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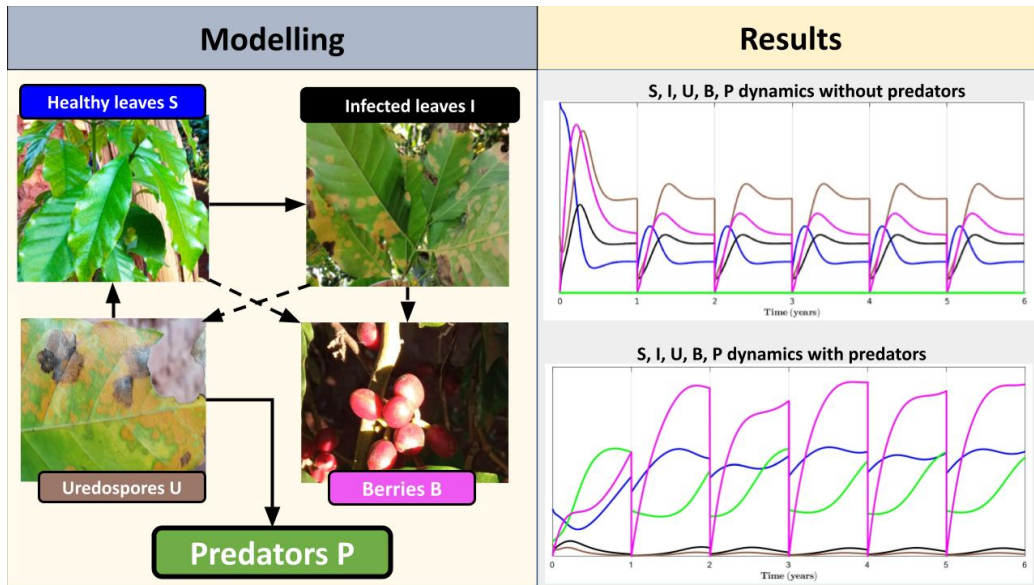


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Graphical Abstract

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Highlights

Impulsive modelling of rust dynamics and predator releases for biocontrol

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- Original impulsive model of multi-seasonal fungal disease dynamics
- Explicit dynamics of predators, acting as predators of spores
- predator-based biocontrol can drastically reduce disease impact on crop production
- predator release frequency modulates the control efficiency

Impulsive modelling of rust dynamics and predator releases for biocontrol

Clotilde Djuikem^{a,*}, Frédéric Grognard^a, Suzanne Touzeau^{a,b}

^a*Université Côte d'Azur, Inria, INRAE, CNRS, Sorbonne Université, BIOCORE, France*

^b*Université Côte d'Azur, INRAE, CNRS, ISA, France*

Abstract

Fungal diseases cause serious damages in crop worldwide. In particular, coffee leaf rust (CLR), caused by fungus *Hemileia vastatrix* attacks coffee leaves and reduces coffee yield. This paper presents a multi-seasonal model of the CLR development in the coffee plantation with continuous dynamics during the rainy season and a discrete event to represent the simpler dynamics during the dry season. Biological control using predators through one or more discrete introduction events over the year is then added. Analytical and semi-numerical studies are performed to identify how much and how frequently predators need to be introduced through the definition of a threshold value, as a function of various parameters. We show that the best strategy to efficiently control the disease depends on the predator mortality: low mortality parasites need be released only once a year, while high mortality parasites should be released more frequently to ensure their persistence in the plantation. This work hence provides qualitative and quantitative bases for the deployment of predator-based biocontrol, a promising alternative to fungicides for rust control.

Keywords: crop protection, coffee leaf rust, hybrid model, seasonality, Floquet theory, stability

*Corresponding author

Email address: `clotilde.djuikem@inria.fr` (Clotilde Djuikem)

1. Introduction

Many of the most serious crop diseases are caused by fungi such as rusts [1]. Mathematical models of fungal diseases have received a lot of attention from researchers. For instance, Pivonia et al. studied the seasonal appearance of rusts in the United States, in particular soybean rust, thanks to a general disease model [2]. Rimbaud et al. investigated how the spatial deployment of resistant cultivars affects the resistance efficiency and durability, using a demogenetic model, for a seasonal crop infected by a fungal-like pathogen [3]. Fleming used a mathematical model to prove that a complex of polyphagous non-synchronised predators and parasites is likely to control only low-density cereal rust populations, but the author also proved that control at low rust density can delay epidemic development and thus substantially reduce yield losses [4]. Mammeri et al. studied the impact of spatial heterogeneities on the spread and control of grapevine powdery mildew [5], but only during a cropping season. There are fewer models of fungal diseases that target perennial hosts. For instance, Ravigné et al. looked at the impact of sexual and asexual reproduction on the epidemiological dynamics and showed that they could induce cyclic persistence of the disease, as it can be observed for banana Sigatoka diseases [6]. Desprez-Loustau et al. developed a seasonal eco-evolutionary model of oak powdery mildew in Europe, based on a within-season and between-season transmission trade-off, which captures the main features of the disease, that is seasonality and pathogen species coexistence [7].

Plant growth and disease spread may be affected by seasonal patterns, which then need to be included in epidemiological models. It is the case of crops cultivated in temperate climates or tropical regions that alternate dry and rainy seasons. As the dynamics can substantially differ between the seasons with possibly rapid transitions, impulsive or semi-discrete models have been developed for several plant fungal diseases, as for oak powdery mildew in the study cited above. Among these models, one can also cite Tankam-Chedjou et al. who built a model to describe and control the dynamics of a banana soilborne pest in a multi-seasonal framework, optimising the fallow period durations between cropping seasons [8]. Periodic patterns are not necessarily linked to seasonality and can also be due to impulsive control strategies introduced in the epidemiological models. For instance, in the biological control framework, Nundloll et al. studied the periodic release of predators, natural enemies of the plant pest of interest, and determined

38 the minimal predator rate required to eradicate the pest [9]. Xinzhu et al.
39 formulated and analysed a model with continuous cultural control and with
40 impulsive cultural control strategies such as replanting and/or removing dis-
41 eased plants; they concluded that impulsive removing of diseased plants is
42 more efficient and more economical than continuous removing [10]. Nembot
43 et al. developed a model of cocoa black pod rot disease, caused by *Phy-*
44 *tophthora megakarya*, and showed the impact of periodic impulsive sanitary
45 harvests on the disease dynamics [11]. Semi-discrete models are hence par-
46 ticularly appropriate for seasonal plant and pathogen dynamics, as well as
47 impulsive control strategies.

48 Control methods used to fight fungal diseases include chemical fungicide
49 application, cultural practices, and the use of resistant cultivars [12]. These
50 methods induce significant labour and/or material costs. Moreover, chemical
51 fungicides are harmful to the environment [13], potentially also to farmers
52 and consumers [14]; in addition, they may affect non-target organisms and
53 induce pest resistance [15]. Therefore, researchers are currently investigating
54 alternative control methods such as biocontrol [16, 17]. Several biological
55 agents have been tested to control fungal plant diseases, among which vari-
56 ous bacteria. Antagonist bacteria such as *Bacillus* species make plants more
57 resistant to fungal infections [18, 19, 20, 21]. Other investigations on biocon-
58 trol focused on hyperparasites and predators. For example, several insects
59 such as *Mycodiplosis* (Diptera) [22] and fungi such as *Lecanicillium lecanii*
60 (previously called *Verticillium lecanii*) [23, 24] feed on rust spores.

61 As a foundation for our model, we considered coffee leaf rust (CLR),
62 which is caused by a fungus, *Hemileia vastatrix*. It attacks the lower leaves
63 of the coffee tree and causes premature defoliation, which reduces the photo-
64 synthetic capacity and weakens the tree. Leaf fall causes abortion of a large
65 part of the flowers and fruits, as well as desiccation of shoots. It has direct
66 and indirect economic impacts on coffee production. Direct impacts include
67 decreased quantity and quality of yield. In some cases, more than 70% of the
68 coffee production is lost [25, 26]. Indirect impacts include increased costs to
69 control the disease.

70 The fungus *Hemileia vastatrix* is a basidiomycete, which, like most fungi,
71 produces spores (used for reproduction). Its control is achieved similarly to
72 that of other fungal diseases. For instance, the antagonist bacteria *Bacil-*
73 *lus subtilis*, isolated from the rhizosphere of coffee crops, largely reduced the
74 spread of CLR (up to 68%) under in vitro conditions [21]. The fungus *Clad-*
75 *osporium hemileiae*, inhibits the evolution of *H. vastatrix* mycelium [27].

