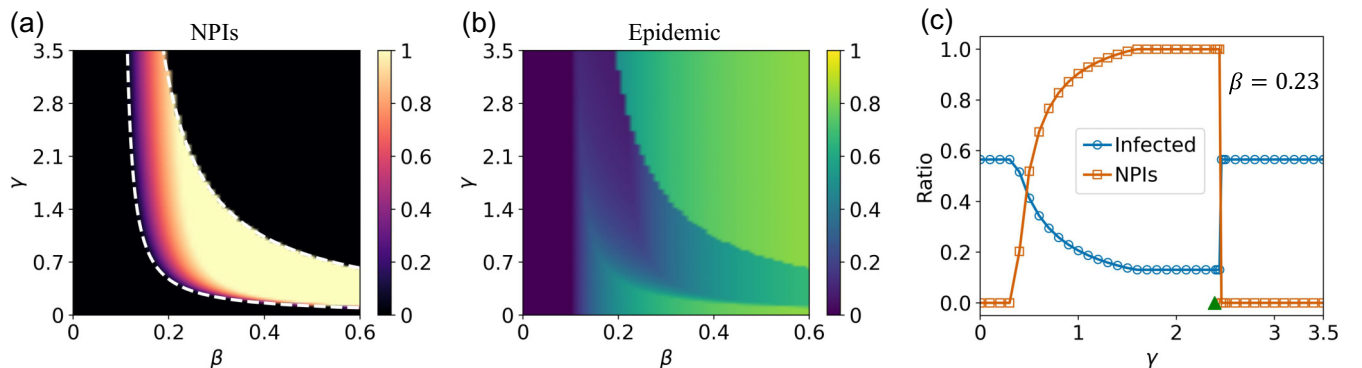


FIG. 4. Impact of socially induced perception error (γ). Phase diagrams about (a) ratio of NPI compliance and (b) ratio of infected individuals are shown under the joint effect of β and γ . The white lines, numerically solved by Eq. (9), depict critical thresholds. Results illustrate that γ has a twofold impact on NPI compliance and disease spreading. (c) The final state is plotted against γ for a representative value of $\beta = 0.23$. As γ increases, epidemic prevalence first decreases and then explosively grows once γ exceeds a critical value γ_c , marked by the green triangle and computed from Eq. (14). Parameters: $N = 2000$, $k = 10$, $\alpha = 0.5$, $\mu = 1$, $C_0 = 1$, $C_I = 6$, $\eta = 0$, and $\omega = 1$.

