



Research article

Dynamical analysis of a chestnut blight model with two control strategies

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Abstract: To investigate how a finite-duration protective intervention interacts with hypovirus-mediated biological control, we formulate a chestnut blight-inspired host–pathogen–hyperparasite model containing susceptible, temporarily protected, pathogen-infected, and hyperparasite-associated host classes. Although no broadly effective and scalable chemical treatment is currently available for chestnut blight, several experimental studies have reported temporary reductions in canker expansion or disease incidence following chemical treatment under specimen-tree, orchard, nursery, or otherwise managed conditions. Motivated by these limited observations, the class M is interpreted as a temporarily protected class rather than as an immunologically immune class. Susceptible hosts enter this class at rate δ and return to the susceptible class after protection is lost at rate ρ . Basic reproduction numbers R_{0IS} and R_{0HS} are derived, and the existence and stability of the boundary, disease-free, and positive equilibria are analyzed. We further introduce the composite protection index $\Theta(\delta, \rho) = 1 + \frac{\delta}{\rho+d}$, which quantifies the combined influence of entry into and loss from the temporarily protected class. Increasing δ decreases the effective reproduction numbers and raises the pathogen-invasion thresholds, whereas increasing ρ has the opposite effect. The parameters δ and ρ also shift the transcritical-bifurcation thresholds associated with hyperparasite invasion and alter the equilibrium distribution among the pathogen-only and hyperparasite-associated classes. Numerical simulations illustrate these threshold shifts and the nonmonotone interaction between temporary protection and hypovirus transmission. The chemical component of the model therefore represents a finite-duration protective effect under idealized conditions, rather than a chemical cure or chemically induced immunity. The results should be interpreted as a theoretical investigation of how temporary protection may affect chestnut blight transmission dynamics, not as a directly validated management prescription for chestnut blight.

Keywords: chestnut blight; hyperparasitism; hypovirulence-associated virus; dynamical model; transcritical bifurcation; control measures

1. Introduction

Climate change and the increasing movement of people and goods have facilitated the geographical spread of plant pathogens, posing growing challenges to agricultural production, forest health, and food security. Chemical control remains an important component of plant disease management because it can suppress outbreaks rapidly. However, repeated or intensive pesticide use may select for resistant pathogen populations, increase management costs, and contribute to environmental contamination and ecological disruption. Pathogen adaptation under sustained selection pressure may further compromise conventional control strategies [1]. These limitations have stimulated interest in more sustainable and integrated approaches to disease management.

Biological control uses living organisms or their products to suppress pathogens and pests. It can reduce reliance on chemical pesticides, although its effectiveness depends on the biological and environmental context. One mechanism of particular interest is hyperparasitism, in which a parasite or pathogen is itself parasitized, giving rise to a host–pathogen–hyperparasite interaction. Hyperparasitism occurs in diverse systems, including bacterial pathogens [2, 3], fungal pathogens [4], and other eukaryotic parasites [5]. For example, bacteriophages infect bacterial pathogens such as *Escherichia coli*, *Salmonella enterica* serovar Typhimurium, and *Klebsiella pneumoniae* [6, 7]. Hyperparasitic fungi can also infect powdery mildew pathogens [8], while hypovirulence-associated mycoviruses infect the chestnut blight fungus *Cryphonectria parasitica* [9].

Biological and chemical agents have also been combined in integrated pest and disease management. Examples include the use of *Metarhizium anisopliae* with chemical pesticides against *Spodoptera frugiperda* [10], and *Bacillus subtilis* with fungicides against wheat root rot [11], tomato late blight [12] and apple anthracnose [13, 14]. Nevertheless, biological control is not universally effective. Its performance may be constrained by environmental suitability, interactions with non-target organisms and native microbial communities, and implementation costs [15]. Moreover, biological control is not necessarily more durable than chemical control [16]. In hyperparasite-based strategies, changes in pathogen virulence and transmission can further complicate long-term outcomes.

Chestnut blight, caused by *C. parasitica*, provides a well-studied example of a host–pathogen–hyperparasite interaction. Infection of the fungus by *Cryphonectria hypovirus 1* (CHV1) can reduce fungal virulence and limit canker development, a phenomenon known as hypovirus-mediated hypovirulence [17, 18]. Hypovirus-based biological control has been successful in some regions, particularly in Europe, but its effectiveness varies geographically. In particular, vegetative incompatibility among fungal strains can restrict horizontal hypovirus transmission, while pathogen population structure and local environmental conditions can affect its establishment and spread.

Chemical treatment is not generally considered a broadly effective or scalable solution for chestnut blight, particularly in natural forests. Nevertheless, several studies have reported partial or short-term disease suppression under experimental or managed conditions. Jaynes and Van Alfen [19] reported reduced canker growth following pressure injection of methyl-2-benzimidazole carbamate. Aksoy and Serdar [20] observed lower disease incidence under selected fungicide treatments in a managed chestnut orchard. Trapiello et al. [21] documented short-term reductions in lesion development after direct agrochemical application, while in vitro experiments have identified fungicides that inhibit the

mycelial growth of *C. parasitica* [22]. These findings do not demonstrate that chemical treatment induces biological immunity or provides a durable cure. Accordingly, the “temporarily protected” host class considered in this study is a phenomenological modeling assumption, not a claim that chemically induced immunity has been established for chestnut trees.

Mathematical models have provided important insights into host–pathogen–hyperparasite interactions. Taylor et al. [23] investigated the acquisition of hypovirulence in a three-trophic-level framework, while Morozov et al. [24] developed a chestnut blight-motivated model to examine hyperparasite establishment and its implications for biological control. More recently, Northrup et al. [25] studied the evolutionary dynamics of hyperparasites, highlighting the role of their cotransmission with primary parasites, or “hitchhiking,” in shaping hyperparasite virulence. Although these studies have advanced understanding of the ecological and evolutionary consequences of hyperparasitism, the influence of a finite period of host protection on invasion thresholds, equilibrium structure, and long-term population dynamics warrants further investigation.

Motivated by this gap, we develop a chestnut blight-inspired host–pathogen–hyperparasite transmission model that incorporates host recruitment, natural mortality, and a temporarily protected host class. The parameter δ denotes the rate at which susceptible hosts enter the protected class, whereas ρ denotes the rate at which protection is lost and hosts return to susceptibility. We investigate the existence and stability of the system’s equilibria, characterize the thresholds associated with transcritical bifurcations, and use numerical simulations to assess how temporary protection influences long-term population dynamics and biological control outcomes. Our aim is to clarify the theoretical implications of a finite protection period, rather than to establish the effectiveness of any specific chemical intervention against chestnut blight.

The remainder of this paper is organized as follows. Section 2 formulates the model and establishes its basic properties. Section 3 analyzes the existence, stability, and bifurcation behavior of the boundary equilibria. Section 4 investigates the existence and stability of the positive equilibrium. Section 5 presents numerical simulations illustrating the effects of key parameters on the system dynamics and potential control outcomes. Section 6 summarizes the main findings, discusses their biological implications, and outlines directions for future research.

2. Materials and methods

In this study, chestnut blight provides the biological structure of the nested “host–pathogen–hyperparasite” interaction. Chemical treatment is not assumed to cure chestnut blight or to generate permanent host immunity. Instead, motivated by the limited short-term suppression reported for individual trees, orchard experiments, and managed conditions [19–22], we introduce a temporarily protected host class. This class represents an idealized period during which host susceptibility is reduced after an external intervention. The model does not specify a particular fungicide, dosage, application method, or field-treatment schedule.

Our model comprises four primary host population compartments: susceptible hosts (S), temporarily protected hosts (M), pathogen-infected hosts without the hyperparasite (I), and hosts associated with both the pathogen and the hyperparasite (H), with the total host population denoted by N . Class M does not represent immunological immunity. It represents an idealized state in which susceptibility to new infection is temporarily reduced. Hosts in class H harbor both the pathogen and

the hyperparasite, whereas class I contains hosts carrying the pathogen without the hyperparasite [26]. Based on the schematic in Figure 1 and the parameter definitions provided in Table 1, model (2.1) is formulated as follows:

$$\begin{cases} \frac{dS}{dt} = \Lambda - (\delta + d)S + \rho M - \beta SI - \eta\beta SH, \\ \frac{dM}{dt} = \delta S - (\rho + d)M, \\ \frac{dI}{dt} = \beta SI - \sigma HI - (d + \alpha)I, \\ \frac{dH}{dt} = \sigma HI + \eta\beta SH - (d + \lambda\alpha)H. \end{cases} \quad (2.1)$$

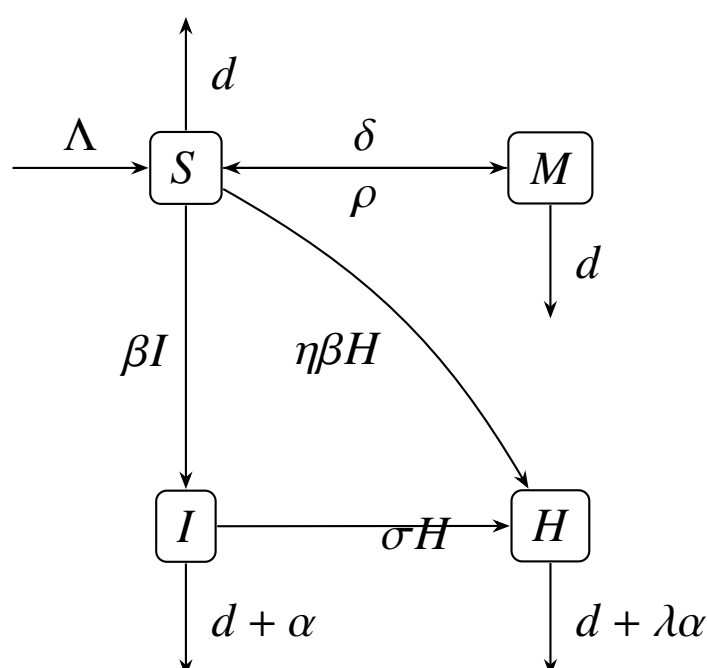


Figure 1. Schematic diagram of the model structure and transmission mechanism.

Table 1. Description of parameters.

Parameter	Parameter Meaning
Λ	host recruitment rate
δ	rate at which susceptible hosts enter the hypothetical temporarily protected class
ρ	rate at which hypothetical temporary protection is lost
d	natural death rate of hosts
β	transmission rate from susceptible hosts S to parasitized hosts I
σ	hyperparasite transmission-conversion rate from class I to class H
η	modification of focal-pathogen transmission by the hyperparasite
α	focal-pathogen-induced host death rate
λ	modification of pathogen-induced mortality in hyperparasite-associated hosts

The temporary-protection process is represented by $S \xrightarrow{\delta} M \xrightarrow{\rho} S$.

The parameter δ is the per-capita rate at which susceptible hosts enter the temporarily protected class, whereas ρ is the rate at which protection is lost. In the absence of natural mortality, the mean protection duration is $T_{\text{protection}} = \frac{1}{\rho}$. Because hosts in class M also experience natural mortality at rate d , their mean residence time in this class is $T_M = \frac{1}{\rho+d}$. The probability that a temporarily protected host returns to the susceptible class before natural death is $P_{M \rightarrow S} = \frac{\rho}{\rho+d}$. Consequently, a larger δ corresponds to more rapid entry into temporary protection, whereas a larger ρ corresponds to a shorter protection period and a more rapid return to susceptibility. These parameters describe transition rates in the theoretical model and should not be interpreted directly as fungicide dosage or efficacy.

The nested host–pathogen–hyperparasite structure is biologically inspired by systems such as hypovirus-associated chestnut blight [18], and it extends the classical models in [9, 23–25, 27]. However, the transition $S \rightarrow M$ at rate δ is a purely hypothetical external-protection mechanism. It is not calibrated to a chemical product, does not represent an effective treatment for chestnut blight, and is included only to study the mathematical interaction between direct host protection and hyperparasite-mediated regulation in a generalized disease system. As in [26], we assume that once a host enters the pathogen-infected class, it cannot return directly to the susceptible class.

For the convenience of subsequent analysis, we define

$$R_{0IS} = \frac{\Lambda\beta}{d(d+\alpha)}, \quad R_{0HS} = \frac{\Lambda\eta\beta}{d(d+\lambda\alpha)}.$$

Here R_{0IS} is the basic reproduction number of chestnut blight when only the pathogen is present and no hypovirus exists in the system, whereas R_{0HS} is the basic reproduction number of the hypovirus when only the hypovirus is present and the pathogen-only class is absent.

It is easy to obtain the positively invariant set of system (2.1):

$$D = \left\{ (S, M, I, H) \mid 0 \leq S + M + I + H \leq \frac{\Lambda}{d}, S \geq 0, M \geq 0, I \geq 0, H \geq 0 \right\}.$$

In what follows, we study the dynamical behavior of model (2.1) on this positively invariant set.