76 The fungus *Lecanicillium lecanii* [28] and the insect *Mycodiplosis* [29, 30]
77 can feed on spores. In all these biocontrol studies, the dose and time of
78 application of the bacteria, hyperparasites or predators affected their effec-
79 tiveness in controlling coffee leaf rust development. In this work, we chose
80 to focus on a *Mycodiplosis*-like predator, which was reported as a promising
81 biocontrol agent and paid special attention to the quantity and frequency of
82 the releases. To this aim, mathematical modelling can help identifying when
83 and how to release a biocontrol agent.

84 Our goal in this paper is to control a fungal disease in the field, by means
85 of predator releases, using a mathematical modelling approach. To achieve
86 this goal, we extend an original impulsive CLR model that we previously
87 developed [31]. In particular, we explicitly represent the predator dynamics,
88 the latter acting as predators of spores. Moreover, we consider in this work
89 discrete releases of the predator instead of a continuous control.

90 This paper is organised as follows. Section 2 is devoted to the formulation
91 of the impulsive model of CLR, with continuous dynamics during the rainy
92 seasons and discrete events for the dry seasons. It also presents the math-
93 ematical analysis of the model using Floquet theory [32] and simulations of
94 this impulsive model to illustrate the theoretical results. In Section 3, we
95 introduce the biocontrol in the model, based on predator yearly releases. We
96 semi-analytically study the stability of the controlled model to obtain the sta-
97 bility regions as functions of parameter values. Section 4 presents the impact
98 of multiple yearly releases of predators, with contrasting results according
99 to the value of the predator mortality rate. The last Section 5 concludes
100 the paper with a discussion of the main results and possible perspectives for
101 future work.

102 **2. Coffee leaf rust dynamics**

103 *2.1. Modeling of CLR*

104 In this section, we formulate a mathematical model for the CLR in the
105 coffee plantation. To do so, we consider the dynamics of fungus during the
106 production season, which corresponds to the rainy season for some countries,
107 through ordinary differential equations; while the non-production period,
108 which is the dry season, is represented by impulses since the dynamics are
109 simpler during that period. More precisely, we consider that in a coffee
110 plantation we can find: susceptible leaves S , which are healthy leaves that
111 have not (yet) been attacked, infected leaves I , uredospores U , which the

112 fungus *H. vastatrix* uses for its asexual reproduction, flowers F and berries
113 B .

114 During the production period with length $T > 0$ which represents the
115 rainy season, the recruitment of healthy leaves occurs at rate Λ . Uredospores
116 are deposited on all leaves at rate ν . They germinate with efficiency ω
117 ([33, 34]) and the susceptible leaves become infected leaves. All leaves
118 undergo natural mortality with baseline rate μ and the infected leaves have
119 an additional mortality rate d due to the disease. Uredospores are produced
120 on infected leaves by the fungus at rate γ and lose their ability to infect
121 coffee leaves at constant rate μ_U . The production of flowers and berries
122 depends on the photosynthesis, which in turn depends on the leaves, infected
123 leaves synthesising less carbon due to rust lesions. Moreover, coffee flowers
124 are carried at the leaf nodes on the branches and are hence also related to
125 leaves. We translate this in the model by flower production at constant rates
126 δ_S and δ_I “by” susceptible and infected leaves respectively, with $\delta_S > \delta_I$.
127 The flowers become berries at rate θ . Flowers and berries have a natural
128 mortality rate μ_F and μ_B , respectively. At the end of the production period,
129 harvest occurs instantaneously and we consider that the dry season or non-
130 production period can be summarised in a discrete time event due to the
131 slow growth of leaves during the dry season. The impact of harvest and
132 dry season reduces the number of susceptible leaves, infected leaves and
133 uredospores with proportions φ_S , φ_I and φ_U respectively. Using the fact
134 that uredospores lose their ability to infect quickly, we assume that φ_U is
135 very close to 0. The representation of this evolution in the coffee plantation
136 over a multi-year period, gives the multi-seasonal model, given in Figure 1,
137 with the continuous dynamics during the rainy season and switching event
138 for the dry season.

139 Using flowchart in Figure 1, we can write the following impulsive differ-
140 ential system:

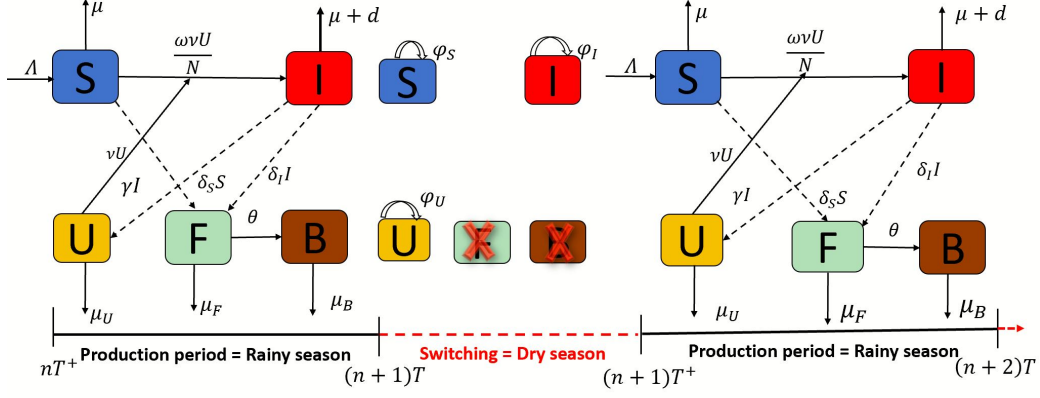


Figure 1: Diagram of the CLR multi-seasonal model. Model equations are given in system (1). State variables are: healthy leaves (S), infected leaves (I), uredospores (U), flowers (F) and berries (B). T corresponds to the length of rainy season and n the year number.

Rainy season for $t \neq nT$:

$$\begin{cases} \dot{S} = \Lambda - \omega\nu U \frac{S}{N} - \mu S, \\ \dot{I} = \omega\nu U \frac{S}{N} - (\mu + d)I, \\ \dot{U} = \gamma I - (\nu + \mu_U)U, \\ \dot{F} = \delta_S S + \delta_I I - (\theta + \mu_F)F, \\ \dot{B} = \theta F - \mu_B B; \end{cases} \quad (1)$$

Dry season:

$$\begin{cases} S(nT^+) = \varphi_S S(nT), \\ I(nT^+) = \varphi_I I(nT), \\ U(nT^+) = \varphi_U U(nT), \\ F(nT^+) = 0; \\ B(nT^+) = 0. \end{cases}$$

141 where $S(t)$, $I(t)$, $U(t)$, $F(t)$, $B(t)$ and $N(t) = S(t) + I(t)$ represent the
 142 number of healthy leaves, infected leaves, uredospores, flowers, berries and

143 total number of leaves, respectively, at time t ; with $0 < \varphi_S, \varphi_I, \varphi_U < 1$ and
 144 φ_U close to 0.

145 2.2. Mathematical analysis

146 Herein, we first presented basic properties of the model (1). Secondly,
 147 the periodic disease-free solution is computed and its stability is proven.
 148 Finally, numerical simulations are performed to illustrate our mathematical
 149 results.

150 2.2.1. Basic properties of the model

151 Let $\mathbb{R}_+^5 = \{X \in \mathbb{R}^5, X \geq 0\}$. Denote by $f = (f_1, f_2, f_3, f_4, f_5)$ the map
 152 given by the right-hand side of system (1) when $t \neq nT$, and consider the
 153 initial conditions $(S(0), I(0), U(0), F(0), B(0)) \in \mathbb{R}_+^* \times \mathbb{R}_+^4$.

154 The solutions of system (1) are non-negative. Indeed, suppose that one
 155 of the variables, that we will denote x , is equal to 0 at some instant, with all
 156 the others being non-negative. A quick analysis shows that $\dot{x} \geq 0$, so that x
 157 cannot become negative. This implies that the solutions are non-negative.