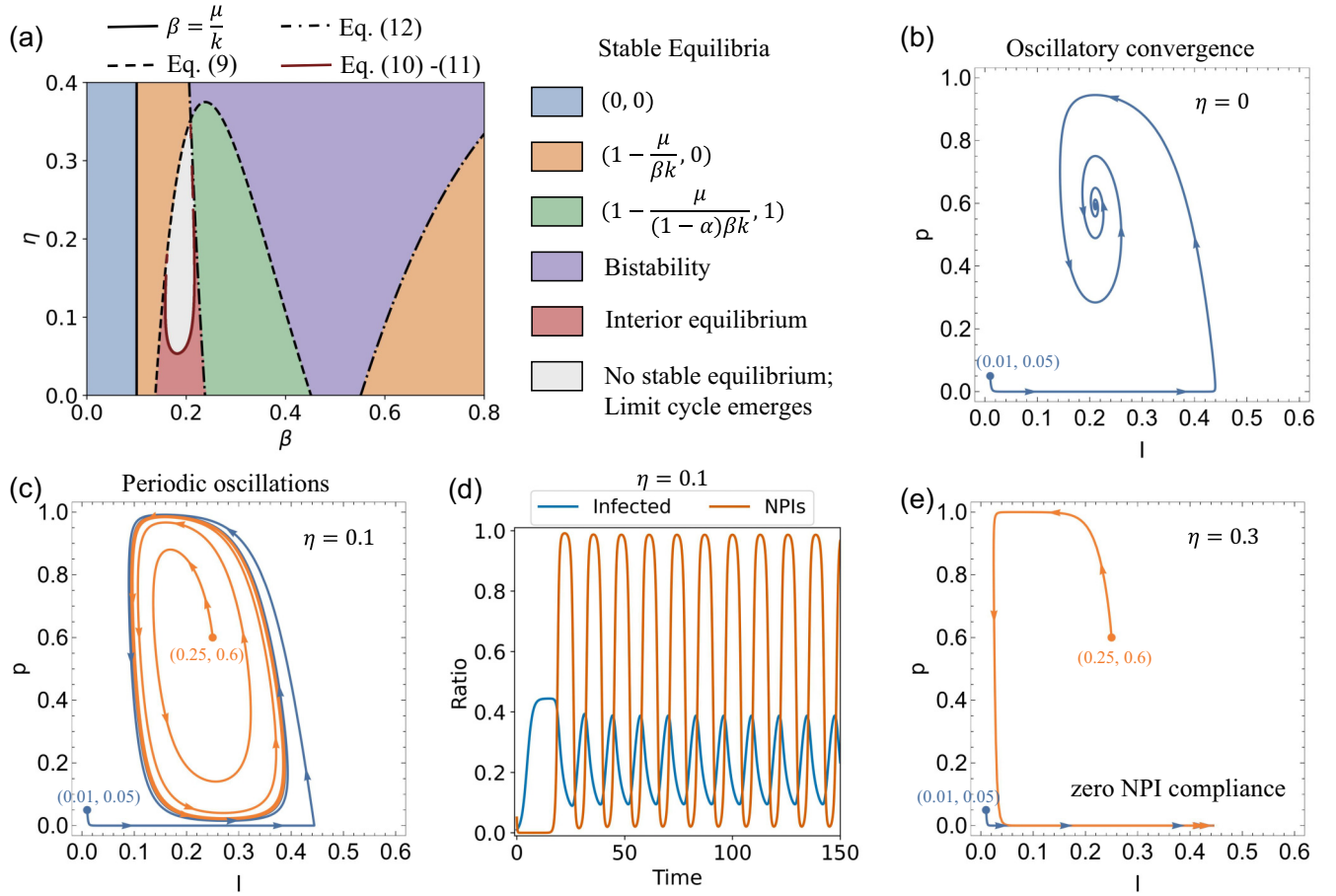


FIG. 5. How incorporating social influence affects the dynamics. (a) β - η phase diagram. Regions with different colors corresponds to different stable equilibria, which is derived by evaluating the eigenvalues of Jacobian matrix. The boundaries are solved by Eqs. (9)–(12). Notably, at moderate η , there is a gray region, where no stable equilibrium exists but a limit cycle emerges. (b)–(e) An intuitive case ($\beta = 0.18$) showing the impact of η . The trajectories are plotted for (b) $\eta = 0$ and (c) $\eta = 0.1$. Each color corresponds to a distinct initial condition. At $\eta = 0.1$, a limit cycle occurs, indicating periodic oscillations. The corresponding temporal evolution is shown in panel (d). (e) When $\eta = 0.3$, the trajectories again converge to a stable equilibrium. Parameters: $k = 10$, $\alpha = 0.5$, $\mu = 1$, $\gamma = 1$, $C_0 = 1$, $C_I = 7$, and $\omega = 5$.

This transition indicates a Hopf bifurcation. We then demonstrate that the limit cycle cannot be induced solely by social influence (see Sec. VI of the Supplemental Material [46]), but rather arises from the coupling between social influence and risk perception. Figures 5(b)–5(e) show intuitive evolution of trajectories under different η . For $\eta = 0$ [see Fig. 5(b)], the temporal evolution of the system initially undergoes oscillations and eventually reaches an interior equilibrium. This corresponds to a steady state where a fraction of individuals complies with NPIs. A marginal increase in η enhances NPI compliance level, reducing disease prevalence (see Fig. S4 in the Supplemental Material [46]). Notably, as η continues to increase, the system undergoes a Hopf bifurcation [47], where its final state shifts from a stable equilibrium to a limit cycle. Figure 5(c) presents a representative case of $\eta = 0.1$, where the trajectories originating from distinct initial conditions converge to a limit cycle. This corresponds to periodic oscillations in both NPI compliance and epidemic prevalence, as shown in Fig. 5(d). This phenomenon implies a vicious cycle in public health, where the population periodically experiences infection peaks.