3. Existence and stability of boundary equilibria

3.1. The hyperparasite-free boundary equilibrium

For $H = 0$ and $I > 0$, the hyperparasite-free boundary equilibrium of system (2.1) is given by

$$E_{01} = \left(\frac{d+\alpha}{\beta}, \frac{\delta(d+\alpha)}{\beta(\rho+d)}, \frac{\Lambda\beta(\rho+d) - d(d+\alpha)(\delta+\rho+d)}{\beta(d+\alpha)(\rho+d)}, 0 \right).$$

Theorem 3.1. *The hyperparasite-free equilibrium E_{01} exists if and only if*

$$R_{0IS} > 1 + \frac{\delta}{\rho+d}.$$

Proof. In order to have $I_{01} > 0$, it is necessary and sufficient that

$$\Lambda\beta(\rho+d) - d(d+\alpha)(\rho+d+\delta) > 0.$$

Dividing both sides by $d(\alpha + d)(\rho + d)$, we obtain

$$R_{0IS} > 1 + \frac{\delta}{\rho + d}.$$

To verify that $E_{01} \in D$, note that

$$S_{01} = \frac{d + \alpha}{\beta} > 0, \quad M_{01} = \frac{\delta(d + \alpha)}{\beta(\rho + d)} > 0,$$

and

$$I_{01} = \frac{\Lambda\beta(\rho + d) - d(d + \alpha)(\rho + d + \delta)}{\beta(d + \alpha)(\rho + d)}.$$

By the existence condition,

$$I_{01} \geq 0 \iff \Lambda\beta(\rho + d) \geq d(d + \alpha)(\rho + d + \delta).$$

Hence all components of E_{01} are nonnegative. Moreover,

$$S_{01} + M_{01} + I_{01} = \frac{\Lambda - \alpha I_{01}}{d} \leq \frac{\Lambda}{d}.$$

Therefore, whenever it exists, $E_{01} \in D$. □

We next discuss the stability of E_{01} .

Theorem 3.2. *If*

$$R_{0IS} + \frac{\eta\beta(\alpha + d)}{\sigma d} > 1 + \frac{\delta}{\rho + d} + \frac{\beta(d + \lambda\alpha)}{\sigma d},$$

the system is unstable at E_{01} . Conversely, if

$$R_{0IS} + \frac{\eta\beta(\alpha + d)}{\sigma d} < 1 + \frac{\delta}{\rho + d} + \frac{\beta(d + \lambda\alpha)}{\sigma d},$$

then the system is globally asymptotically stable at E_{01} .

Proof. The Jacobian matrix at E_{01} is

$$J = \begin{pmatrix} -\delta - d - \beta I_{01} & \rho & -\beta S_{01} & -\eta\beta S_{01} \\ \delta & -(\rho + d) & 0 & 0 \\ \beta I_{01} & 0 & 0 & -\sigma I_{01} \\ 0 & 0 & 0 & \sigma I_{01} + \eta\beta S_{01} - (d + \lambda\alpha) \end{pmatrix}.$$

Consider first the submatrix

$$J_1 = \begin{pmatrix} -\delta - d - \beta I_{01} & \rho & -\beta S_{01} \\ \delta & -(\rho + d) & 0 \\ \beta I_{01} & 0 & 0 \end{pmatrix},$$

whose characteristic polynomial is

$$\lambda^3 + (\rho + 2d + \delta + \beta I_{01})\lambda^2 + [(d + \beta I_{01})(\rho + d) + d\delta + \beta S_{01}\beta I_{01}]\lambda + \beta I_{01}\beta S_{01}(\rho + d) = 0.$$

Let $y = \delta + d + \beta I_{01}$. Then the Hurwitz determinants satisfy

$$\begin{aligned}\Delta_1 &= \rho + d + \delta + d + \beta I_{01} > 0, \\ \Delta_2 &= [(d + \beta I_{01})(\rho + d) + d\delta + \beta S_{01}\beta I_{01}] \cdot [(\rho + d) + y] - \beta S_{01}\beta I_{01}(\rho + d) \\ &= [(d + \beta I_{01})(\rho + d) + d\delta] \cdot [(\rho + d) + y] + y\beta S_{01}\beta I_{01} > 0, \\ \Delta_3 &= \beta S_{01}\beta I_{01}(\rho + d) \cdot \Delta_2 > 0.\end{aligned}$$

Hence the three eigenvalues of J_1 all have negative real parts.

The fourth eigenvalue is

$$\lambda_4 = \sigma I_{01} + \eta \beta S_{01} - (d + \lambda \alpha).$$

Substituting I_{01} , we obtain

$$\lambda_4 = \frac{\Lambda \beta (\rho + d) - \sigma d (\alpha + d) (\rho + d + \delta) + \eta \beta (\alpha + d)^2 (\rho + d) - \beta (\alpha + d) (\rho + d) (d + \lambda \alpha)}{\beta (\alpha + d) (\rho + d)}.$$

If

$$R_{0IS} + \frac{\eta \beta (\alpha + d)}{\sigma d} > 1 + \frac{\delta}{\rho + d} + \frac{\beta (d + \lambda \alpha)}{\sigma d},$$

then $\lambda_4 > 0$, and E_{01} is unstable.

If

$$R_{0IS} + \frac{\eta \beta (\alpha + d)}{\sigma d} < 1 + \frac{\delta}{\rho + d} + \frac{\beta (d + \lambda \alpha)}{\sigma d},$$

$\lambda_4 < 0$, and E_{01} is locally asymptotically stable.

Define the Lyapunov function

$$\begin{aligned}V(S, M, I, H) &= S - S_{01} - S_{01} \ln \frac{S}{S_{01}} \\ &\quad + \frac{\rho}{\rho + d} \left(M - M_{01} - M_{01} \ln \frac{M}{M_{01}} \right) \\ &\quad + I - I_{01} - I_{01} \ln \frac{I}{I_{01}} + H.\end{aligned}\tag{3.1}$$

Clearly,

$$V(S, M, I, H) \geq 0,$$

and

$$V(S, M, I, H) = 0$$

if and only if

$$(S, M, I, H) = E_{01}.$$

Let

$$x = \frac{S}{S_{01}}, \quad y = \frac{M}{M_{01}}, \quad z = \frac{I}{I_{01}}.$$

Then

$$\begin{aligned}\dot{V} &= \left(1 - \frac{S_{01}}{S}\right) S' + \frac{\rho}{\rho + d} \left(1 - \frac{M_{01}}{M}\right) M' \\ &\quad + \left(1 - \frac{I_{01}}{I}\right) I' + H' .\end{aligned}$$

Substituting system (2.1) into the above expression and using the equilibrium identities at E_{01} , we obtain

$$\begin{aligned}\dot{V} = & -\frac{S_{01}d(d+\delta+\rho)(x-1)^2}{\rho+d} \frac{1}{x} \\ & -\frac{S_{01}\delta\rho(x-y)^2}{\rho+d} \frac{1}{xy} \\ & -\beta S_{01}I_{01} \frac{(x-1)^2}{x} \\ & + [\sigma I_{01} + \eta\beta S_{01} - (d + \lambda\alpha)] H.\end{aligned}$$

Denote

$$\lambda_4 = \sigma I_{01} + \eta\beta S_{01} - (d + \lambda\alpha).$$

Under the condition

$$R_{0IS} + \frac{\eta\beta(\alpha+d)}{\sigma d} < 1 + \frac{\delta}{\rho+d} + \frac{\beta(d+\lambda\alpha)}{\sigma d},$$

we have

$$\lambda_4 < 0.$$

Therefore,

$$\dot{V} \leq 0$$

for all $(S, M, I, H) \in D_{01}$.

Moreover, $\dot{V} = 0$ implies

$$x = 1, \quad x = y, \quad H = 0.$$

Hence

$$S = S_{01}, \quad M = M_{01}, \quad H = 0.$$

On this set, the first equation of system (2.1) becomes

$$S' = \Lambda - (\delta + d)S_{01} + \rho M_{01} - \beta S_{01}I.$$

Using

$$\Lambda - (\delta + d)S_{01} + \rho M_{01} = \beta S_{01}I_{01},$$

we obtain

$$S' = \beta S_{01}(I_{01} - I).$$

For a trajectory to remain in the set $\{\dot{V} = 0\}$, it is necessary that

$$S' \equiv 0.$$

Thus

$$I = I_{01}.$$

Consequently, the largest invariant subset of

$$\{(S, M, I, H) \in D_{01} : \dot{V} = 0\}$$

is the singleton

$$\{E_{01}\}.$$

By LaSalle's invariance principle, every solution starting from D_{01} converges to E_{01} . Combining this global attractivity with the local asymptotic stability proved above, E_{01} is globally asymptotically stable relative to D_{01} .

Therefore, E_{01} is globally asymptotically stable relative to

$$D_{01} = \{(S, M, I, H) \in D : S > 0, M > 0, I > 0, H \geq 0\}.$$

□

Theorem 3.3. When $\sigma = \sigma_{01}$, the system undergoes a transcritical bifurcation at E_{01} .

Proof. In the proof of Theorem 3.2, if

$$\lambda_4 = \sigma I_{01} + \eta \beta S_{01} - (d + \lambda \alpha) = 0,$$

$$\sigma I_{01} = (d + \lambda \alpha) - \eta \beta S_{01},$$

and

$$I_{01} > 0,$$

so

$$(d + \lambda \alpha) - \eta(\alpha + d) > 0.$$

Moreover, the existence of I_{01} implies that

$$\Lambda \beta(\rho + d) - d(\alpha + d)(\rho + d + \delta) > 0.$$

Taking

$$\sigma_{01} = \frac{\beta(\rho + d)(\alpha + d) [(d + \lambda \alpha) - \eta(\alpha + d)]}{\Lambda \beta(\rho + d) - d(\alpha + d)(\rho + d + \delta)}$$

as the bifurcation parameter and using $I_{01} > 0$ together with $\sigma > 0$, we obtain $(d + \lambda \alpha) - \eta(\alpha + d) > 0$. The existence of

$$I_{01} = \frac{\Lambda \beta(\rho + d) - d(\alpha + d)(\rho + d + \delta)}{\beta(\alpha + d)(\rho + d)}$$

also implies

$$\Lambda \beta(\rho + d) - d(\alpha + d)(\rho + d + \delta) > 0.$$

Hence $\sigma_{01} > 0$.

We use the Sotomayor theorem [28]. The right eigenvector associated with the zero eigenvalue is

$$V = \begin{pmatrix} \sigma_{01}(\rho + d) \\ \frac{\delta \sigma_{01}}{\alpha + d} \\ -\frac{\sigma_{01}(\rho + d)(d + \beta I_{01}) - d \delta \sigma_{01} - \eta \beta(\rho + d)(\alpha + d)}{\alpha + d} \\ \beta(\rho + d) \end{pmatrix} = \begin{pmatrix} V_1 \\ V_2 \\ V_3 \\ V_4 \end{pmatrix},$$

whereas the corresponding left eigenvector is

$$\mathbf{W} = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 1 \end{pmatrix}.$$

Since $f_\sigma(E_{01}, \sigma_{01}) = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}$, we have

$$\mathbf{W}^T \cdot f_\sigma(E_{01}, \sigma_{01}) = 0.$$

Moreover,

$$Df_\sigma(E_{01}, \sigma_{01}) = \begin{pmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -I_{01} \\ 0 & 0 & 0 & I_{01} \end{pmatrix},$$

and therefore

$$\mathbf{W}^T \cdot Df_\sigma(E_{01}, \sigma_{01}) \cdot \mathbf{V} = V_4 I_{01} = I_{01}\beta(\rho + d) \neq 0.$$

For the second-order term,

$$\mathbf{W}^T \cdot D^2 f(E_{01}, \sigma_{01}) \cdot (\mathbf{V}, \mathbf{V}) = 2(\eta\beta V_1 V_4 + \sigma_{01} V_3 V_4) = 2V_4(\eta\beta V_1 + \sigma_{01} V_3).$$

Furthermore,

$$\begin{aligned} \eta\beta V_1 + \sigma_{01} V_3 &= \eta\beta\sigma_{01}(\rho + d) + \sigma_{01} \cdot \frac{-\sigma_{01}(\rho + d)(d + \beta I_{01}) + d\delta\sigma_{01} + \eta\beta(\rho + d)(\alpha + d)}{\alpha + d} \\ &= \sigma_{01} \cdot \frac{\eta\beta(\rho + d)(\alpha + d) - \sigma_{01}(\rho + d)(d + \beta I_{01}) - d\delta\sigma_{01} + \eta\beta(\rho + d)(\alpha + d)}{\alpha + d}. \end{aligned}$$

After simplification,

$$\eta\beta V_1 + \sigma_{01} V_3 < 0,$$

and hence

$$\mathbf{W}^T \cdot D^2 f(E_{01}, \sigma_{01}) \cdot (\mathbf{V}, \mathbf{V}) \neq 0.$$

Therefore, when $\sigma = \sigma_{01}$, the system undergoes a transcritical bifurcation at E_{01} . $\square$

3.2. The hypovirus-dominated boundary equilibrium

For $I = 0$ and $H > 0$, the hypovirus-dominant boundary equilibrium of system (2.1) is given by

$$E_{02} = \left(\frac{d + \lambda\alpha}{\eta\beta}, \frac{\delta(d + \lambda\alpha)}{\eta\beta(\rho + d)}, 0, \frac{\Lambda\eta\beta(\rho + d) - d(d + \lambda\alpha)(\delta + \rho + d)}{\eta\beta(d + \lambda\alpha)(\rho + d)} \right).$$

Theorem 3.4. *The boundary equilibrium E_{02} exists if and only if*

$$R_{0HS} > 1 + \frac{\delta}{\rho + d}.$$

Proof. For $H_{02} > 0$, we need

$$\Lambda\eta\beta(\rho + d) - d(d + \lambda\alpha)(\rho + d + \delta) > 0.$$

Dividing both sides by $d(d + \lambda\alpha)(\rho + d)$ gives the existence condition

$$R_{0HS} > 1 + \frac{\delta}{\rho + d}.$$

To verify that $E_{02} \in D$, note that

$$S_{02} = \frac{d + \lambda\alpha}{\eta\beta} > 0, \quad M_{02} = \frac{\delta(d + \lambda\alpha)}{\eta\beta(\rho + d)} > 0,$$

and

$$H_{02} = \frac{\Lambda\eta\beta(\rho + d) - d(d + \lambda\alpha)(\rho + d + \delta)}{\eta\beta(d + \lambda\alpha)(\rho + d)}.$$

By the existence condition,

$$H_{02} \geq 0 \iff \Lambda\eta\beta(\rho + d) \geq d(d + \lambda\alpha)(\rho + d + \delta).$$

Hence all components are nonnegative, and

$$S_{02} + M_{02} + H_{02} = \frac{\Lambda - \lambda\alpha H_{02}}{d} \leq \frac{\Lambda}{d}.$$

Therefore $E_{02} \in D$ whenever it exists. □

Theorem 3.5. *If*

$$R_{0HS} + \frac{\eta\beta(d + \alpha)}{\sigma d} < \frac{\beta(d + \lambda\alpha)}{\sigma d} + 1 + \frac{\delta}{\rho + d},$$

E_{02} is unstable. *If*

$$R_{0HS} + \frac{\eta\beta(d + \alpha)}{\sigma d} > \frac{\beta(d + \lambda\alpha)}{\sigma d} + 1 + \frac{\delta}{\rho + d},$$

E_{02} is globally asymptotically stable.