158 By adding the first and second equations of system (1), we obtain the
 159 dynamics of total leaves which satisfies:

$$\begin{cases} \dot{N} = \Lambda - \mu N - dI, & t \neq nT; \\ N(nT^+) = \varphi_S S(nT) + \varphi_I I(nT). \end{cases} \quad (2)$$

160 From equation (2), and using the non-negativity of variable I , we obtain

$$\begin{cases} \Lambda - (\mu + d)N \leq \dot{N} \leq \Lambda - \mu N, & t \neq nT; \\ 0 \leq N(nT^+) \leq N(nT). \end{cases}$$

Solving the above equation for $t \in (nT, (n+1)T]$, yields

$$\frac{\Lambda}{\mu + d} + \left(N(nT^+) - \frac{\Lambda}{\mu + d} \right) e^{-(\mu+d)(t-nT)} \leq N(t) \leq \frac{\Lambda}{\mu} + \left(N(nT^+) - \frac{\Lambda}{\mu} \right) e^{-\mu(t-nT)}$$

It follows that $\min(\Lambda/(\mu + d), N(nT^+)) \leq N(t) \leq \max(\Lambda/\mu, N(nT^+))$ for
 $t \in (nT, (n+1)T]$. The impulse with $0 < \varphi_S, \varphi_I < 1$ then imposes:

$$\min(\varphi_S, \varphi_I)N((n+1)T) \leq N((n+1)T^+) \leq N((n+1)T).$$

161 and hence

$$\min(\varphi_S, \varphi_I) \min(\Lambda/(\mu + d), N(nT^+)) \leq N(t) \leq \max(\Lambda/\mu, N(nT^+)) \quad (3)$$

for $t \in (nT, (n+1)T^+]$. Applying the R.H.S. recursively, we get

$$N(t) \leq \max(\Lambda/\mu, N(0)) = \Gamma_N$$

162 The recursive application of the L.H.S. of (3) does not yield a single lower
 163 bound, but ensures that there is a positive lower bound to N over each
 164 T -interval.

165 For $\Lambda/(\mu+d) < N(0) < \Lambda/\mu$, replacing S and I by this upper bound value
 166 in the uredospore and flower dynamics, we obtain the following systems:

$$\begin{cases} \dot{U} \leq \gamma \frac{\Lambda}{\mu} - (\nu + \mu_U)U, & t \neq nT; \\ U(nT^+) = \varphi_U U(nT); \end{cases} \quad (4)$$

$$\begin{cases} \dot{F} \leq \delta_S \frac{\Lambda}{\mu} + \delta_I \frac{\Lambda}{\mu} - (\theta + \mu_F)F, & t \neq nT; \\ F(nT^+) = 0. \end{cases} \quad (5)$$

167 Identifying the values where the upper bounds of (4) and (5) are equal
 168 to 0, one has $U(t) \leq \max(\frac{\gamma\Lambda}{\mu(\nu+\mu_U)}, U(0))$ and $F(t) \leq \max(\frac{\Lambda(\delta_S+\delta_I)}{\mu(\theta+\mu_F)}, F(0))$.
 169 Replace the upper bound of F in the berry dynamics, we obtain

$$\begin{cases} \dot{B} \leq \theta \frac{\Lambda(\delta_S + \delta_I)}{\mu(\theta + \mu_F)} - \mu_B B, & t \neq nT; \\ B(nT^+) = 0. \end{cases} \quad (6)$$

170 Identifying the values where the upper bounds of (6) is equal to 0, one has
 171 $B(t) \leq \max(\frac{\Lambda\theta(\delta_S+\delta_I)}{\mu(\theta+\mu_F)\mu_B}, B(0))$. Hence, we have shown the boundedness of S ,
 172 I , U , F and B . Also the region given by

$$G = \left\{ (S, I, U, F, B) \in \mathbb{R}_+^5 \mid \begin{aligned} S(t) + I(t) &\leq \frac{\Lambda}{\mu}, & U(t) &\leq \frac{\gamma\Lambda}{\mu(\nu + \mu_U)}, \\ F(t) &\leq \frac{\Lambda(\delta_S + \delta_I)}{\mu(\theta + \mu_F)}, & B(t) &\leq \frac{\Lambda\theta(\delta_S + \delta_I)}{\mu(\theta + \mu_F)\mu_B} \end{aligned} \right\}$$

173 is positively invariant for impulsive system (1).

174 We have hence shown the non-negativity and boundedness of the so-
 175 lutions. Moreover, the smooth properties of the right side of system (1)
 176 guarantee the existence and uniqueness of the solutions of this system.

177 Since the flower and berry state variables F and B are not present in the
 178 other equations of system (1), we do not need to consider F and B in the rest
 179 of the mathematical analysis. We then analyse this new system:

$$\begin{cases} \dot{S} = \Lambda - \frac{\omega\nu U}{N}S - \mu S, & t \neq nT; \\ \dot{I} = \frac{\omega\nu U}{N}S - (\mu + d)I, & t \neq nT; \\ \dot{U} = \gamma I - (\nu + \mu_U)U, & t \neq nT; \\ S(nT^+) = \varphi_S S(nT); \\ I(nT^+) = \varphi_I I(nT); \\ U(nT^+) = \varphi_U U(nT). \end{cases} \quad (7)$$

180 *2.2.2. Periodic disease-free solution and its stability*

181 The periodic disease-free solution (PDFS) occurs when $I = 0$ and $U = 0$.
 182 Replacing these values in system (7), we obtain

$$\begin{cases} \dot{S} = \Lambda - \mu S, & t \neq nT; \\ S(nT^+) = \varphi_S S(nT). \end{cases}$$

183 Solving the above equation for $t \in (nT, (n+1)T]$, yields

$$S(t) = \frac{\Lambda}{\mu} + \left(S(nT^+) - \frac{\Lambda}{\mu} \right) e^{-\mu(t-nT)}. \quad (8)$$

184 This implies that for $t = (n+1)T$, one has

$$S((n+1)T) = \frac{\Lambda}{\mu} + \left(S(nT^+) - \frac{\Lambda}{\mu} \right) e^{-\mu T}.$$

185 Using the impulsive condition $S((n+1)T^+) = \varphi_S S((n+1)T)$ yields

$$S((n+1)T^+) = \varphi_S \left(\frac{\Lambda}{\mu} + \left(S(nT^+) - \frac{\Lambda}{\mu} \right) e^{-\mu T} \right). \quad (9)$$

186 The fixed point of equation (9) is given by

$$S(nT^+) = \frac{\Lambda \varphi_S (1 - e^{-\mu T})}{\mu (1 - \varphi_S e^{-\mu T})} > 0. \quad (10)$$

187 Substituting the value of $S(nT^+)$ into equation (8), for $t \in (nT, (n+1)T]$,
 188 the solution is

$$S^T(t) = \frac{\Lambda}{\mu} \left[1 - \frac{(1 - \varphi_S)e^{\mu T}}{e^{\mu T} - \varphi_S} e^{-\mu(t-nT)} \right]. \quad (11)$$

189 Finally, the PDFS is $X^T(t) = (S^T(t), 0, 0)$, where $S^T(t)$ is defined above.

190 For stability analysis, we use Floquet theory and its implementation on
 191 impulsive systems as developed in [32]; it transforms the analysis of the im-
 192 pulsive system into that of a discrete mapping over the period. We first
 193 study the local stability of the PDFS $X^T(t)$ using small amplitude per-
 194 turbation methods. For that, let us denote $\tilde{X}(t) = X(t) - X^T(t)$, where
 195 $X(t) = (S(t), I(t), U(t))^T$ and $\tilde{X}(t)$ is understood to be small amplitude
 196 perturbations. Substituting the expression of $\tilde{X}(t)$ in system (7) gives

$$\begin{cases} \dot{\tilde{S}} = -\frac{\omega\nu\tilde{U}}{\tilde{N} + S^T(t)}(\tilde{S} + S^T(t)) - \mu\tilde{S}, & t \neq nT; \\ \dot{\tilde{I}} = \frac{\omega\nu\tilde{U}}{\tilde{N} + S^T(t)}(\tilde{S} + S^T(t)) - (\mu + d)\tilde{I}, & t \neq nT, \\ \dot{\tilde{U}} = \gamma\tilde{I} - (\nu + \mu_U)\tilde{U}, & t \neq nT; \\ \tilde{S}(nT^+) = \varphi_S\tilde{S}(nT); \\ \tilde{I}(nT^+) = \varphi_I\tilde{I}(nT); \\ \tilde{U}(nT^+) = \varphi_U\tilde{U}(nT). \end{cases} \quad (12)$$

197 where $\tilde{N}(t) = \tilde{S}(t) + \tilde{I}(t)$ and $N^T(t) = S^T(t)$.