Furthermore, we find that the period of the cycle increases rapidly as η grows (see Fig. S4 in the Supplemental Material [46]). This period expansion is because the limit cycle moves closer to the saddle equilibrium $(1 - \mu/\beta k, 0)$, where the trajectory experiences a drastically reduced evolutionary speed. This also implies a prolonged infection peak within each cycle. However, as η grows further beyond the threshold derived by Eq. (9), the equilibrium $(1 - \mu/\beta k, 0)$ becomes stable and the limit cycle vanishes [see Fig. 5(e)]. In this situation, the population faces a worst-case scenario where no one complies with NPIs, thereby leading to a widespread disease outbreak.

In addition, we demonstrate that a larger ω expands the parameter region associated with periodic oscillations, while narrowing the region corresponding to a stable interior equilibrium (see Sec. VIII of the Supplemental Material [46]).

C. Results on networked populations

We further consider network structure of populations and perform model simulations by using Gillespie algorithm (see

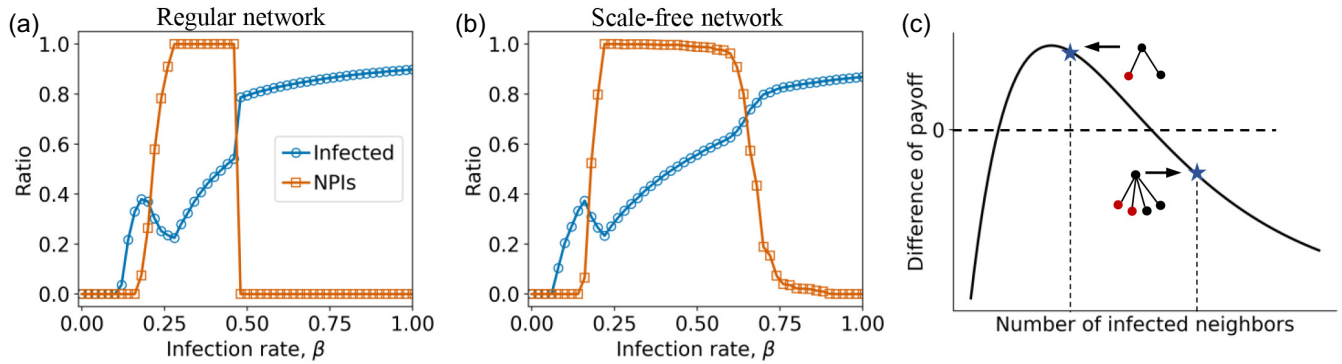


FIG. 6. Results in networked populations. We present simulation results on (a) a regular network and (b) a heterogeneous scale-free network generated by Barabási-Albert algorithm [48]. Both show that NPI compliance level exhibits a nonmonotonic trend as β grows. The main discrepancy is that the decline occurs more gradually on the scale-free network. (c) An intuitive explanation for this discrepancy. The payoff difference between abandoning and adopting NPIs is plotted against the number of infected neighbors. As indicated by the star markers, the payoff differences for a two-degree node and a four-degree node exhibit opposite signs, prompting the nodes to adopt divergent strategies. Parameters: $N = 2000$, $\langle k \rangle = 10$, $\alpha = 0.5$, $\mu = 1$, $\gamma = 1$, $C_0 = 1$, $C_I = 6$, $\omega = 1$, and $\eta = 0$.