Proof. The Jacobian matrix at E_{02} is

$$J = \begin{pmatrix} -\delta - d - \eta\beta H_{02} & \rho & -\beta S_{02} & -\eta\beta S_{02} \\ \delta & -(\rho + d) & 0 & 0 \\ 0 & 0 & \beta S_{02} - \sigma H_{02} - (d + \alpha) & 0 \\ \eta\beta H_{02} & 0 & \sigma H_{02} & 0 \end{pmatrix}.$$

Consider the submatrix

$$J_2 = \begin{pmatrix} -\delta - d - \eta\beta H_{02} & \rho & -\beta S_{02} \\ \delta & -(\rho + d) & 0 \\ \eta\beta H_{02} & 0 & 0 \end{pmatrix}.$$

Let $z = \delta + d + \eta\beta H_{02}$. Its characteristic polynomial is

$$\lambda^3 + (\rho + d + z)\lambda^2 + [(d + \eta\beta H_{02})(\rho + d) + d\delta + \eta\beta S_{02} \cdot \eta\beta H_{02}] \lambda + \eta\beta H_{02} \cdot \eta\beta S_{02}(\rho + d) = 0.$$

By the Hurwitz criterion, the three eigenvalues of J_2 all have negative real parts.

The remaining critical eigenvalue is

$$\lambda_3 = \beta S_{02} - \sigma H_{02} - (d + \alpha).$$

If

$$R_{0HS} + \frac{\eta\beta(d + \alpha)}{\sigma d} < \frac{\beta(d + \lambda\alpha)}{\sigma d} + 1 + \frac{\delta}{\rho + d},$$

substituting the equilibrium values

$$S_{02} = \frac{d + \lambda\alpha}{\eta\beta}, \quad H_{02} = \frac{\Lambda\eta\beta(\rho + d) - d(d + \lambda\alpha)(\rho + d + \delta)}{\eta\beta(d + \lambda\alpha)(\rho + d)}$$

into λ_3 , we obtain

$$\begin{aligned} \lambda_3 &= \beta \cdot \frac{d + \lambda\alpha}{\eta\beta} - \sigma \cdot \frac{\Lambda\eta\beta(\rho + d) - d(d + \lambda\alpha)(\rho + d + \delta)}{\eta\beta(d + \lambda\alpha)(\rho + d)} - (d + \alpha) \\ &= \frac{\beta(d + \lambda\alpha)^2(\rho + d) - \sigma\Lambda\eta\beta(\rho + d) + \sigma d(d + \lambda\alpha)(\rho + d + \delta) - (d + \alpha)\eta\beta(d + \lambda\alpha)(\rho + d)}{\eta\beta(d + \lambda\alpha)(\rho + d)}. \end{aligned}$$

Since the denominator $\eta\beta(d + \lambda\alpha)(\rho + d) > 0$, the sign of λ_3 is determined solely by its numerator. Under the condition

$$R_{0HS} + \frac{\eta\beta(d + \alpha)}{\sigma d} < \frac{\beta(d + \lambda\alpha)}{\sigma d} + 1 + \frac{\delta}{\rho + d},$$

the numerator is positive and hence $\lambda_3 > 0$, which implies the instability of E_{02} .

If

$$R_{0HS} + \frac{\eta\beta(d + \alpha)}{\sigma d} > \frac{\beta(d + \lambda\alpha)}{\sigma d} + 1 + \frac{\delta}{\rho + d},$$

$$\begin{aligned} \lambda_3 &= \beta \cdot \frac{d + \lambda\alpha}{\eta\beta} - \sigma \cdot \frac{\Lambda\eta\beta(\rho + d) - d(d + \lambda\alpha)(\rho + d + \delta)}{\eta\beta(d + \lambda\alpha)(\rho + d)} - (d + \alpha) < 0 \\ &= \frac{\beta(d + \lambda\alpha)^2(\rho + d) - \sigma\Lambda\eta\beta(\rho + d) + \sigma d(d + \lambda\alpha)(\rho + d + \delta) - (d + \alpha)\eta\beta(d + \lambda\alpha)(\rho + d)}{\eta\beta(d + \lambda\alpha)(\rho + d)} < 0. \end{aligned}$$

Thus E_{02} is locally asymptotically stable.

Define the Lyapunov function

$$\begin{aligned} V(S, M, I, H) &= S - S_{02} - S_{02} \ln \frac{S}{S_{02}} \\ &\quad + \frac{\rho}{\rho + d} \left(M - M_{02} - M_{02} \ln \frac{M}{M_{02}} \right) \\ &\quad + I + H - H_{02} - H_{02} \ln \frac{H}{H_{02}}. \end{aligned}$$

Clearly,

$$V(S, M, I, H) \geq 0,$$

and

$$V(S, M, I, H) = 0$$

if and only if

$$(S, M, I, H) = E_{02}.$$

Let

$$x = \frac{S}{S_{02}}, \quad y = \frac{M}{M_{02}}, \quad z = \frac{H}{H_{02}}.$$

Then

$$\begin{aligned} \dot{V} = & \left(1 - \frac{S_{02}}{S}\right) S' + \frac{\rho}{\rho + d} \left(1 - \frac{M_{02}}{M}\right) M' \\ & + I' + \left(1 - \frac{H_{02}}{H}\right) H'. \end{aligned}$$

Substituting system (2.1) into the above expression and using the equilibrium identities at E_{02} , we obtain

$$\begin{aligned} \dot{V} = & - \frac{S_{02}d(d + \delta + \rho)}{\rho + d} \frac{(x - 1)^2}{x} \\ & - \frac{S_{02}\delta\rho}{\rho + d} \frac{(x - y)^2}{xy} \\ & - \eta\beta S_{02}H_{02} \frac{(x - 1)^2}{x} \\ & + [\beta S_{02} - \sigma H_{02} - (d + \alpha)] I. \end{aligned}$$

Denote

$$\lambda_3 = \beta S_{02} - \sigma H_{02} - (d + \alpha).$$

Under the condition

$$R_{0HS} + \frac{\eta\beta(d + \alpha)}{\sigma d} > \frac{\beta(d + \lambda\alpha)}{\sigma d} + 1 + \frac{\delta}{\rho + d},$$

we have

$$\lambda_3 < 0.$$

Therefore,

$$\dot{V} \leq 0$$

for all $(S, M, I, H) \in D_{02}$.

Moreover, $\dot{V} = 0$ implies

$$x = 1, \quad x = y, \quad I = 0.$$

Hence

$$S = S_{02}, \quad M = M_{02}, \quad I = 0.$$

On this set, the first equation of system (2.1) becomes

$$S' = \Lambda - (\delta + d)S_{02} + \rho M_{02} - \eta\beta S_{02}H.$$

Using

$$\Lambda - (\delta + d)S_{02} + \rho M_{02} = \eta\beta S_{02}H_{02},$$

we obtain

$$S' = \eta\beta S_{02}(H_{02} - H).$$

For a trajectory to remain in the set $\{\dot{V} = 0\}$, it is necessary that

$$S' \equiv 0.$$

Thus

$$H = H_{02}.$$

Consequently, the largest invariant subset of

$$\{(S, M, I, H) \in D_{02} : \dot{V} = 0\}$$

is the singleton

$$\{E_{02}\}.$$

By LaSalle's invariance principle, every solution starting from D_{02} converges to E_{02} . Combining this global attractivity with the local asymptotic stability proved above, E_{02} is globally asymptotically stable relative to D_{02} .

□

Theorem 3.6. When $\sigma = \sigma_{02}$, the system undergoes a transcritical bifurcation at E_{02} .

Proof. In the proof of Theorem 3.5, if

$$\lambda_3 = \beta S_{02} - \sigma H_{02} - (d + \alpha) = 0,$$

$$\frac{d + \lambda\alpha}{\eta} - (d + \alpha) = \sigma H_{02},$$

substituting H_{02} into the above, we obtain

$$\sigma_{02} = \frac{\beta(\rho + d)(d + \lambda\alpha)[(d + \lambda\alpha) - \eta(d + \alpha)]}{\Lambda\eta\beta(\rho + d) - d(d + \lambda\alpha)(\rho + d + \delta)},$$

as the bifurcation parameter. Since $H_{02} > 0$, we have $d + \lambda\alpha - \eta(d + \alpha) > 0$. The existence of H_{02} also implies

$$\Lambda\eta\beta(\rho + d) - d(d + \lambda\alpha)(\rho + d + \delta) > 0,$$

so $\sigma_{02} > 0$.

The right eigenvector is

$$V = \begin{pmatrix} \sigma_{02}(\rho + d) \\ \frac{\delta\sigma_{02}}{\eta\beta(\rho + d)} \\ -\frac{\sigma_{02}(\rho + d)(d + \eta\beta H_{02}) - d\delta\sigma_{02} - (\rho + d)(\lambda\alpha + d)}{\eta\beta S_{02}} \\ V_4 \end{pmatrix} = \begin{pmatrix} V_1 \\ V_2 \\ V_3 \\ V_4 \end{pmatrix},$$

and the left eigenvector is

$$\mathbf{W} = \begin{pmatrix} 0 \\ 0 \\ 1 \\ 0 \end{pmatrix}.$$

Because

$$f_{\sigma}(E_{02}, \sigma_{02}) = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \end{pmatrix},$$

we have

$$\mathbf{W}^T \cdot f_{\sigma}(E_{02}, \sigma_{02}) = 0.$$

Moreover,

$$Df_{\sigma}(E_{02}, \sigma_{02}) = \begin{pmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & -H_{02} & 0 \\ 0 & 0 & H_{02} & 0 \end{pmatrix},$$

so that

$$\mathbf{W}^T \cdot Df_{\sigma}(E_{02}, \sigma_{02}) \cdot \mathbf{V} = -H_{02}V_3 \neq 0,$$

and

$$\mathbf{W}^T \cdot D^2 f(E_{02}, \sigma_{02}) \cdot (\mathbf{V}, \mathbf{V}) = 2(\beta V_1 - \sigma_{02} V_4)V_3 \neq 0.$$

Therefore the system undergoes a transcritical bifurcation at E_{02} . □

3.3. The disease-free equilibrium

We next consider the case in which the chestnut orchard is free from both chestnut blight and the hypovirus. The corresponding disease-free equilibrium is $E_{03} = (S_{03}, M_{03}, 0, 0)$.

Thus

$$E_{03} = \left(\frac{\Lambda(\rho + d)}{d(\delta + \rho + d)}, \frac{\delta\Lambda}{d(\delta + \rho + d)}, 0, 0 \right).$$

Clearly $S_{03} > 0$, $M_{03} > 0$, $I = 0$, and $H = 0$. Moreover,

$$S_{03} + M_{03} = \frac{\Lambda(\rho + d) + \Lambda\delta}{d(d + \rho + \delta)} = \frac{\Lambda}{d},$$

so $E_{03} \in D$.

The corresponding Jacobian matrix is

$$J_3 = \begin{pmatrix} -\delta - d & \rho & -\beta S_{03} & -\eta\beta S_{03} \\ \delta & -(\rho + d) & 0 & 0 \\ 0 & 0 & \beta S_{03} - (d + \alpha) & 0 \\ 0 & 0 & 0 & \eta\beta S_{03} - (d + \lambda\alpha) \end{pmatrix}.$$

The 2×2 block

$$\begin{pmatrix} -\delta - d & \rho \\ \delta & -(\rho + d) \end{pmatrix}$$

has two eigenvalues with negative real parts. Thus the stability is determined by the remaining two eigenvalues.