198 The linearization of system (12) in the neighbourhood of $0_{\mathbb{R}^3}$ is

$$\begin{cases} \dot{\tilde{X}}(t) = A\tilde{X}(t), & t \neq nT; \\ \tilde{X}(nT^+) = \text{diag}(\varphi_S, \varphi_I, \varphi_U)\tilde{X}(nT); \end{cases} \quad (13)$$

199 where

$$A = \begin{pmatrix} -\mu & 0 & -\omega\nu \\ 0 & -(\mu + d) & \omega\nu \\ 0 & \gamma & -(\nu + \mu_U) \end{pmatrix}.$$

200 Solving the first equation of system (13) for $t \in (0, T]$ yields

$$\tilde{X}(t) = \Phi_A(t)\tilde{X}(0^+), \quad (14)$$

201 where

$$\Phi_A(t) = e^{At} = \begin{pmatrix} e^{-\mu t} & * & * \\ 0 & \psi_{22}(t) & \psi_{23}(t) \\ 0 & \psi_{32}(t) & \psi_{33}(t) \end{pmatrix}$$

202 and

$$\begin{cases} \psi_{22}(t) = \frac{1}{2\beta} [(\beta + k_1 - k_2)e^{\lambda_1 t} + (\beta - k_1 + k_2)e^{\lambda_2 t}], \\ \psi_{23}(t) = \frac{\nu\omega}{\beta} [-e^{\lambda_1 t} + e^{\lambda_2 t}], \psi_{32}(t) = \frac{\gamma}{\beta} [-e^{\lambda_1 t} + e^{\lambda_2 t}], \\ \psi_{33}(t) = \frac{1}{2\beta} [(\beta - k_1 + k_2)e^{\lambda_1 t} + (\beta + k_1 - k_2)e^{\lambda_2 t}], \end{cases}$$

203 with

$$\begin{cases} k_1 = \mu + d, & k_2 = \nu + \mu_U, \\ \alpha = k_1 + k_2, & \beta = \sqrt{(k_1 - k_2)^2 + 4\gamma\omega\nu}, \\ \lambda_1 = -\frac{\alpha}{2} - \frac{\beta}{2} \text{ and } \lambda_2 = -\frac{\alpha}{2} + \frac{\beta}{2}. \end{cases}$$

204 Observe that $\psi_{22}(t) > 0$, indeed $\beta^2 > (k_1 - k_2)^2$, which implies $\beta - k_1 + k_2 >$
 205 0 and $\beta + k_1 - k_2 > 0$.

206 Using the impulsive conditions $\tilde{X}(nT^+) = \text{diag}(\varphi_S, \varphi_I, \varphi_U)\tilde{X}(nT)$, the
 207 solution given by equation (14) becomes

$$\tilde{X}(nT^+) = \text{diag}(\varphi_S, \varphi_I, \varphi_U)\Phi_A(T)\tilde{X}((n-1)T^+). \quad (15)$$

208 Using [32], from the above equation, the monodromy matrix $M = \text{diag}(\varphi_S, \varphi_I, \varphi_U)\Phi_A(T)$,
 209 *i.e.*

$$M = \begin{pmatrix} \varphi_S e^{-\mu T} & * & * \\ 0 & \varphi_I \psi_{22}(T) & \varphi_I \psi_{23}(T) \\ 0 & \varphi_U \psi_{32}(T) & \varphi_U \psi_{33}(T) \end{pmatrix}.$$

210 Due to the block-triangular form of monodromy matrix M , there is no need
 211 to calculate the exact form of (*) for the following analysis.

212 According to [32], the Floquet multipliers of the monodromy matrix M
 213 are given by $\chi_1 = \varphi_S e^{-\mu T} < 1$ and the eigenvalues of the sub-matrix

$$M_1 = \begin{pmatrix} \varphi_I \psi_{22}(T) & \varphi_I \psi_{23}(T) \\ \varphi_U \psi_{32}(T) & \varphi_U \psi_{33}(T) \end{pmatrix}. \quad (16)$$

214 The PDFS $X^T(t)$ is locally asymptotically stable if the Floquet multi-
 215 pliers of the monodromy matrix M stay inside the unit circle [32], which

216 is equivalent to the Floquet multipliers of sub-matrix M_1 staying inside the
 217 unit circle. To obtain this result, the submonodromy matrix M_1 needs to
 218 satisfy the following Jury conditions [35]:

$$\begin{cases} -tr(M_1) - \det(M_1) < 1, \\ \det(M_1) < 1, \\ tr(M_1) - \det(M_1) < 1, \end{cases} \quad (17)$$

219 where

$$\begin{cases} tr(M_1) = \varphi_I \psi_{22}(T) + \varphi_U \psi_{33}(T), \\ \det(M_1) = \varphi_I \varphi_U (\psi_{22}(T) \psi_{33}(T) - \psi_{23}(T) \psi_{32}(T)). \end{cases}$$

220 Using the hypothesis $\varphi_U \rightarrow 0$, since $\psi_{22}(T) > 0$, conditions (17) hold if and
 221 only if $\mathcal{R} = \varphi_I \psi_{22}(T) < 1$. Finally, we obtain the following Lemma for local
 222 stability.

223 **Lemma 1.** *The PDFS $X^T(t)$ is locally asymptotically stable provided that*
 224 *$\mathcal{R} < 1$ and unstable otherwise, where*

$$\mathcal{R} = \frac{\varphi_I}{2\beta} [(\beta + k_1 - k_2)e^{\lambda_1 T} + (\beta - k_1 + k_2)e^{\lambda_2 T}]. \quad (18)$$

225 Moreover, the following result about the global asymptotic stability of
 226 the PDFS $X^T(t)$ of system (7) holds.

227 **Theorem 2.** *The PDFS $X^T(t)$ of system (7) is globally asymptotically stable*
 228 *if $\mathcal{R} < 1$ and unstable otherwise.*

229 *Proof.* Let us prove the global attractivity of the PDFS $X^T(t)$. Consider the
 230 following subsystem:

$$\begin{cases} \dot{I} = \frac{\omega \nu U}{N} S - (\mu + d)I, & t \neq nT; \\ \dot{U} = \gamma I - (\nu + \mu_U)U, & t \neq nT; \\ I(nT^+) = \varphi_I I(nT); \\ U(nT^+) = \varphi_U U(nT). \end{cases} \quad (19)$$

231 The trivial solution of system (19) is $(0, 0)$ and the system is cooperative
 232 because $\partial \dot{I} / \partial U = \frac{\omega \nu S}{N} > 0$, $\partial \dot{U} / \partial I = \gamma > 0$. As $S(t)/N(t) < 1$, one has that

233 system (19) is upper bounded by the following cooperative system:

$$\begin{cases} \dot{I}_1 = \omega\nu U_1 - (\mu + d)I_1, & t \neq nT; \\ \dot{U}_1 = \gamma I_1 - (\nu + \mu_U)U_1, & t \neq nT; \\ I_1(nT^+) = \varphi_I I_1(nT); \\ U_1(nT^+) = \varphi_U U_1(nT). \end{cases} \quad (20)$$

234 Applying Kamke's theorem [36], one has that $I(t) \leq I_1(t), U(t) \leq U_1(t)$
 235 when $I_1(0) = I(0)$ and $U_1(0) = U(0)$. When $\mathcal{R} < 1$, using the previous result
 236 of local stability, $(I_1(t), U_1(t)) \rightarrow (0^+, 0^+)$. Then, one has

$$(I_1(t), U_1(t)) \rightarrow (0^+, 0^+) \Rightarrow (I(t), U(t)) \rightarrow (0^+, 0^+).$$

237 Since $U \rightarrow 0^+$, for any arbitrary positive ε_U there exists $t_0 > 0$ such that
 238 for $t \geq t_0$, $U(t) \leq \varepsilon_U$. Using non-negativity of the solutions and the fact that
 239 $S(t)/N(t) \leq 1$ into the first equation of system (7), one has :

$$\Lambda - \omega\nu\varepsilon_U - \mu S(t) \leq \dot{S}(t) \leq \Lambda - \mu S(t)$$