Sec. I of the Supplemental Material [46]). Figure 6(a) shows the case on a regular network where each node has the same degree. Similarly, as the infection rate β grows, the NPI compliance level initially increases but then abruptly drops to zero. This indicates that the catastrophic NPI abandonment, i.e., the emergent dilemma, persists on regular networks. Figure 6(b) shows the results on heterogeneous networks. We find that the pattern of an initial rise followed by a decline in NPI compliance remains valid. In contrast, this decline is more gradual than that on regular networks. This is because network heterogeneity induces a divergence in optimal strategies of individuals. Specifically, for a given infection prevalence, abandoning NPIs yields a higher payoff for higher-degree nodes, while maintaining NPIs is more advantageous for lower-degree nodes, as indicated by stars in Fig. 6(c).

IV. CONCLUSION

Human behavioral evolution and epidemic spreading are deeply intertwined in the real world. However, unraveling this interplay and its ultimate impact remains a challenge, largely due to the inherent complexity introduced by nonlinear feedbacks and social influences. This work proposes a behavioral epidemic model by incorporating these multifaceted mechanisms and theoretically analyzes the critical thresholds. Our nonlinear perception mechanism is related to, but distinct from, complex contagion: both depend nonlinearly on the accumulation of relevant exposures. However, complex contagion typically exhibits social reinforcement through multiple exposures [49], whereas our model introduces nonlinearity through the transformation from epidemic prevalence to perceived infection risk, thereby influencing behavioral adoption. In contrast to amplification characteristic of complex contagion, the prevalence-driven feedback in our framework can influence behavioral adoption in a nonmonotonic manner. Crucially, we demonstrate that these mechanisms give rise to rich emergent phenomena.

First, as the infection rate increases, NPI compliance initially rises, effectively suppressing epidemic prevalence, which reproduces classic findings [33,34]. However, our

model reveals a fundamentally distinct phenomenon: Once the infection rate surpasses a critical threshold, NPI compliance undergoes an abrupt collapse to zero, triggering an explosive surge in infections. This abrupt decline indicates a social dilemma: Abandoning NPIs becomes individually optimal at high infection rates, yet this behavior is collectively detrimental. We further confirm that this dilemma persists within networked populations. Furthermore, the threshold for this decline is sensitive to initial conditions, revealing the emergence of bistability.

Moreover, we investigate the impact of risk perception error driven by social factors, such as information diffusion. Notably, while socially induced overestimation promotes NPI compliance and suppresses epidemic prevalence at low actual infection rates, it paradoxically produces the opposite impact at high rates. This indicates that communicating high infection rates through social campaigns is not always effective.

Finally, we uncover how social influences drive complex oscillatory dynamics. In absence of social influence, the system exhibits oscillatory convergence, successfully reproducing the classic “oscillatory tragedy” [28,29]. Differently, our analysis reveals that as social influence intensifies, the system undergoes a Hopf bifurcation and forms a limit cycle. This transition shifts the dynamics into sustained periodic oscillations, trapping the population in a vicious cycle marked by endless epidemic waves.

Our findings complement previous studies showing that game-environment feedback can generate oscillatory dynamics [35–40], by demonstrating that periodic oscillations can arise from a distinct epidemic-behavior feedback. More critically, such feedback produces a distinct social dilemma: Nonlinear infection risk perception can trigger an abrupt collapse of NPI compliance at high infection rates, making NPI abandonment individually favorable but collectively harmful.

Our work reveals how nonlinear feedback and social influence fundamentally reshape epidemic trajectories, transcending the conventional insights of behavioral epidemic dynamics. Despite that, several limitations remain that warrant further investigation. First, while our current analysis focuses on the classic SIS model, the framework and

coevolutionary mechanisms can be extended to other epidemic models [50]. Second, for analytical tractability, our model uses an aggregate behavioral variable $p(t)$ to capture population-level NPI compliance. This treatment is consistent with previous studies [23,32]. A natural extension would be to distinguish four states, namely, healthy adopters, healthy nonadopters, infected adopters, and infected nonadopters, and to incorporate more transition rules between states [33], which could be explored in future research. Additionally, a rigorous theoretical framework is required to mathematically prove the existence of periodic behaviors within complex networks. Moreover, incorporating more realistic cognitive features, such as bounded rationality and inertia, deserves further exploration [51].

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DATA AVAILABILITY

There are no publicly available research data or software supporting this manuscript. Requests for further information or data should be sent to the authors.

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