Theorem 3.7. *The system undergoes a transcritical bifurcation at E_{03} if and only if $\beta = \beta^*$ and*

$$R_{0HS} < 1 + \frac{\delta}{\rho + d}.$$

Proof. When

$$R_{0IS} = 1 + \frac{\delta}{\rho + d},$$

take

$$\beta^* = \frac{d(d + \alpha)(d + \rho + \delta)}{\Lambda(\rho + d)}.$$

Then $\lambda_3 = 0$, namely $\beta S_{03} = d + \alpha$. The right eigenvector corresponding to $\lambda_3 = 0$ is

$$V = \begin{pmatrix} -\frac{(d + \alpha)(\rho + d)}{d(d + \rho + \delta)} \\ -\frac{d(d + \rho + \delta)}{\delta(d + \alpha)} \\ 1 \\ 0 \end{pmatrix} = \begin{pmatrix} V_1 \\ V_2 \\ V_3 \\ V_4 \end{pmatrix},$$

and the corresponding left eigenvector is

$$W = \begin{pmatrix} 0 \\ 0 \\ 1 \\ 0 \end{pmatrix}.$$

Let

$$f_\beta = \begin{pmatrix} -SI - \eta SH \\ 0 \\ SI \\ \eta SH \end{pmatrix}.$$

Then

$$f_\beta(E_{03}, \beta^*) = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}, \quad W^T \cdot f_\beta(E_{03}, \beta^*) = 0.$$

The derivative of the Jacobian with respect to β is

$$Df_\beta = \begin{pmatrix} -I - \eta H & 0 & -S & -\eta S \\ 0 & 0 & 0 & 0 \\ I & 0 & S & 0 \\ \eta H & 0 & 0 & \eta S \end{pmatrix},$$

whose value at the equilibrium is

$$Df_{\beta}(E_{03}, \beta^*) = \begin{pmatrix} 0 & 0 & -S_{03} & -\eta S_{03} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & S_{03} & 0 \\ 0 & 0 & 0 & \eta S_{03} \end{pmatrix}.$$

Hence

$$\begin{aligned} W^T \cdot Df_{\beta}(E_{03}, \beta^*) \cdot V &= (0 \ 0 \ 1 \ 0) \begin{pmatrix} 0 & 0 & -S_{03} & -\eta S_{03} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & S_{03} & 0 \\ 0 & 0 & 0 & \eta S_{03} \end{pmatrix} \begin{pmatrix} V_1 \\ V_2 \\ V_3 \\ V_4 \end{pmatrix} \\ &= S_{03} V_3 \neq 0. \end{aligned}$$

Moreover,

$$D^2 f(E_{03}, \beta^*) \cdot (V, V) = \begin{pmatrix} -2\beta^*(V_1 V_3 + \eta V_1 V_4) \\ 0 \\ 2(\beta^* V_1 V_3 - \sigma V_3 V_4) \\ 2(\eta \beta^* V_1 V_4 + \sigma V_3 V_4) \end{pmatrix},$$

and thus

$$W^T \cdot D^2 f(E_{03}, \beta^*) \cdot (V, V) = 2(\beta^* V_1 V_3 - \sigma V_3 V_4).$$

Since $V_3 = 1$ and $V_4 = 0$, this reduces to

$$W^T \cdot D^2 f(E_{03}, \beta^*) \cdot (V, V) = -\frac{2\beta^*(\rho + d)(d + \alpha)}{d(d + \rho + \delta)} < 0.$$

Therefore the system undergoes a transcritical bifurcation at E_{03} when $\beta = \beta^*$ and $R_{0HS} < 1 + \frac{\delta}{\rho + d}$. $\square$

Theorem 3.8. *The system undergoes a transcritical bifurcation at E_{03} if and only if*

$$R_{0IS} < 1 + \frac{\delta}{\rho + d} \quad \text{and} \quad \beta = \beta_0.$$

Proof. When

$$R_{0HS} = 1 + \frac{\delta}{\rho + d},$$

take

$$\beta_0 = \frac{d + \lambda\alpha}{\eta S_{03}} = \frac{d(d + \lambda\alpha)(d + \rho + \delta)}{\Lambda\eta(d + \rho)}.$$

The right eigenvector corresponding to $\lambda_4 = 0$ is

$$V = \begin{pmatrix} -\frac{(d + \lambda\alpha)(\rho + d)}{d(d + \rho + \delta)} \\ -\frac{d(d + \rho + \delta)}{\delta(d + \lambda\alpha)} \\ 0 \\ 1 \end{pmatrix} = \begin{pmatrix} V_1 \\ V_2 \\ V_3 \\ V_4 \end{pmatrix},$$

and the corresponding left eigenvector is

$$\mathbf{W} = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 1 \end{pmatrix}.$$

Since

$$f_{\beta}(E_{03}, \beta_0) = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \end{pmatrix},$$

we have

$$\mathbf{W}^T \cdot f_{\beta}(E_{03}, \beta_0) = 0.$$

Moreover,

$$Df_{\beta}(E_{03}, \beta_0) = \begin{pmatrix} 0 & 0 & -S_{03} & -\eta S_{03} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & S_{03} & 0 \\ 0 & 0 & 0 & \eta S_{03} \end{pmatrix},$$

and

$$\begin{aligned} \mathbf{W}^T \cdot Df_{\beta}(E_{03}, \beta_0) \cdot \mathbf{V} &= (0 \ 0 \ 0 \ 1) \begin{pmatrix} 0 & 0 & -S_{03} & -\eta S_{03} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & S_{03} & 0 \\ 0 & 0 & 0 & \eta S_{03} \end{pmatrix} \begin{pmatrix} V_1 \\ V_2 \\ V_3 \\ V_4 \end{pmatrix} \\ &= \eta S_{03} \neq 0. \end{aligned}$$

The second-order term is

$$D^2 f(E_{03}, \beta_0) \cdot (\mathbf{V}, \mathbf{V}) = \begin{pmatrix} -2\beta_0(V_1 V_3 + \eta V_1 V_4) \\ 0 \\ 2(\beta_0 V_1 V_3 - \sigma V_3 V_4) \\ 2(\eta \beta_0 V_1 V_4 + \sigma V_3 V_4) \end{pmatrix}.$$

Since $V_3 = 0$ and $V_4 = 1$, this becomes

$$D^2 f(E_{03}, \beta_0) \cdot (\mathbf{V}, \mathbf{V}) = \begin{pmatrix} -2\beta_0 \eta V_1 \\ 0 \\ 0 \\ 2\eta \beta_0 V_1 \end{pmatrix},$$

and hence

$$\mathbf{W}^T \cdot D^2 f(E_{03}, \beta_0) \cdot (\mathbf{V}, \mathbf{V}) = 2\eta \beta_0 V_1 = -\frac{2(d + \lambda \alpha)^2}{\Lambda} \neq 0.$$

Therefore the system undergoes a transcritical bifurcation at E_{03} when $R_{0IS} < 1 + \frac{\delta}{\rho + d}$ and $\beta = \beta_0$. $\square$

Theorem 3.9. *The system undergoes a codimension-three Bogdanov-Takens (BT) bifurcation at E_{03} if and only if*

$$\eta = \eta^* \quad \text{and} \quad \beta = \beta^*.$$

Proof. When

$$R_{0IS} = 1 + \frac{\delta}{\rho + d} \quad \text{and} \quad R_{0HS} = 1 + \frac{\delta}{\rho + d},$$

define

$$\eta^* = \frac{d(d + \lambda\alpha)(d + \rho + \delta)}{\Lambda\beta(\rho + d)}, \quad \beta^* = \frac{d(d + \alpha)(d + \rho + \delta)}{\Lambda(\rho + d)}, \quad \mu = \begin{pmatrix} \eta^* \\ \beta^* \end{pmatrix}.$$

Let

$$F(u, \mu) = \begin{pmatrix} \sigma HI + \eta\beta SH - (d + \lambda\alpha)H \\ \beta SI - \sigma HI - (d + \alpha)I \\ \delta S - (\rho + d)M \\ \Lambda - (\delta + d)S + \rho M - \beta SI - \eta\beta SH \end{pmatrix}.$$

Expanding $F(u, \mu)$ at (u_0, μ_0) gives

$$\begin{aligned} F(u, \mu) &= DF(u_0, \mu_0)(u - u_0) + F_\mu(u_0, \mu_0)(\mu - \mu_0) + \frac{1}{2}D^2F(u_0, \mu_0)(u - u_0, u - u_0) \\ &\quad + DF_{\mu\mu}(u_0, \mu_0)(u - u_0)(\mu - \mu_0) + \cdots. \end{aligned}$$

The Jacobian matrix is

$$A = DF(u_0, \mu_0) = \begin{pmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & -(\rho + d) & \delta \\ -\eta\beta^* S_{03} & -\beta^* S_{03} & \rho & -\delta - d \end{pmatrix},$$

and

$$F_\mu(u_0, \mu_0) = \begin{pmatrix} 0 & 0 \\ 0 & 0 \\ 0 & 0 \\ 0 & 0 \end{pmatrix}.$$

Let T_1 and T_2 be generalized eigenvectors of A associated with the eigenvalue $\lambda = 0$, and let $T = (T_1, T_2, T_0)$, where T_0 is the generalized eigenvector of

$$\begin{pmatrix} -d & 0 \\ 0 & -(\rho + \delta + d) \end{pmatrix}.$$

Let

$$T^{-1} = \begin{pmatrix} Z_1^T \\ Z_2^T \\ Z_0^T \end{pmatrix},$$

with $Z_2^T A = 0$ and $Z_1^T A = Z_2^T$. Then

$$T^{-1}AT = \begin{pmatrix} 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & -d & 1 \\ 0 & 0 & 0 & -(\rho + \delta + d) \end{pmatrix}.$$

Furthermore,

$$a = \frac{1}{2} Z_1^T D^2 F(u_0, \mu_0)(T_1, T_1) = 0,$$

and

$$b = Z_1^T D^2 F(u_0, \mu_0)(T_1, T_2) + Z_2^T D^2 F(u_0, \mu_0)(T_1, T_1) = (\eta + 1)\beta.$$

Since $a = 0$ and $b \neq 0$, the system undergoes a codimension-three BT bifurcation at E_{03} when $\eta = \eta^*$ and $\beta = \beta^*$. $\square$

4. Existence and stability of the positive equilibrium and effects of the temporary-protection parameters δ, ρ

4.1. Existence and stability of the positive equilibrium

When chestnut blight and the hypovirus coexist in the orchard, system (2.1) admits a positive equilibrium $E^* = (S^*, M^*, I^*, H^*)$, given by

$$E^* = \left(\frac{\Lambda\sigma(\rho + d)}{d\sigma(\delta + \rho + d) + \beta(\rho + d)[(d + \lambda\alpha) - \eta(d + \alpha)]}, \frac{\delta S^*}{\rho + d}, \frac{(d + \lambda\alpha) - \eta\beta S^*}{\sigma}, \frac{\beta S^* - (d + \alpha)}{\sigma} \right).$$

We next investigate the existence conditions and global stability of this equilibrium.

Theorem 4.1. *If $R_{0HS} < R_{0IS}$ and*

$$R_{0HS} + \frac{\sigma\Lambda}{(d + \alpha)(d + \lambda\alpha)} R_{0HS} < \frac{\sigma\Lambda}{(d + \alpha)(d + \lambda\alpha)} \left(1 + \frac{\delta}{\rho + d} \right) + R_{0IS} < R_{0HS} + \frac{\sigma\Lambda}{(d + \alpha)(d + \lambda\alpha)} R_{0IS},$$

the positive equilibrium E^* exists and is unique.

Proof. The existence of E^* requires all coordinates to be positive, namely

$$\begin{cases} d\sigma(\rho + d + \delta) + \beta(\rho + d)[(d + \lambda\alpha) - \eta(d + \alpha)] > 0, \\ d\sigma(d + \lambda\alpha)(\rho + d + \delta) + \beta(\rho + d)(d + \lambda\alpha)[(\lambda\alpha + d) - \eta(d + \lambda\alpha)] - \eta\beta\sigma\Lambda(\rho + d) > 0, \\ \sigma\Lambda\beta(\rho + d) - d\sigma(\alpha + d)(\rho + d + \delta) - \beta(\rho + d)(\alpha + d)[(d + \lambda\alpha) - \eta(\alpha + d)] > 0. \end{cases}$$

Under the condition $R_{0HS} < R_{0IS}$, we have

$$\frac{\eta}{d + \lambda\alpha} < \frac{1}{d + \alpha},$$

that is,

$$\frac{\eta\beta\sigma\Lambda(\rho + d)}{d + \lambda\alpha} < \frac{\beta\sigma\Lambda(\rho + d)}{d + \alpha}.$$

Hence

$$\eta\beta(\rho + d)(\alpha + d) + \frac{\eta\beta\sigma\Lambda(\rho + d)}{d + \lambda\alpha} < d\sigma(\rho + d + \delta) + \beta(\rho + d)(d + \lambda\alpha) < \frac{\beta\sigma\Lambda(\rho + d)}{d + \alpha} + \eta\beta(\rho + d)(\alpha + d).$$

Equivalently,

$$\frac{\Lambda\eta\beta}{d(d+\alpha)} + \frac{\eta\beta\delta\Lambda^2}{d(d+\alpha)(d+\lambda\alpha)^2} < \frac{\sigma\Lambda(\rho+d+\delta)}{(d+\alpha)(d+\lambda\alpha)(\rho+d)} + \frac{\Lambda\beta}{d(d+\alpha)} < \frac{\Lambda\eta\beta}{d(d+\lambda\alpha)} + \frac{\sigma\Lambda^2\beta}{d(d+\lambda\alpha)^2(d+\lambda\alpha)}.$$

After further simplification, we obtain

$$R_{0HS} + \frac{\sigma\Lambda}{(d+\alpha)(d+\lambda\alpha)}R_{0HS} < \frac{\sigma\Lambda}{(d+\alpha)(d+\lambda\alpha)}\left(1 + \frac{\delta}{\rho+d}\right) + R_{0IS} < R_{0HS} + \frac{\sigma\Lambda}{(d+\alpha)(d+\lambda\alpha)}R_{0IS}.$$

Under this condition, (S^*, M^*, I^*, H^*) exists and is unique. If $R_{0HS} \geq R_{0IS}$,

$$\frac{\eta\beta\sigma\Lambda(\rho+d)}{d+\lambda\alpha} \geq \frac{\beta\sigma\Lambda(\rho+d)}{d+\alpha},$$

and no positive equilibrium exists.