240 Applying the comparison principle on the above differential inequalities and
 241 using the impulsive condition, one has $S_1(t) \leq S(t) \leq S_2(t)$, with $S_1(t)$ and
 242 $S_2(t)$ the solutions of the following impulsive differential equations respec-
 243 tively:

$$\begin{cases} \dot{S}_1 = \Lambda - \omega\nu\varepsilon_U - \mu S_1, & t \neq nT; \\ S_1(nT^+) = \varphi_S S_1(nT), \end{cases}$$

244 and

$$\begin{cases} \dot{S}_2 = \Lambda - \mu S_2, & t \neq nT; \\ S_2(nT^+) = \varphi_S S_2(nT). \end{cases}$$

245 One can observe that $S_1(t) \rightarrow S_{\varepsilon_U}^T(t) = \frac{\Lambda - \omega\nu\varepsilon_U}{\mu} \left[1 - \frac{(1 - \varphi_S)e^{\mu T}}{e^{\mu T} - \varphi_S} e^{-\mu(t - nT)} \right]$ and
 246 $S_2(t) \rightarrow S^T(t)$ asymptotically, where $S^T(t)$ is given in equation (11). This
 247 implies that

$$S_{\varepsilon_U}^T(t) \leq S(t) \leq S^T(t). \quad (21)$$

248 Since we can take ε_U as small as we want since $U(t) \rightarrow 0$, we obtain
 249 $S_{\varepsilon_U}^T(t) \rightarrow S^T(t)$ when $\varepsilon_U \rightarrow 0$. Finally, using (21), one has that $S(t) \rightarrow$

250 $S^T(t)$. This implies that the PDFS is globally attractive. In other words,
 251 independently from the initial conditions (S_0, I_0, U_0) , one has that

$$(S(t), I(t), U(t)) \rightarrow (S^T(t), 0, 0)$$

252 This concludes the proof of global asymptotic stability. □

253 *2.3. Numerical simulations of CLR dynamics*

254 Herein, we present the results of numerical simulations of system (1)
 255 using ode45 in Matlab to integrate the differential equations during the rainy
 256 seasons, with the impulses giving each year's initial conditions. We take
 257 $T = 250$ days, which is the length of rainy season in Cameroon [37] and also
 258 corresponds to the length of coffee production period. For the simulations, we
 259 suppose that all leaves are initially healthy, which means that $I(0) = 0$ leaf,
 260 with $S(0) = 500$ leaves. Infection is initiated by uredospores, with $U(0) =$
 261 3000 spores. There are no flowers and berries initially, $F(0) = 0$ flower and
 262 $B(0) = 0$ berry, because simulations start at the beginning of the production
 263 period. The initial conditions are then

$$(S(0), I(0), U(0), F(0), B(0)) = (500, 0, 3000, 0, 0). \quad (22)$$

264 Table 1 summarises the parameter definitions and values used in this
 265 paper.

266 In Theorem 2, we show that when the spectral radius \mathcal{R} is greater than
 267 one, the periodic disease-free solution (PDFS) is unstable. In this case, CLR
 268 can establish itself, which is confirmed by the solid curve of Figure 2, drawn
 269 for $\mathcal{R} = 2.53 > 1$.

270 The dashed curve of Figure 2, drawn for $\mathcal{R} = 0.27 < 1$, shows that, for the
 271 initial conditions considered, the number of infected leaves and uredospores
 272 converges towards zero. The system then converges to the PDFS. We can
 273 observe in subplot (a) that, once the stationary regime is established, there
 274 are considerably less healthy leaves with endemic CLR (solid curve) than
 275 when CLR disappears (dashed curve). The same phenomenon can be ob-
 276 served in subplot (d), which represents the berry trajectories. At the end of
 277 the 6th year, the number of berries is 9844 without disease (dashed curve)
 278 and 4290 with disease (solid curve), indicating a yield loss larger than 56%
 279 due to CLR.

Table 1: Description and values of parameters for system (23).

Symbol	Biological meaning	Value	Source
T	Rainy season duration	250 days	[37]
Λ	Recruitment rate of S	8 leaves/day	Assumed
ω	Germination efficiency	0.065 leaf/spore	Assumed
μ	Mortality rate of leaves	0.0034/day	Assumed
γ	Sporulation rate by I	2 spores/leaf.day	[38]
d	Mortality rate due to CLR	0.056/day	Assumed
ν	Deposition rate	0.09/day	[38]
μ_U	Mortality rate of U	0.015/day	Assumed
φ_S	Survival proportion of S	0.7	Assumed
φ_I	Survival proportion of I	0.4	Assumed
φ_U	Survival proportion of U	0.1	Assumed
δ_S	Flower production rate by S	0.08 flowers/leaf.day	[39]
δ_I	Flower production rate by I	0.04 flowers/leaf.day	[39]
μ_F	Mortality rate of F	$2,4 \times 10^{-3}$ /day	[40]
θ	Maturation rate of F	0.0055/day	[41]
μ_B	Mortality rate of B	$2,4 \times 10^{-3}$ /day	[40]
Λ_P	Yearly released quantity of P	<i>variable</i> predators	Assumed
a	Consumption rate	<i>variable</i> spores/predator.day	Assumed
e	Biomass transformation rate	0.7 predators/spore	Assumed
K	Saturation constant of P	100000 spores	Assumed
μ_P	Mortality rate of P	0.003 & 0.1/day	Assumed
φ_P	Survival proportion of P	0.3	Assumed

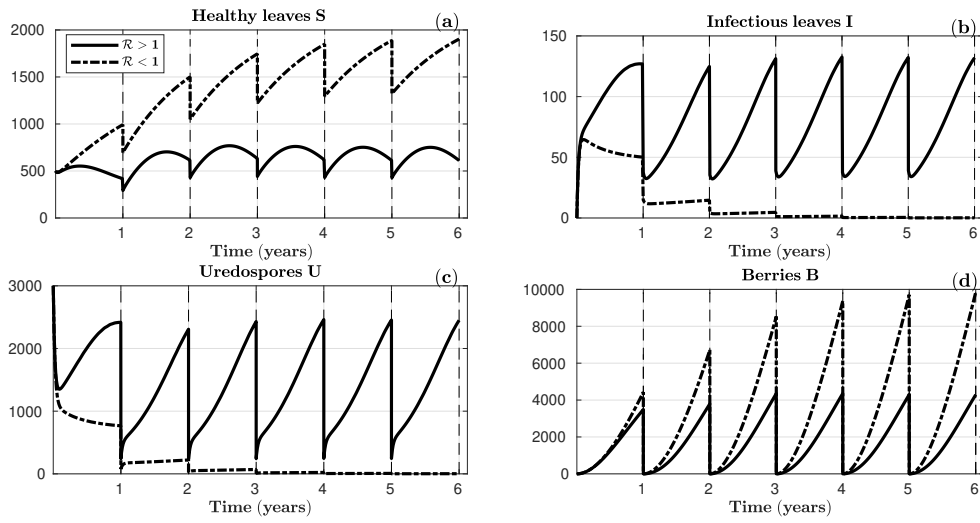


Figure 2: Impact of CLR on the production of coffee berries. The plots present the trajectories of impulsive differential system (7) when the PDFS is stable (dashed curve, $\gamma = 1.6$ spores/leaf.day $\Rightarrow \mathcal{R} = 0.27 < 1$) and when the PDFS is unstable (solid curve, default value $\gamma = 2$ spores/leaf.day $\Rightarrow \mathcal{R} = 2.53 > 1$). Subplots represent: (a) healthy leaves S , (b) infected leaves I , (c) uredospores U and (d) berries B . Remaining parameter values are given in Table 1 and initial conditions by (22).