To verify that $E^* \in D$, note that under the existence condition,

$$S^* > 0, \quad M^* > 0, \quad I^* > 0, \quad H^* > 0.$$

Moreover,

$$H^* \geq 0 \iff S^* \geq \frac{d+\alpha}{\beta}, \quad I^* \geq 0 \iff S^* \leq \frac{d+\lambda\alpha}{\eta\beta}.$$

Hence all coordinates are nonnegative. Finally,

$$S^* + M^* + I^* + H^* = \frac{\Lambda - \alpha(I^* + \lambda H^*)}{d} \leq \frac{\Lambda}{d},$$

which implies $E^* \in D$. □

Theorem 4.2. If $R_{0HS} < R_{0IS}$ and

$$R_{0HS} + \frac{\sigma\Lambda}{(d+\alpha)(d+\lambda\alpha)}R_{0HS} < \frac{\sigma\Lambda}{(d+\alpha)(d+\lambda\alpha)}\left(1 + \frac{\delta}{\rho+d}\right) + R_{0IS} < R_{0HS} + \frac{\sigma\Lambda}{(d+\alpha)(d+\lambda\alpha)}R_{0IS},$$

the model is locally asymptotically stable at E^* .

Proof. The Jacobian matrix at E^* is

$$J_4 = \begin{pmatrix} -\delta - d - \beta I^* - \eta\beta H^* & \rho & -\beta S^* & -\eta\beta S^* \\ \delta & -(\rho+d) & 0 & 0 \\ \beta I^* & 0 & 0 & -\sigma I^* \\ \eta\beta H^* & 0 & \sigma H^* & 0 \end{pmatrix}. \quad (4.1)$$

Its characteristic polynomial is

$$\begin{aligned} &\lambda^4 + (\rho+d+x)\lambda^3 + [\eta\beta S^* \cdot \eta\beta H^* + \sigma I^* \cdot \sigma H^* + \beta I^* \cdot \beta S^* + x(\rho+d) - \rho\delta]\lambda^2 \\ &+ [\sigma I^* \cdot \sigma H^* x + (\rho+d)(\eta\beta S^* \cdot \eta\beta H^* + \sigma I^* \cdot \sigma H^* + \beta S^* \cdot \beta I^*)]\lambda + [x(\rho+d) - \beta\delta]\sigma I^* \cdot \sigma H^* = 0, \end{aligned}$$

where

$$x = \delta + d + \beta I^* + \eta \beta H^*.$$

Let

$$a = \beta S^*, \quad b = \beta I^*, \quad c = \beta H^*.$$

Then the Hurwitz determinants satisfy

$$\Delta_1 = \rho + d + x > 0.$$

Furthermore,

$$\begin{aligned} \Delta_2 &= [(\rho + d) + x] [\eta a \cdot \eta c + \sigma I^* \cdot \sigma H^* + ab + x(\rho + d) - \rho \delta] \\ &\quad - [\sigma I^* \cdot \sigma H^* x + (\rho + d)(\eta a \cdot \eta c + \sigma I^* \cdot \sigma H^* + ab)] \\ &= x(\rho + d)^2 - \rho \delta(\rho + d) + (\eta a \cdot \eta c + ab)x + x^2(\rho + d) - x\rho \delta. \end{aligned}$$

Because

$$\begin{aligned} x(\rho + d)^2 - \rho \delta(\rho + d) &= (\rho + d)[x(\rho + d) - \rho \delta] \\ &= (\rho + d)[(\delta + d + \beta + \eta c)(\rho + d) - \rho \delta] > 0, \end{aligned}$$

we have $\Delta_2 > 0$. Similarly,

$$\begin{aligned} \Delta_3 &= (\rho + d)^2(\eta a \cdot \eta c + ab)[x(\rho + d) - \rho \delta] + x^2 \sigma I^* \cdot \sigma H^*(\eta a \cdot \eta c + ab) \\ &\quad + x(\rho + d)(\eta a \cdot \eta c + ab)(\eta a \cdot \eta c + \sigma I^* \cdot \sigma H^* + ab) \\ &\quad + x(\rho + d)(\eta a \cdot \eta c + ab)[x(\rho + d) - \rho \delta] > 0, \end{aligned}$$

and

$$\Delta_4 = a_4 \cdot \Delta_3 = [x(\rho + d) - \rho \delta] \sigma I^* \cdot \sigma H^* \cdot \Delta_3 > 0.$$

Therefore E^* is locally asymptotically stable. □

4.2. Effects of δ and ρ on the model

4.2.1. Effect on the composite protection index $\Theta(\delta, \rho)$

To describe the joint effect of entry into and loss from the temporarily protected class, define

$$\Theta(\delta, \rho) = 1 + \frac{\delta}{\rho + d}.$$

The partial derivatives of Θ are

$$\frac{\partial \Theta}{\partial \delta} = \frac{1}{\rho + d} > 0, \quad \frac{\partial \Theta}{\partial \rho} = -\frac{\delta}{(\rho + d)^2} < 0.$$

Thus, increasing δ strengthens the temporary-protection effect, whereas increasing ρ weakens it by shortening the protection period.

4.2.2. Effect on the effective reproduction numbers R_{0IS} and R_{0HS}

At the disease-free equilibrium, the susceptible and temporarily protected components can be written as

$$S_{03} = \frac{\Lambda}{d\Theta}, \quad M_{03} = \frac{\Lambda}{d} \frac{\Theta - 1}{\Theta}.$$

The effective reproduction numbers in the presence of temporary protection are therefore

$$\mathcal{R}_I(\delta, \rho) = \frac{\beta S_{03}}{d + \alpha} = \frac{R_{0IS}}{\Theta}, \quad \mathcal{R}_H(\delta, \rho) = \frac{\eta \beta S_{03}}{d + \lambda \alpha} = \frac{R_{0HS}}{\Theta}.$$

Their sensitivities satisfy

$$\frac{\partial \mathcal{R}_I}{\partial \delta} = -\frac{R_{0IS}}{\Theta^2(\rho + d)} < 0, \quad \frac{\partial \mathcal{R}_H}{\partial \delta} = -\frac{R_{0HS}}{\Theta^2(\rho + d)} < 0,$$

and

$$\frac{\partial \mathcal{R}_I}{\partial \rho} = \frac{R_{0IS}\delta}{\Theta^2(\rho + d)^2} > 0, \quad \frac{\partial \mathcal{R}_H}{\partial \rho} = \frac{R_{0HS}\delta}{\Theta^2(\rho + d)^2} > 0.$$

Consequently, a larger δ decreases both effective reproduction numbers, whereas a larger ρ increases them.

4.2.3. Effect on the infected populations at the boundary equilibria

The existence conditions for the two nontrivial boundary equilibria become

$$E_{01} \text{ exists} \iff R_{0IS} > \Theta,$$

and

$$E_{02} \text{ exists} \iff R_{0HS} > \Theta.$$

The disease-free equilibrium is locally asymptotically stable if

$$\max \{\mathcal{R}_I(\delta, \rho), \mathcal{R}_H(\delta, \rho)\} < 1,$$

or equivalently,

$$\max \{R_{0IS}, R_{0HS}\} < \Theta.$$

Therefore, increasing δ enlarges the disease-free stability region, whereas increasing ρ reduces it.

4.2.4. Effect on the existence conditions of boundary equilibria E_{01} and E_{02}

The pathogen-only component of E_{01} can be rewritten as

$$I_{01} = \frac{\Lambda}{d + \alpha} - \frac{d}{\beta} \Theta,$$

and the temporarily protected component is

$$M_{01} = \frac{d + \alpha}{\beta} (\Theta - 1).$$

Hence

$$\frac{\partial I_{01}}{\partial \delta} = -\frac{d}{\beta(\rho + d)} < 0, \quad \frac{\partial I_{01}}{\partial \rho} = \frac{d\delta}{\beta(\rho + d)^2} > 0.$$

Similarly, for E_{02} ,

$$H_{02} = \frac{\Lambda}{d + \lambda\alpha} - \frac{d}{\eta\beta}\Theta,$$

and

$$M_{02} = \frac{d + \lambda\alpha}{\eta\beta}(\Theta - 1).$$

Therefore,

$$\frac{\partial H_{02}}{\partial \delta} = -\frac{d}{\eta\beta(\rho + d)} < 0, \quad \frac{\partial H_{02}}{\partial \rho} = \frac{d\delta}{\eta\beta(\rho + d)^2} > 0.$$

These results show that a larger entry rate into temporary protection reduces the equilibrium sizes of both boundary infected classes, whereas faster loss of protection has the opposite effect.

4.2.5. Effect on the transcritical bifurcation thresholds σ_{01} and σ_{02}

Define

$$K = (d + \lambda\alpha) - \eta(d + \alpha).$$

Under the biologically relevant condition

$$K > 0,$$

the hyperparasite-invasion eigenvalue at E_{01} is

$$\lambda_H^{(01)} = \sigma I_{01} - K.$$

The corresponding transcritical-bifurcation threshold is

$$\sigma_{01} = \frac{\beta(d + \alpha)K}{\Lambda\beta - d(d + \alpha)\Theta},$$

provided that

$$\Lambda\beta - d(d + \alpha)\Theta > 0.$$

Because

$$\frac{\partial \sigma_{01}}{\partial \Theta} = \frac{\beta d(d + \alpha)^2 K}{[\Lambda\beta - d(d + \alpha)\Theta]^2} > 0,$$

we obtain

$$\frac{\partial \sigma_{01}}{\partial \delta} > 0, \quad \frac{\partial \sigma_{01}}{\partial \rho} < 0.$$

Thus, increasing δ raises the hypovirus-transmission threshold required for invasion of the pathogen-only equilibrium. This occurs because temporary protection reduces the size of the pathogen-only class that serves as the substrate for hyperparasite transmission. At E_{02} , the pathogen-invasion eigenvalue can be written as

$$\lambda_I^{(02)} = \frac{K}{\eta} - \sigma H_{02}.$$

The corresponding threshold is

$$\sigma_{02} = \frac{\beta(d + \lambda\alpha)K}{\Lambda\eta\beta - d(d + \lambda\alpha)\Theta},$$

provided that

$$\Lambda\eta\beta - d(d + \lambda\alpha)\Theta > 0.$$

Its derivative is

$$\frac{\partial\sigma_{02}}{\partial\Theta} = \frac{\beta d(d + \lambda\alpha)^2 K}{[\Lambda\eta\beta - d(d + \lambda\alpha)\Theta]^2} > 0.$$

Consequently,

$$\frac{\partial\sigma_{02}}{\partial\delta} > 0, \quad \frac{\partial\sigma_{02}}{\partial\rho} < 0.$$

When both boundary equilibria exist, the positive coexistence equilibrium lies between the two transcritical thresholds:

$$\sigma_{01} < \sigma < \sigma_{02}.$$

Increasing δ shifts both transcritical thresholds toward larger values of σ , whereas increasing ρ shifts them toward smaller values. The width of the coexistence interval need not vary monotonically because the two thresholds generally have different parameter sensitivities. The pathogen-transmission threshold at the disease-free equilibrium is

$$\beta_I^*(\delta, \rho) = \frac{d(d + \alpha)}{\Lambda}\Theta.$$

4.2.6. Effect on the pathogen-transmission thresholds (β thresholds)

The corresponding threshold for the hyperparasite-associated infection pathway is

$$\beta_H^*(\delta, \rho) = \frac{d(d + \lambda\alpha)}{\Lambda\eta}\Theta.$$

Their sensitivities satisfy

$$\frac{\partial\beta_I^*}{\partial\delta} = \frac{d(d + \alpha)}{\Lambda(\rho + d)} > 0, \quad \frac{\partial\beta_H^*}{\partial\delta} = \frac{d(d + \lambda\alpha)}{\Lambda\eta(\rho + d)} > 0,$$

and

$$\frac{\partial\beta_I^*}{\partial\rho} = -\frac{d(d + \alpha)\delta}{\Lambda(\rho + d)^2} < 0, \quad \frac{\partial\beta_H^*}{\partial\rho} = -\frac{d(d + \lambda\alpha)\delta}{\Lambda\eta(\rho + d)^2} < 0.$$

Thus, increasing δ raises the pathogen-transmission rate required to destabilize the disease-free equilibrium, whereas increasing ρ lowers this threshold. At the simultaneous threshold,

$$\beta_{BT}^* = \frac{d(d + \alpha)}{\Lambda}\Theta,$$

and substitution into the second zero-eigenvalue condition gives

$$\eta_{BT}^* = \frac{d + \lambda\alpha}{d + \alpha}.$$

Therefore, δ and ρ shift the β -coordinate of the codimension point through Θ , while the corresponding value of η_{BT}^* remains unchanged after the simultaneous-threshold conditions are imposed.

4.2.7. Effect on the positive coexistence equilibrium

For the positive equilibrium, define

$$D^* = d\sigma\Theta + \beta K.$$

Then

$$\begin{aligned} S^* &= \frac{\Lambda\sigma}{D^*}, \\ M^* &= (\Theta - 1)S^*, \\ I^* &= \frac{(d + \lambda\alpha) - \eta\beta S^*}{\sigma}, \\ H^* &= \frac{\beta S^* - (d + \alpha)}{\sigma}. \end{aligned}$$

Differentiation with respect to Θ yields

$$\begin{aligned} \frac{\partial S^*}{\partial \Theta} &= -\frac{\Lambda d\sigma^2}{(D^*)^2} < 0, \\ \frac{\partial M^*}{\partial \Theta} &= \frac{\Lambda\sigma[d\sigma + \beta K]}{(D^*)^2} > 0, \\ \frac{\partial I^*}{\partial \Theta} &= \frac{\eta\beta\Lambda d\sigma}{(D^*)^2} > 0, \end{aligned}$$

and

$$\frac{\partial H^*}{\partial \Theta} = -\frac{\beta\Lambda d\sigma}{(D^*)^2} < 0.$$

Combining these results with the derivatives of Θ , we have

$$\frac{\partial S^*}{\partial \delta} < 0, \quad \frac{\partial M^*}{\partial \delta} > 0, \quad \frac{\partial I^*}{\partial \delta} > 0, \quad \frac{\partial H^*}{\partial \delta} < 0,$$

whereas

$$\frac{\partial S^*}{\partial \rho} > 0, \quad \frac{\partial M^*}{\partial \rho} < 0, \quad \frac{\partial I^*}{\partial \rho} < 0, \quad \frac{\partial H^*}{\partial \rho} > 0.$$

The increase of I^* with δ in the coexistence regime does not imply that temporary protection promotes pathogen transmission. Rather, increasing δ reduces both S^* and H^* , and the decrease in the hyperparasite-associated class weakens the conversion pathway from I to H . Consequently, the equilibrium composition may shift from the hyperparasite-associated class toward the pathogen-only class even while the total infection pressure decreases. Indeed, if

$$0 < \eta < 1,$$

then

$$\frac{\partial(I^* + H^*)}{\partial \Theta} = -\frac{\beta(1 - \eta)\Lambda d\sigma}{(D^*)^2} < 0.$$

Therefore,

$$\frac{\partial(I^* + H^*)}{\partial \delta} < 0, \quad \frac{\partial(I^* + H^*)}{\partial \rho} > 0.$$

Thus, stronger temporary protection can reduce the total equilibrium population associated with infection, although it may simultaneously reduce the relative contribution of hypovirus-associated hosts. This result illustrates that temporary protection and hypovirus transmission interact through a nonmonotone ecological tradeoff.

5. Numerical simulations

To illustrate the thresholds and bifurcation phenomena obtained in the theoretical analysis, we present numerical simulations focusing on three groups of parameters. First, σ is used as the bifurcation parameter near the boundary equilibria E_{01} and E_{02} . Second, the stability regions near the codimension point are investigated in the (β, η) plane. Third, the effects of the temporary-protection parameters δ and ρ are examined through their influence on the effective reproduction numbers, bifurcation thresholds, and equilibrium populations. The numerical analysis is intended to illustrate the mathematical consequences of a finite protection period and does not correspond to a specific fungicide, dosage, or treatment schedule.

We first take σ as the bifurcation parameter to study the transcritical bifurcation near the hyperparasite-free boundary equilibrium E_{01} and its dynamical manifestation; the result is shown in Figure 2.

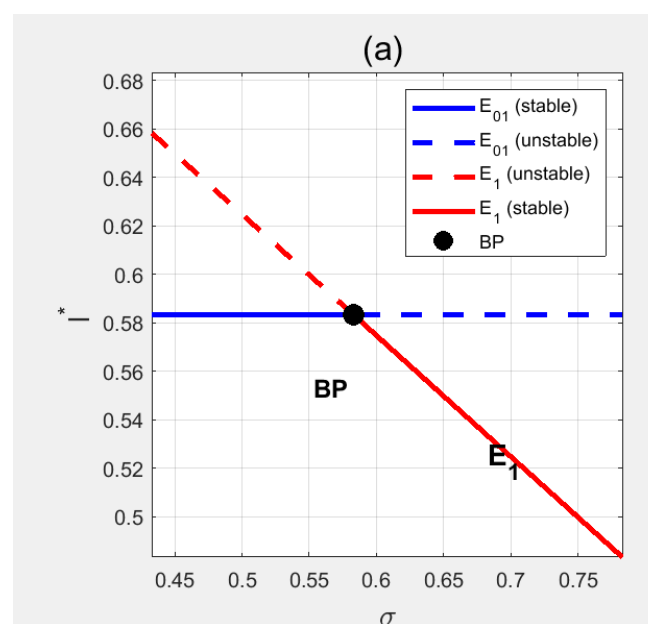


Figure 2. Transcritical bifurcation at E_{01} .

Table 2. Parameter values used for the one-parameter bifurcation analysis near E_{01} .

Figure	Λ	δ	d	ρ	β	η	α	λ	Varied parameter
Figures 2–5	1	0.2	0.2	0.1	0.8	0.5	0.8	0.8	$\sigma \in [0.01, 1]$

Figure 2 shows that when $\sigma < \sigma_c$, the boundary equilibrium E_{01} (blue branch E) is stable, whereas the corresponding endemic branch E_1 is unstable. When $\sigma > \sigma_c$, the two branches exchange stability:

E_{01} loses stability and E_1 becomes a stable attractor. Biologically, when $\sigma < \sigma_c$, the long-term state of the system is one in which hosts infected by chestnut blight persist ($I > 0$), whereas the hypovirus class eventually disappears ($H = 0$). When $\sigma > \sigma_c$, the long-term state becomes one in which both pathogen-infected hosts ($I > 0$) and hypovirus-associated hosts ($H > 0$) persist. Thus Figure 2 indicates that below the threshold the hypovirus cannot spread in the environment, whereas above the threshold it can successfully infect the chestnut blight fungus and induce coexistence among susceptible hosts, temporarily immune hosts, parasitized hosts, and hyperparasitized hosts.

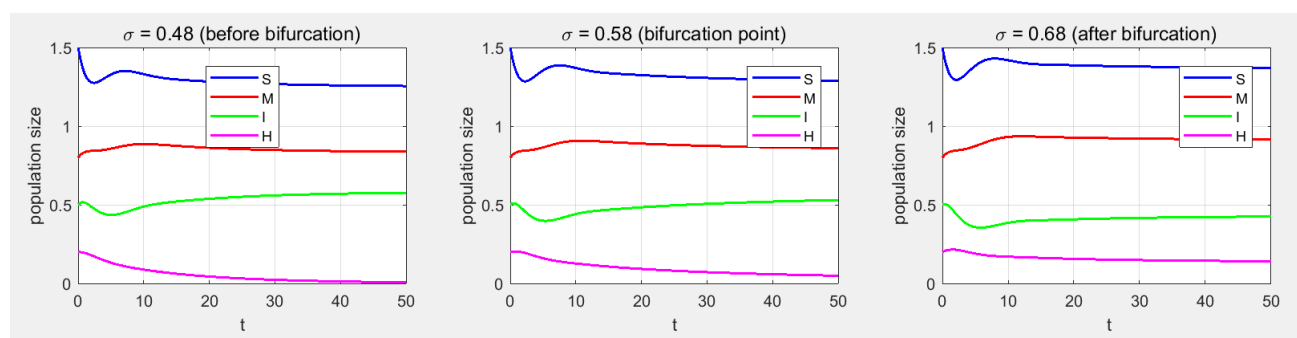


Figure 3. Time-series plots near E_{01} for different bifurcation regimes.

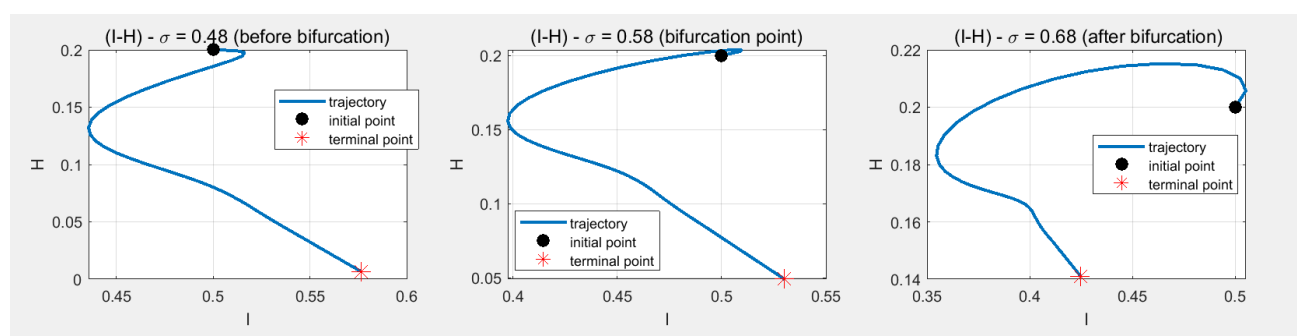


Figure 4. Phase portraits near E_{01} for different bifurcation regimes.

Figures 3 and 4 correspond to the three cases $\sigma < \sigma_c$, $\sigma = \sigma_c$, and $\sigma > \sigma_c$. The black dots and red stars denote the initial and terminal points in the phase portraits, respectively. Before the bifurcation, trajectories approach a boundary attractor ($H \rightarrow 0$). After the bifurcation, they approach an interior attractor ($H \rightarrow H^*$). And near the critical value, a clear critical slowing-down phenomenon appears. The biological interpretation is as follows. When σ is below the threshold, the number of hyperparasitized hosts decreases gradually even if the initial value is relatively large, and the system finally returns to a steady state with only parasitized hosts, with phase trajectories ending on the boundary $H = 0$. At the critical value, the decline of H becomes markedly slower. This means that the system recovers more slowly from hyperparasite invasion, thereby creating conditions for the long-term persistence of hyperparasitized hosts. When σ is above the threshold, the hyperparasitized population stabilizes at a clearly positive level, the parasitized host population decreases relative to its initial state, and the temporarily immune class becomes larger. One may also observe a peak in the

temporarily immune class and a trough in the parasitized class. This suggests that the initial chemical treatment is not yet sufficiently effective against chestnut blight, although the whole system gradually stabilizes over time.

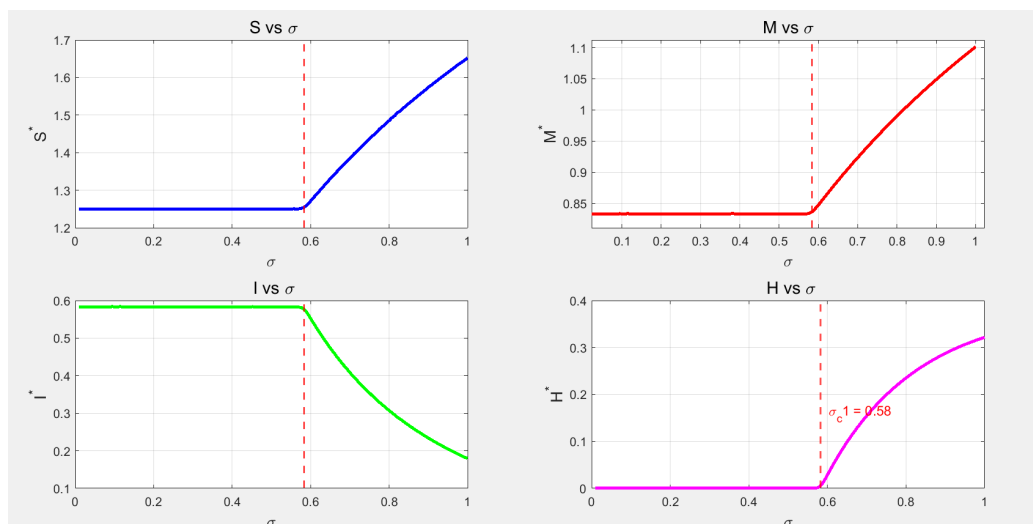


Figure 5. Population levels as σ varies.

Figure 5 shows that when σ is below the threshold, hyperparasitized hosts cannot persist in the environment, indicating that the infection rate of the hypovirus is too low for successful long-term establishment. Once σ exceeds the threshold, the system enters a regime in which the hyperparasitized class persists permanently, and the larger σ is, the higher the steady-state level of this class becomes. A further observation is that, as σ increases beyond the threshold, the number of parasitized hosts declines markedly while the number of susceptible hosts rises again. The reason is that stronger hypovirus infectivity reduces the parasitized class. This weakens the infection pathway of chestnut blight to susceptible hosts (βSI) and in turn increases the equilibrium level of susceptible hosts.

In summary, σ acts as a key coupling strength governing successful hypovirus invasion and introduces a clear invasion threshold. When $\sigma < \sigma_c$, the long-term system state is chestnut blight only, without hypovirus. When $\sigma > \sigma_c$, a transcritical bifurcation occurs and the system moves to an endemic state in which chestnut blight and the hypovirus coexist, with critical slowing down appearing near the bifurcation point.

We next investigate the transcritical bifurcation near the other boundary equilibrium E_{02} (where the pathogen-only class is eliminated) so as to describe the mechanism by which sufficiently large σ may drive the system from coexistence to the elimination of chestnut blight.

The corresponding numerical result is shown in Figure 6. When $\sigma < \sigma_c$, the endemic equilibrium E^* is stable while the boundary equilibrium E_{02} is unstable. At $\sigma = \sigma_c$, E^* and E_{02} exchange stability. When $\sigma > \sigma_c$, E_{02} becomes stable and E^* becomes unstable. Biologically, when $\sigma < \sigma_c$, even a small invasion of chestnut blight can still infect healthy chestnut trees rapidly and persist for a long time. In this case, biological control can only alleviate the disease or lead to coexistence, but cannot eradicate it completely. When $\sigma > \sigma_c$, hyperparasitic biological control becomes dominant and chestnut blight is gradually eliminated.