280 **3. Biocontrol of CLR using predators**

281 To limit the impact of CLR on coffee berry production, we now intro-
 282 duce a biocontrol agent in the model, more specifically a predator such as
 283 *Mycodiplosis*, which consumes uredospores of *H. vastatrix*. The new model is
 284 then an extension of system (7), to which we add an equation for the pred-
 285 ators (or predators) P , that prey on the uredospores with consumption rate
 286 a , biomass transformation rate e and mortality rate μ_P . A proportion φ_P
 287 survives the dry season. Our aim is to identify how many and when to release
 288 predators in order to control CLR. In this section, we assume that a quantity
 289 Λ_P of predators is released once a year, at the start of the production season.
 290 This translates into a Λ_P jump in the predator population at each switching
 291 moment. The model with biocontrol is then given by

$$\begin{aligned}
 & \text{Rainy season for } t \neq nT : \\
 & \left\{ \begin{aligned}
 \dot{S} &= \Lambda - \omega\nu U \frac{S}{N} - \mu S; \\
 \dot{I} &= \omega\nu U \frac{S}{N} - (\mu + d)I; \\
 \dot{U} &= \gamma I - (\nu + \mu_U)U - aP \frac{U}{K + U}; \\
 \dot{P} &= eaP \frac{U}{K + U} - \mu_P P; \\
 \dot{F} &= \delta_S S + \delta_I I - (\theta + \mu_F)F, \\
 \dot{B} &= \theta F - \mu_B B.
 \end{aligned} \right. \tag{23}
 \end{aligned}$$

Dry season:

$$\left\{ \begin{aligned}
 S(nT^+) &= \varphi_s S(nT); \\
 I(nT^+) &= \varphi_I I(nT); \\
 U(nT^+) &= \varphi_U U(nT); \\
 P(nT^+) &= \varphi_P P(nT) + \Lambda_P; \\
 F(nT^+) &= 0. \\
 B(nT^+) &= 0.
 \end{aligned} \right.$$

292 System (23) has the same properties as system (7) for the existence,
 293 uniqueness, non-negativity and boundness of solutions. We pursue the math-
 294 ematical analysis and simulations of system (23) without the \dot{F} and \dot{B} equa-
 295 tions, since berry variable B is not present in the other equations.

296 *3.1. Controlled periodic disease-free solution and its stability*

297 The controlled periodic disease-free solution (cPDFS) occurs when $I = 0$
 298 and $U = 0$. The healthy leaves dynamics are unchanged compared to the
 299 PDFS and still satisfy (11) while the predator dynamics are

$$\begin{cases} \dot{P} = -\mu_P P, & t \neq nT; \\ P(nT^+) = \varphi_P P(nT) + \Lambda_P, & t = nT. \end{cases} \quad (24)$$

300 Solving equation (24) yields

$$P((n+1)T) = P(nT^+)e^{-\mu_P T}.$$

301 Combined, the above equation and the impulsive condition give

$$P((n+1)T^+) = \varphi_P P(nT^+)e^{-\mu_P T} + \Lambda_P.$$

302 Then, solving for the fixed point of the above discrete equation and using
 303 the dynamics of predator at the cPDFS yields

$$P^T(t) = \frac{\Lambda_P}{1 - \varphi_P e^{-\mu_P T}} e^{-\mu_P (t-nT)}. \quad (25)$$

304 Thus, the cPDFS for system (23) is $Y^T(t) = (S^T(t), 0, 0, P^T(t))$, where
 305 $S^T(t)$ and $P^T(t)$ are given in equation (11) and (25), respectively. We have
 306 the following result for the global stability of cPDFS.

307 **Lemma 3.** *The cPDFS $Y^T(t) = (S^T(t), 0, 0, P^T(t))$ of system (23) is glob-*
 308 *ally asymptotically stable when $\mathcal{R} < 1$.*

309 *Proof.* Let $\tilde{Y}(t) = Y(t) - Y^T(t)$ where $Y(t) = (S(t), I(t), U(t), P(t))$ and
 310 $\tilde{Y}(t)$ is understood as to be small amplitude perturbations. Substituting the

311 expression of $\tilde{Y}(t)$ in the system (7) gives

$$\begin{cases}
\dot{\tilde{S}} = -\frac{\omega\nu\tilde{U}}{\tilde{N} + S^T(t)}(\tilde{S} + S^T(t)) - \mu\tilde{S}, & t \neq nT; \\
\dot{\tilde{I}} = \frac{\omega\nu\tilde{U}}{\tilde{N} + S^T(t)}(\tilde{S} + S^T(t)) - (\mu + d)\tilde{I}, & t \neq nT; \\
\dot{\tilde{U}} = \gamma\tilde{I} - (\nu + \mu_U)\tilde{U} - a(\tilde{P} + P^T(t))\frac{\tilde{U}}{K + \tilde{U}}, & t \neq nT; \\
\dot{\tilde{P}} = ea(\tilde{P} + P^T(t))\frac{\tilde{U}}{K + \tilde{U}} - \mu_P\tilde{P}, & t \neq nT; \\
\tilde{S}(nT^+) = \varphi_S\tilde{S}(nT); \\
\tilde{I}(nT^+) = \varphi_I\tilde{I}(nT); \\
\tilde{U}(nT^+) = \varphi_U\tilde{U}(nT); \\
\tilde{P}(nT^+) = \varphi_P\tilde{P}(nT);
\end{cases} \quad (26)$$

312 where $\tilde{N}(t) = \tilde{S}(t) + \tilde{I}(t)$ and $N^T(t) = S^T(t)$.

313 The linearisation of system (26) around $0_{\mathbb{R}^4}$ is

$$\begin{cases}
\dot{\tilde{Y}}(t) = G(t)\tilde{Y}(t), & t \neq nT; \\
\tilde{Y}(nT^+) = \text{diag}(\varphi_S, \varphi_I, \varphi_U, \varphi_P)\tilde{Y}(nT);
\end{cases} \quad (27)$$

314 where

$$G(t) = \begin{pmatrix}
-\mu & 0 & -\omega\nu & 0 \\
0 & -(\mu + d) & \omega\nu & 0 \\
0 & \gamma & -(\nu + \mu_U) - \frac{aP^T(t)}{K} & 0 \\
0 & 0 & \frac{eaP^T(t)}{K} & -\mu_P
\end{pmatrix}.$$

315 Solving system (27) for $t \in (0, T]$ yields $\tilde{Y}(t) = \Phi_G(t)\tilde{Y}(0^+)$, with $\Phi_G(t)$ the
316 fundamental matrix that satisfies

$$\frac{d\Phi_G(t)}{dt} = G(t)\Phi_G(t),$$

317 where $\Phi_G(0) = I$. Re-ordering the variables as follows, $(\tilde{S}, \tilde{P}, \tilde{I}, \tilde{U})$, we note
318 that the system is block-triangular. The stability is then determined by
319 considering separately (\tilde{S}, \tilde{P}) and the pair (\tilde{I}, \tilde{U}) . The Floquet multipliers
320 for the first two are $\varphi_S e^{-\mu T}$ and $\varphi_P e^{-\mu_P T}$, which are smaller than one. With

321 this in mind, the stability of system (27) reduces to the stability of the
 322 following sub-system

$$\begin{cases} \dot{\tilde{Y}}_1(t) = G_1(t)\tilde{Y}_1(t), & t \neq nT; \\ \tilde{Y}(nT^+) = \text{diag}(\varphi_I, \varphi_U)\tilde{Y}(nT); \end{cases} \quad (28)$$

323 where

$$Y_1(t) = (I(t), U(t)) \quad \text{and} \quad G_1(t) = \begin{pmatrix} -(\mu + d) & \omega\nu \\ \gamma & -(\nu + \mu_U) - \frac{aP^T(t)}{K} \end{pmatrix}.$$

324 One can observe that

$$G_1(t) < B = \begin{pmatrix} -(\mu + d) & \omega\nu \\ \gamma & -(\nu + \mu_U) \end{pmatrix}.$$

325 The monodromy matrix associated to matrix B is the matrix M_1 given in
 326 equation (16), which is stable if $\mathcal{R} < 1$, where \mathcal{R} is given in equation (18).
 327 Using the fact that system (28) is cooperative, we can conclude that, when
 328 $\mathcal{R} < 1$, the cPDFS $Y^T(t)$ is globally asymptotically stable, which implies
 329 that CLR dwindles until extinction. \square

330 3.2. Semi-numerical analysis of the controlled model

331 In order to analyse system (23) when $\mathcal{R} > 1$, we need to compute the
 332 monodromy matrix associated with matrix $G_1(t)$. This is done by numeri-
 333 cally solving the linear system (28) using the initial conditions $\tilde{Y}_1(0^+) = (1, 0)$
 334 and $\tilde{Y}_1(0^+) = (0, 1)$. The two solutions evaluated at time T are put together
 335 to obtain the fundamental matrix. Then we use the impulsive condition to
 336 obtain the monodromy matrix.

337 The monodromy matrix associated with \tilde{Y}_1 is

$$M_{G_1} = \begin{pmatrix} \varphi_I & 0 \\ 0 & \varphi_U \end{pmatrix} \Phi_{G_1}(T).$$

338 We analyse the spectral radius \mathcal{R}_c of this matrix and check that it is smaller
 339 than one for the local stability of the cPDFS $Y^T(t)$ of system (23). We term
 340 this method “semi-numerical” as it requires numerical computations, but not
 341 extensive simulations of the system [42].

342 In Figure 3, we plot the threshold level of the spectral radius $\mathcal{R}_c = 1$ of
 343 controlled system (23) for parameter pairs (Λ_P, a) (subplot (a)) and (ν, γ)

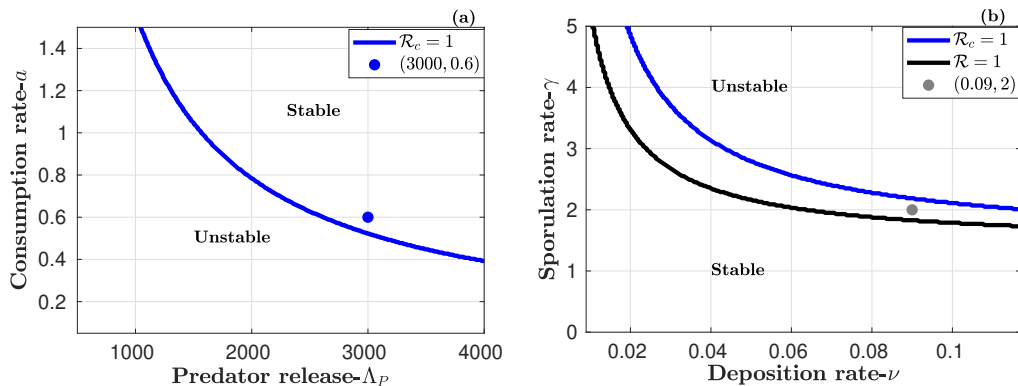


Figure 3: Stability regions of periodic disease-free solutions as functions of model parameters. Blue curves represent the threshold $\mathcal{R}_C = 1$ separating the stable and unstable regions of the model with predator (23), for different values of parameter pair (Λ_P, a) in subplot (a) and (ν, γ) in subplot (b); moreover, the black curve $\mathcal{R} = 1$ in subplot (b) separates the stable and unstable regions of the model without predator (1). The blue dot in subplot (a) corresponds to the (Λ_P, a) values used for the blue curve in subplot (b). Similarly, the grey dot in subplot (b) corresponds to the default (ν, γ) values used for the blue curve in subplot (a). Remaining parameter values are given in Table 1.

344 (subplot (b)), as well as the threshold stability level $\mathcal{R} = 1$ of uncontrolled
 345 system (1) for parameter pair (ν, γ) . In subplot (a), the regions below and
 346 above the blue curve are, respectively, the unstable and stable regions of the
 347 cPDFS of controlled system (23). As expected, stability of the cPDFS is
 348 guaranteed for large values of the yearly quantity of predators released Λ_P ,
 349 and of the predator consumption rate a . Also, when the predator capacity
 350 to consume uredospores is higher, less predators are needed for the extinc-
 351 tion of the disease, and vice versa. The blue dot corresponds to parameter
 352 values ensuring a stable cPDFS, values that are used for the blue curve in
 353 subplot (b).

354 In subplot (b), as in subplot (a), the blue curve separates the unstable
 355 and stable regions of the cPDFS of the controlled system; moreover, the black
 356 curve separates these two regions for the uncontrolled system. The parameter
 357 region between these two curves hence represents the gain obtained by adding
 358 the biocontrol: in this region, CLR goes extinct with biocontrol, but persists
 359 without. Subplot (b) shows that, in order to eliminate CLR through the
 360 proposed biocontrol effort, the sporulation and deposition rate should not be
 361 too large.

362 *3.3. Asymptotic behaviour for yearly releases*

363 Herein, we investigate the asymptotic behaviour of system (23) when
364 predators are released once a year at the beginning of each production period.
365 To do so, we consider the parameter values given by the two dots in the
366 stability regions of Figure 3. We point out that the gray dot in subplot (b)
367 of Figure 3 is in the instability region of system (23) without control which
368 means that the CLR would persist in the coffee plantation if unchecked. We
369 consider the initial condition given in equation (22), adding the initial release
370 of predators $P(0^+) = \Lambda_P = 3000$, which yields

$$(S(0^+), I(0^+), U(0^+), P(0^+), F(0^+), B(0^+)) = (500, 0, 3000, 3000, 0, 0). \quad (29)$$

371 Figure 4 compares the temporal dynamics of system (1) without control
372 (black curves) and model (23) with control (blue curves). From this figure,
373 one can observe that under the actions of biocontrol, the number of healthy
374 leaves increases and becomes stationary (see Figure 4(a)), while the number
375 of infected leaves and uredospores dwindles until extinction (see Figures 4(b)
376 and (c)). During the first year, the quantity of predators (predators) increases
377 through uredospore consumption. From the second year on, the number of
378 uredospores is very close to zero, so that the solution is close to the cPDFS.
379 Biocontrol using predators reduces drastically CLR in the coffee plantation.

380 **4. Multiple releases of predators per year**

381 In this section, we suppose that the yearly quantity Λ_P of predators is
382 released at different times over the year: it is uniformly divided into m
383 releases of size $\frac{\Lambda_P}{m}$, with release interval $\frac{T}{m}$. We thus obtain an impulsive
384 model with a new switching condition for the predator, given by

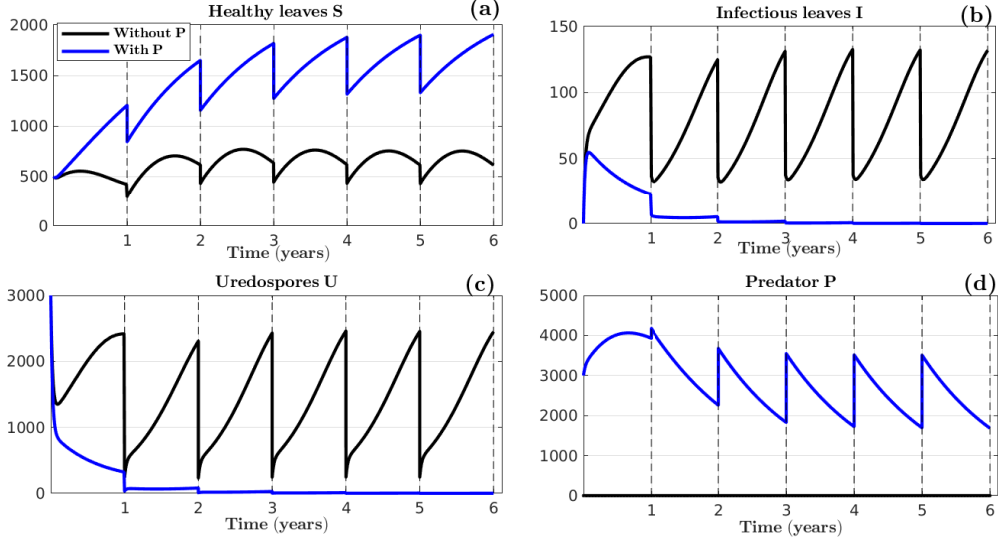


Figure 4: Impact of biocontrol on the system dynamics. 6-year simulations of model (1) without predator (black curve) and model (23) with predator (blue curve), using initial conditions (29): (a) healthy leaves S ; (b) infected leaves I ; (c) uredospores U and (d) predators P . All parameter values are given in Table 1 and correspond to $\mathcal{R} = 2.53 > 1$.