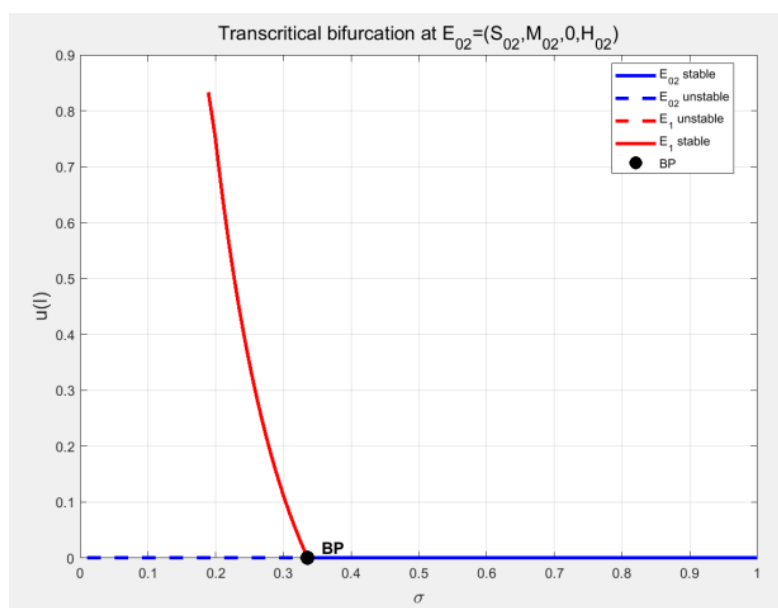


Figure 6. Transcritical bifurcation at the boundary equilibrium E_{02} .

Table 3. Parameter values used for the one-parameter bifurcation analysis near E_{02} .

Figure	Λ	δ	d	ρ	β	η	α	λ	Varied parameter
Figures 6–9	1	0.2	0.2	0.1	0.8	0.5	0.6	0.6	$\sigma \in [0.01, 1]$

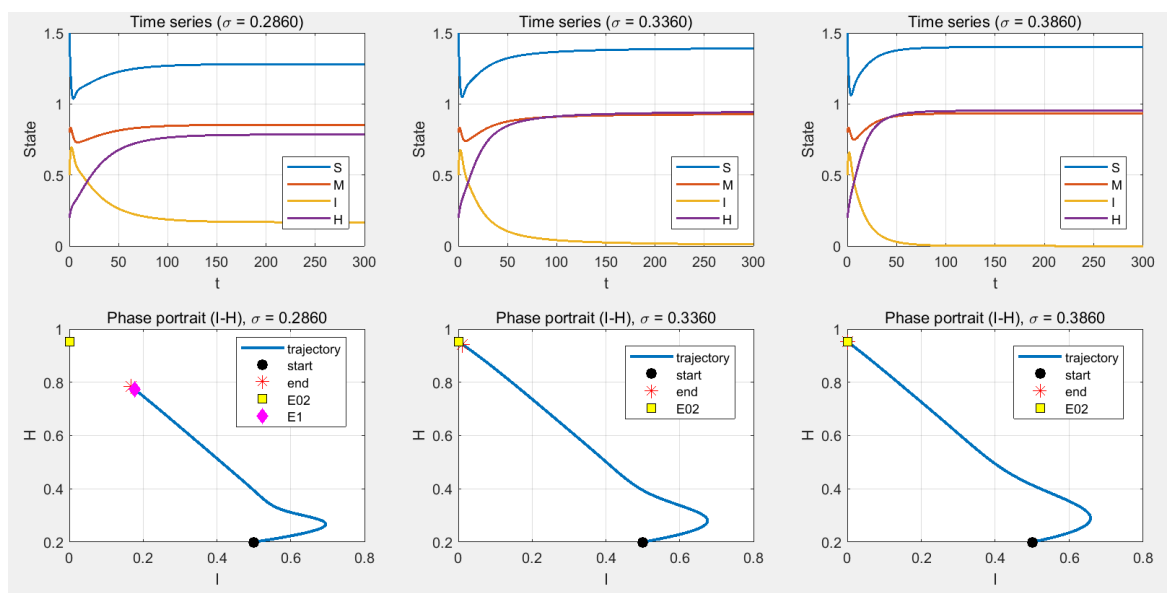


Figure 7. Time series and trajectories in the I - H plane.

Figure 7 shows that when σ is below the threshold σ_c , $I(t)$ does not vanish but converges to a positive steady state corresponding to the coexistence equilibrium E^* . The terminal point of the phase trajectory therefore lies in the interior region $I > 0$, $H > 0$. When σ is close to σ_c , the convergence

rate decreases, again indicating critical slowing down. When σ exceeds σ_c , $I(t)$ decays to 0, while $H(t)$ converges to H_{02} . In this case, the terminal point lies on the boundary attractor E_{02} . Biologically, when $\sigma < \sigma_c$, the hyperparasite (hypovirus) can suppress chestnut blight to some extent but cannot eradicate it, so the disease persists in the long run. When $\sigma \approx \sigma_c$, the number of diseased chestnut trees decreases and the hypovirus infection process against chestnut blight is prolonged. When $\sigma > \sigma_c$, hypovirus-based biological control becomes effective and chestnut blight is gradually eliminated.

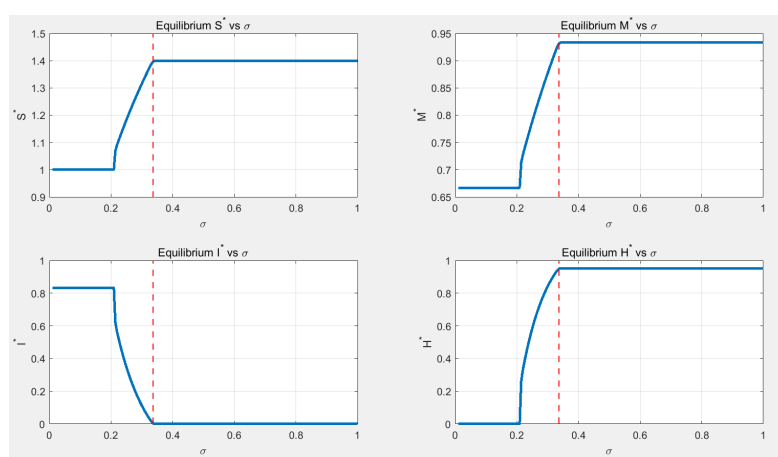


Figure 8. Population levels as σ varies.

Figure 8 shows that when σ is below the threshold, $H \approx 0$ and I remains at a relatively high level. This indicates that the transmission-conversion rate of the hypovirus is insufficient to achieve effective control, so chestnut blight remains endemic. When σ takes intermediate values (still below the threshold), H increases, I decreases, and both S and M increase, showing that biological control begins to take effect and the disease is alleviated, although not eradicated. When σ exceeds the threshold, H increases and converges to the steady state H_{02} , whereas I decreases to 0, meaning that the biological control agent is sufficiently effective to remove chestnut blight in the long run.

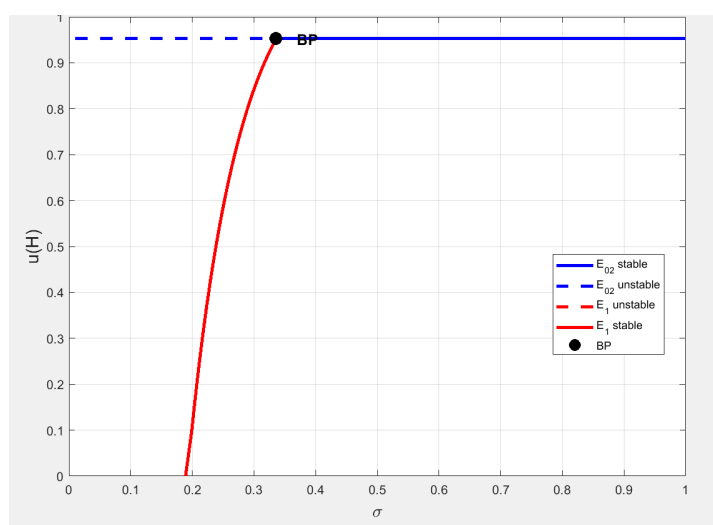


Figure 9. Bifurcation diagram of $u(H)$ with respect to σ .

Figure 9 plots H against σ . When $\sigma > \sigma_c$, the system ultimately stabilizes at $H = H_{02}$, so the hypovirus can maintain a long-term control effect. When $\sigma < \sigma_c$, H still exists, but the system remains in a coexistence state between the hypovirus and chestnut blight.

In summary, when $\sigma < \sigma_c$, the system remains for a long time in a coexistence state between chestnut blight and the hypovirus, while chestnut blight still dominates the population dynamics. When $\sigma > \sigma_c$, the hypovirus gradually suppresses pathogen transmission and eventually eliminates chestnut blight, thereby achieving the core goal of biological control.

Beyond the one-parameter bifurcation analysis, the system may also exhibit higher-codimension bifurcation structures under multiparameter coupling. We choose β and η as bifurcation parameters and plot the phase portrait, eigenvalue contours, and stability regions near the Bogdanov–Takens bifurcation point, as shown in Figure 10. Figure 10(a) shows that different initial conditions can lead to different asymptotic outcomes and that the initial value of H influences the trajectory. This sensitivity is a property of the model and does not specify an inoculation amount for practical use.

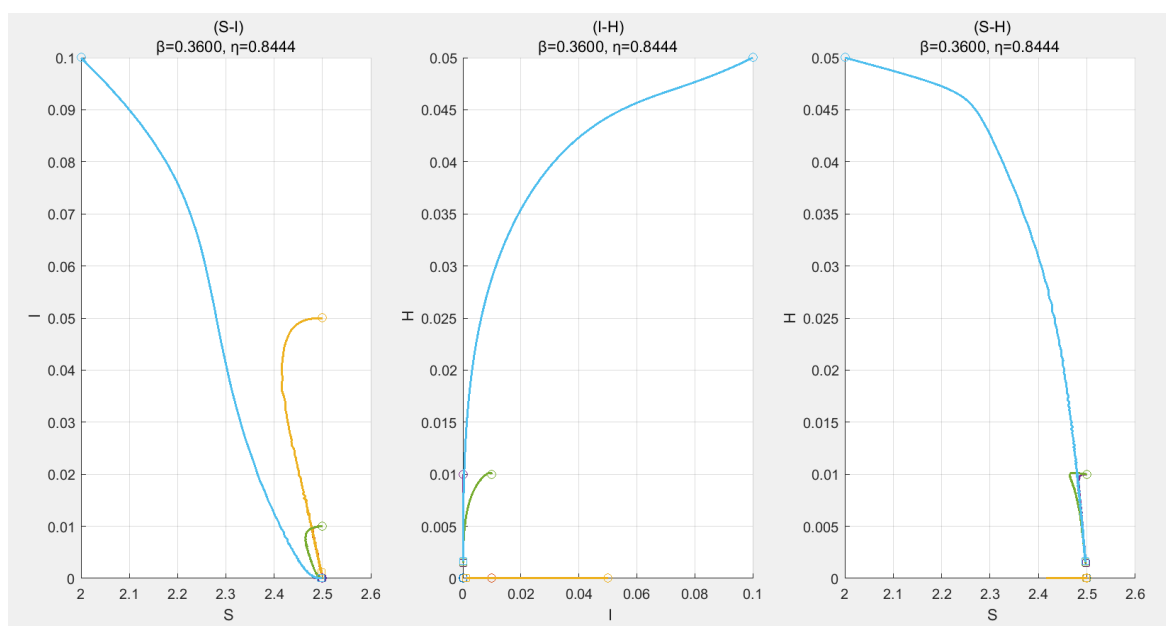
Figure 10(b) shows that when the eigenvalues λ_3 and λ_4 simultaneously approach or equal zero, the system reaches the critical state corresponding to the BT bifurcation point. When $\beta \approx \beta^*$, the model lies near the threshold between decay and persistence of the focal pathogen. If $\beta < \beta^*$, the pathogen-infected classes tend to decline; if $\beta > \beta^*$, persistence or rapid growth becomes possible within the model.

The parameter-space analysis in Figure 10(c) indicates that when β and η fall in the yellow region, the disease-free equilibrium is stable, whereas parameter combinations in the purple region destabilize that equilibrium. The colored regions are mathematical stability regions. Translating them into orchard practices would require empirical estimates linking particular interventions to β and η , together with field validation; such a translation is outside the scope of this study.

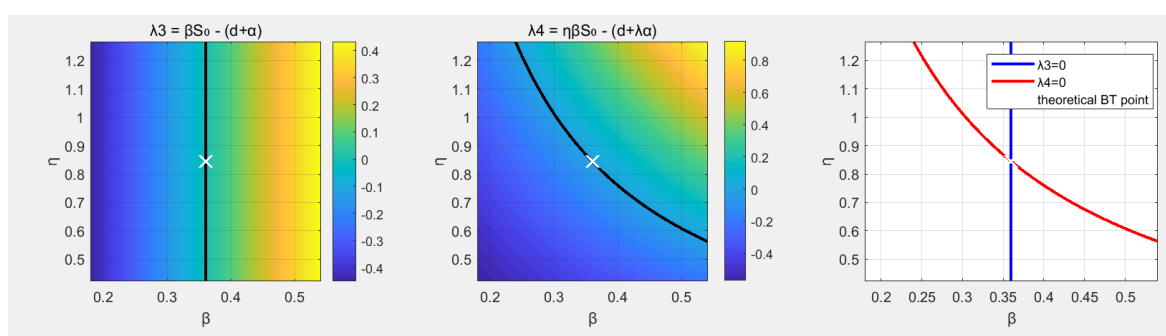
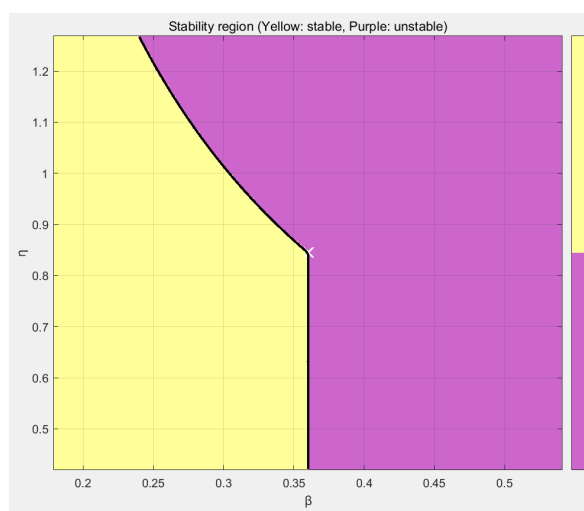
When the system is in the coexistence regime, Figure 11 illustrates the joint effects of δ and σ . Here δ is the transition rate from the susceptible class to the temporarily protected class, rather than a direct measure of fungicide concentration. Increasing δ transfers more hosts into class M and reduces the equilibrium susceptible population. At the same time, the reduction in pathogen availability may increase the value of σ required for successful establishment of the hyperparasite. The simulations are consistent with the analytical threshold

$$\sigma_{01} = \frac{\beta(d + \alpha) [(d + \lambda\alpha) - \eta(d + \alpha)]}{\Lambda\beta - d(d + \alpha) \left(1 + \frac{\delta}{\rho + d}\right)}. \quad (5.1)$$

For fixed ρ , this threshold increases with δ . Thus, stronger temporary protection may directly suppress infection while simultaneously reducing the pathogen-only population through which the hypovirus spreads. This produces a tradeoff between direct temporary protection and hyperparasite establishment. Within the positive-equilibrium region, increasing δ generally increases M^* and decreases S^* and H^* . The pathogen-only component I^* may increase because the conversion pathway from I to H becomes weaker as H^* declines. Nevertheless, when $0 < \eta < 1$, the total infection-associated equilibrium population $I^* + H^*$ decreases with δ . These results describe equilibrium redistribution within the mathematical model and should not be interpreted as evidence for a particular chemical dose or treatment sequence.



(a) Phase portrait near the BT point.

(b) Contours of λ_3 and λ_4 and their intersection.

(c) Stability region (yellow: stable; purple: unstable).

Figure 10. Characteristics of the BT bifurcation and analysis of stability regions.

Table 4. Parameter values used for the two-parameter analysis near the codimension point.

Figure	Λ	δ	d	ρ	σ	α	λ	Varied parameters
Figure 10	1	0.5	0.2	0.3	0.9	0.7	0.8	(β, η)

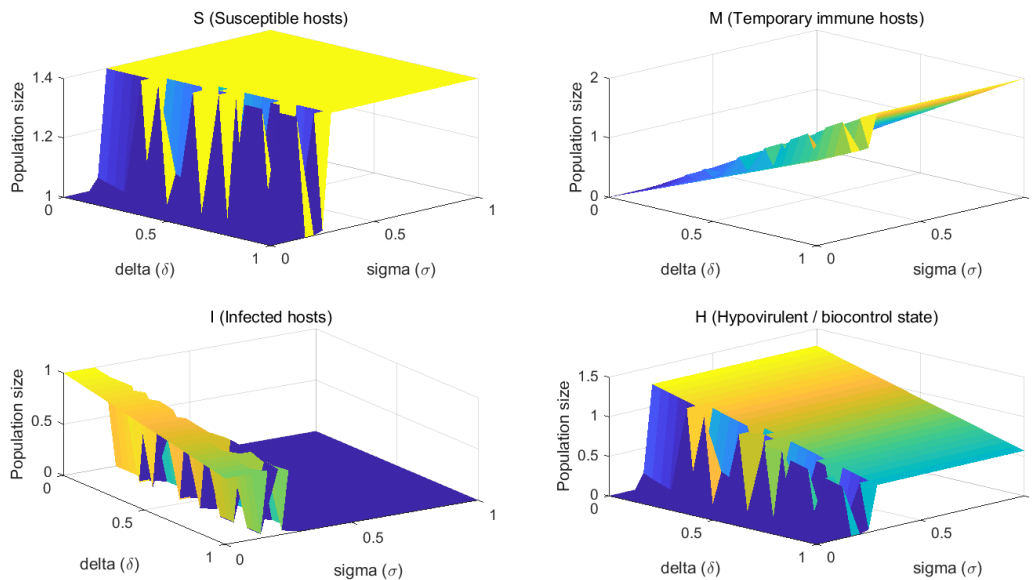
**Figure 11.** Variation of different host populations with δ and σ (3D surfaces).**Table 5.** Parameter values used for the joint (δ, σ) -effect simulations.

Figure	Λ	ρ	d	β	η	α	λ	Varied parameters
Figure 11	1	0.5	0.2	0.8	0.5	0.6	0.6	(δ, σ)

Taken together, under ideal conditions, the data and figures suggest the following management implication. If hyperparasite-based biological control is to be applied after preliminary chemical treatment in a chestnut orchard, chestnut varieties with relatively strong disease resistance should be selected, and chemicals of moderate intensity should be used. In addition, hypoviruses whose infectivity exceeds the threshold should be introduced as early as possible during the outbreak to achieve effective chemical-biological synergy.

The simulations also confirm that the intrinsic disease resistance and chemical response of chestnut trees significantly regulate the population dynamics. The effect of the hypovirus is not simply a monotone enhancement, but is constrained by a threshold governed by key parameters such as σ . When σ does not cross the threshold, the system may remain in a state of chestnut blight only. After the threshold is crossed, coexistence and disease alleviation become possible, and when σ is sufficiently large, complete elimination of chestnut blight may even occur. The numerical results show that increasing σ can change the system outcome substantially, while decreasing β can push the system back into a safer stable region (the stable region shown in Figure 10(c)). Therefore, under limited resources, management efforts should prioritize two tasks: improving the effective spread of

the hypovirus and reducing the basic transmission potential of the disease. The synergy between δ and σ indicates that moderate chemical control can create favorable conditions for establishing biological control. However, overly strong, overly early, or excessive chemical treatment may weaken the basis for hypovirus spread and induce more complicated risks.

In addition, the environmental adaptability of the hypovirus differs geographically. In some regions, climate and soil conditions may inhibit virus survival and reproduction within the chestnut blight fungus, or interfere with the interaction between the two, thereby reducing biological control efficacy. Different chestnut varieties may respond heterogeneously to both the pathogen and the hypovirus. Chestnut blight nevertheless remains a useful biological example of a host–pathogen–hyperparasite interaction. The performance of CHV1 differs among regions because vegetative compatibility diversity, host density, and pathogen-population structure can restrict horizontal virus transmission [18]. These empirical complications emphasize that the parameter σ is an idealized transmission coefficient rather than a directly transferable field value. Environmental conditions and host genotype may further alter hyperparasite survival, transmission, and effects on the pathogen. Such factors are not represented explicitly in the present ordinary differential equation system. Together with the absence of an effective chemical treatment for chestnut blight, this limitation prevents direct use of the model as a chestnut-blight management plan.

6. Conclusions

In this study, we developed and analyzed a chestnut blight-inspired host–pathogen–hyperparasite model that includes susceptible, temporarily protected, pathogen-infected, and hyperparasite-associated host classes. The temporarily protected class represents an idealized finite period of reduced susceptibility following an external intervention. Susceptible hosts enter this class at rate δ and return to the susceptible class after protection is lost at rate ρ . This transition is intended to describe a temporary protective effect rather than permanent immunity or a chemical cure for chestnut blight.

The model analysis identified the conditions governing the existence and stability of the disease-free, pathogen-only, hyperparasite-associated, and positive coexistence equilibria. The effects of the temporary-protection parameters can be summarized by

$$\Theta(\delta, \rho) = 1 + \frac{\delta}{\rho + d}. \quad (6.1)$$

The effective reproduction numbers are given by

$$\mathcal{R}_I(\delta, \rho) = \frac{R_{0IS}}{\Theta(\delta, \rho)}, \quad \mathcal{R}_H(\delta, \rho) = \frac{R_{0HS}}{\Theta(\delta, \rho)}. \quad (6.2)$$

Consequently, increasing δ reduces both effective reproduction numbers and enlarges the parameter region in which the disease-free equilibrium is stable. In contrast, increasing ρ shortens the mean duration of temporary protection and increases the effective reproduction numbers.

The parameters δ and ρ also affect the existence of the boundary equilibria. The pathogen-only and hyperparasite-associated equilibria exist, respectively, when

$$R_{0IS} > 1 + \frac{\delta}{\rho + d} \quad (6.3)$$

and

$$R_{0HS} > 1 + \frac{\delta}{\rho + d}. \quad (6.4)$$

Thus, faster entry into the temporarily protected class increases the invasion threshold for both infection pathways, whereas faster loss of protection produces the opposite effect.

The analysis further showed that temporary protection changes the transcritical-bifurcation thresholds associated with the invasion and persistence of the hyperparasite. In particular, the critical hyperparasite-transmission threshold at the pathogen-only equilibrium is

$$\sigma_{01} = \frac{\beta(d + \alpha) [(d + \lambda\alpha) - \eta(d + \alpha)]}{\Lambda\beta - d(d + \alpha) \left(1 + \frac{\delta}{\rho + d}\right)}. \quad (6.5)$$

For biologically feasible parameter values, σ_{01} increases with δ and decreases with ρ . Therefore, temporary protection may reduce pathogen transmission directly while simultaneously reducing the pathogen-only population available for hyperparasite transmission. As a result, stronger temporary protection may increase the hyperparasite-transmission rate required for successful establishment.

At the positive equilibrium, increasing δ transfers more hosts into the temporarily protected class and decreases the susceptible and hyperparasite-associated populations. The pathogen-only population may increase because the reduction in the hyperparasite-associated class weakens the transition from pathogen-only infection to hyperparasite-associated infection. Nevertheless, when

$$0 < \eta < 1, \quad (6.6)$$

the total infection-associated equilibrium population satisfies

$$\frac{\partial(I^* + H^*)}{\partial\delta} < 0, \quad \frac{\partial(I^* + H^*)}{\partial\rho} > 0. \quad (6.7)$$

This result indicates that stronger temporary protection can reduce the overall infection-associated population even when the equilibrium distribution shifts from the hyperparasite-associated class toward the pathogen-only class.

The numerical simulations support the analytical results and illustrate how δ and ρ alter equilibrium populations, stability regions, and bifurcation thresholds. The results also reveal that the interaction between temporary protection and hyperparasite transmission is not necessarily monotone. Increasing the intensity or duration of temporary protection may directly reduce pathogen invasion but may also limit the establishment of the hyperparasite by reducing its available transmission pathway.

The temporary-protection mechanism considered in this model should be interpreted cautiously. Although some experimental studies have reported reductions in canker expansion, fungal growth, or disease incidence following chemical treatment under individual-tree, orchard, laboratory, or other managed conditions, these observations do not establish a broadly effective chemical treatment or chemically induced immunity for chestnut blight. Accordingly, the parameters δ and ρ represent abstract transition rates and have not been calibrated to a particular chemical compound, dosage, treatment schedule, or field application.

The present model also omits several mechanisms that may be important for chestnut blight transmission, including spatial heterogeneity, vegetative compatibility among fungal strains,

environmental variation, differences among chestnut genotypes, pathogen–hypovirus coevolution, and regional variation in hypovirus transmission. Therefore, the model provides a theoretical framework for studying the interaction between finite-duration protection and hyperparasite-mediated regulation rather than a directly applicable chestnut blight management plan.

Future studies should investigate whether experimental data can be used to estimate the transition rates δ and ρ and determine whether temporary protection can be distinguished from a direct reduction in pathogen transmission. Spatial, stochastic, and data-calibrated extensions should also incorporate vegetative compatibility structure, host resistance, hypovirus transmission, and environmental variability. Such developments may provide a more biologically detailed understanding of chestnut blight transmission and improve the connection between theoretical threshold results and observable disease dynamics.

Plant diseases pose a serious threat to global agricultural and forestry production, and the strengths and weaknesses of chemical and biological control are highly complementary. Chemical control acts quickly and efficiently and can suppress outbreaks rapidly, but long-term overuse aggravates pathogen resistance and pollutes the environment. Biological control, by contrast, offers long-term action, environmental friendliness, and a lower tendency to induce resistance, and can establish sustained ecological suppression. However, its effect is easily influenced by environmental conditions and often develops slowly, so its use alone is limited. Therefore, the synergistic use of both methods represents an optimal management path. Chemical treatment can be applied at the beginning of outbreaks to reduce the initial pathogen burden rapidly, after which biological control measures, such as the release of natural enemies or the application of microbial agents, can establish long-term ecological regulation [17], thereby sustaining suppression of disease occurrence and development and achieving complementary advantages and additive efficacy. In the future, the field of biological control will continue to explore novel control resources, such as bacteria, fungi, and viruses isolated from extreme environments with special antimicrobial activity, including high-temperature, high-salinity, and deep-sea habitats [29], as well as new biologically derived agents such as antimicrobial peptides [30]. Nevertheless, the strong influence of environmental conditions and the long time required for effective control remain important constraints, so integrated chemical–biological control is likely to remain the core direction of agricultural and forestry disease management.

Overall, the synergistic application of biological and chemical control overcomes the limitations of single-control approaches through complementary mechanisms, mutual risk suppression, and additive efficacy. While maintaining disease-control performance, it can reduce chemical use, lower environmental risk, delay resistance development, and unify green control with sustainable pest and disease management. It therefore provides new ideas and methods for agricultural disease management and has broad application prospects and considerable developmental potential.

Use of AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

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Conflict of interest

The authors declare there are no conflicts of interest.

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