Rainy season for $t \neq nT$:

$$\begin{cases} \dot{S} = \Lambda - \omega\nu U \frac{S}{N} - \mu S; \\ \dot{I} = \omega\nu U \frac{S}{N} - (\mu + d)I; \\ \dot{U} = \gamma I - (\nu + \mu_U)U - aP \frac{U}{K + U}; \\ \dot{P} = eaP \frac{U}{K + U} - \mu_P P, \\ \dot{F} = \delta_S S + \delta_I I - (\theta + \mu_F)F \\ \dot{B} = \theta F - \mu_B B. \end{cases} \quad t \neq nT + \frac{j}{m}T;$$

Predator releases for $j \in \{1, \dots, m-1\}$:

$$P\left(nT + \frac{j}{m}T^+\right) = P\left(nT + \frac{j}{m}T\right) + \frac{\Lambda_P}{m}; \quad (30)$$

Dry season:

$$\begin{cases} S(nT^+) = \varphi_S S(nT); \\ I(nT^+) = \varphi_I I(nT); \\ U(nT^+) = \varphi_U U(nT); \\ P(nT^+) = \varphi_P P(nT) + \frac{\Lambda_P}{m}; \\ F(nT^+) = 0 \\ B(nT^+) = 0. \end{cases} \quad 23$$

385 System (30) has the same properties as system (7) for the existence,
 386 uniqueness, non-negativity, and boundedness of solution. We made the math-
 387 ematical analysis of system (30) without \dot{F} and \dot{B} equations, since B is not
 388 present in other equations.

389 *4.1. Multiple release controlled periodic disease-free solution and its stability*

390 To compute the multiple release controlled periodic disease-free solution
 391 (m-cPDFS), we only consider the first year for readability purposes, which
 392 means that $t \in (0, T]$. The m-cPDFS of biocontrol system (30) occurs when
 393 $I = 0$ and $U = 0$, so healthy leaf dynamics are unchanged compared to the
 394 PDFS and still satisfy (11); the predator dynamics are

$$\begin{cases} \dot{P} = -\mu_P P, & t \neq nT; \\ P\left(\frac{j}{m}T^+\right) = P\left(\frac{j}{m}T\right) + \frac{\Lambda_P}{m}, & \text{for } j = 1, \dots, m-1; \\ P(T^+) = \varphi_P P\left(\frac{(m-1)}{m}T\right) + \frac{\Lambda_P}{m}. \end{cases} \quad (31)$$

395 The expression of the m-cPDFS is established in Appendix B.1. We
 396 obtain $Z^T(t) = (S^T(t), 0, 0, P_{m,j}^T(t))$, for $t \in \left[\frac{jT}{m}, \frac{(j+1)T}{m}\right]$, where $S^T(t)$ is given
 397 by (11) and $P_{m,j}^T(t)$ by

$$P_{m,j}^T(t) = \left[P(0^+) e^{-\mu_P \frac{jT}{m}} + \frac{\Lambda_P}{m} \left(\frac{1 - e^{-\mu_P \frac{jT}{m}}}{1 - e^{-\mu_P \frac{T}{m}}} \right) \right] e^{-\mu_P (t - \frac{jT}{m})}, \quad (32)$$

398 with

$$P(0^+) = \frac{\Lambda_P}{m} \left[\frac{\varphi_P \left(e^{-\mu_P \frac{T}{m}} - e^{-\mu_P T} \right) + 1 - e^{-\mu_P \frac{T}{m}}}{(1 - \varphi_P e^{-\mu_P T}) \left(1 - e^{-\mu_P \frac{T}{m}} \right)} \right]. \quad (33)$$

399 **Lemma 4.** *The m-cPDFS $Z^T(t) = (S^T(t), 0, 0, P_{m,j}^T(t))$ of controlled model*
 400 *with multiple releases, is globally asymptotically stable when $\mathcal{R} < 1$*

401 *Proof.* The proof is similar to the proof of Lemma 3 which holds for one
 402 release per year. \square

403 To present the impact of the multiple release strategy on CLR control,
 404 we first compute a proxy of the control intensity over the season, namely

405 the yearly average number of predators present in the plantation for the m-
 406 cPDFS. This quantity depends on m , the number of predator releases per
 407 year. Its expression, established in Appendix B.2, is

$$g(m) = \int_0^T P^T(t)dt = \sum_{j=0}^{m-1} \int_{\frac{jT}{m}}^{\frac{(j+1)T}{m}} P_{m,j}^T(t)dt = \frac{\Gamma}{m(e^{\mu_P \frac{T}{m}} - 1)} + \frac{\Lambda_P}{\mu_P}, \quad (34)$$

408 with

$$\Gamma = \frac{-\Lambda_P(1 - \varphi_P)(1 - e^{-\mu_P T})}{\mu_P(1 - \varphi_P e^{-\mu_P T})}.$$

409 Differentiating $g(m)$ with respect to m , one has

$$g'(m) = \frac{\Gamma e^{\mu_P \frac{T}{m}} \left(e^{-\mu_P \frac{T}{m}} + \frac{\mu_P T}{m} - 1 \right)}{m^2 (e^{\mu_P \frac{T}{m}} - 1)^2}.$$

410 Since $e^{-\mu_P \frac{T}{m}}$ is always larger than its first order approximation $1 - \frac{\mu_P T}{m}$ for
 411 $\frac{T}{m} > 0$, all factors are positive except Γ , so that $g'(m) < 0$. This allows
 412 to conclude that the yearly average number of predators decreases with the
 413 number of releases per year, without CLR (quantity computed for the m-
 414 cPDFS). One might then think that spreading Λ_P over several releases is less
 415 efficient than releasing everything at the beginning of the season. This is
 416 investigated below through simulation.

417 We use two contrasted predator mortality rates to simulate the solution
 418 $P_{m,j}^T(t)$ in the m-cPDFS. Subplots (a) and (b) of Figure 5 present the simu-
 419 lation of $P_{m,j}^T(t)$ for various release frequencies and for both mortality rates
 420 over three years; they illustrate the important impact these two factors on
 421 the m-cPDFS.

422 4.2. Impact of release frequency on CLR control

423 Herein, we present the impact of the yearly predator release frequency on
 424 CLR control. This is first done using the semi-numerical analysis of the model
 425 with multiple releases, to show the (Λ_p, a) -stability region of the m-cPDFS
 426 (as in Figure 3), for different values of the release number m . Secondly, we
 427 present simulations of the dynamic behaviour of system (30).

428 In all simulations, the epidemiological parameters are set to their value in
 429 Table 1. The only parameters that vary are the ones related to the predators:
 430 Λ_p, a, μ_p and m . As in Figure 5, we use two contrasted values for the predator

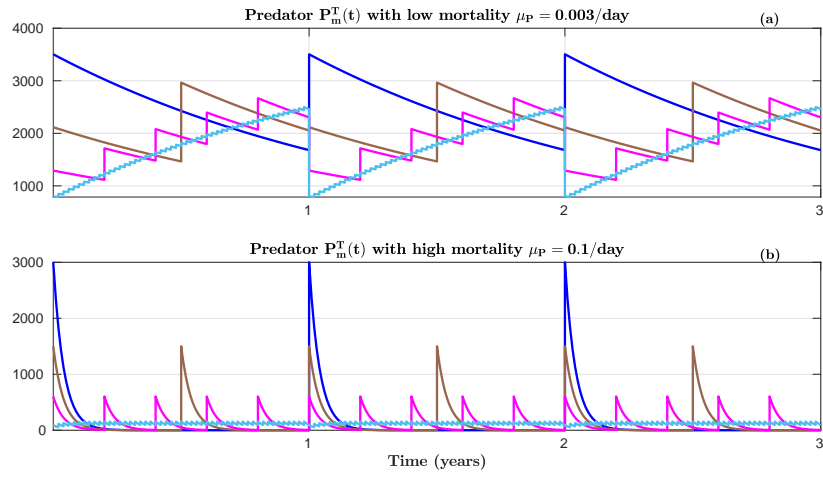


Figure 5: Impact of the predator mortality and its release frequency on its dynamics without CLR. The plots present the predator dynamics of the m-cPDFS (multiple release controlled periodic disease-free solution) of system (30) for: (a) the default low mortality rate $\mu_P = 0.003/day$; and (b) a high mortality rate $\mu_P = 0.1/day$. Various release frequencies are plotted: $m = 1$ (blue curve), $m = 2$ (brown curve), $m = 5$ (magenta curve) and $m = 50$ (light blue curve) releases per year. Initial condition is $P(0^+)$ given in (33), so the solutions are periodic. The yearly released quantity is $\Lambda_P = 3000$ predators. Remaining parameter values are given in Table 1.

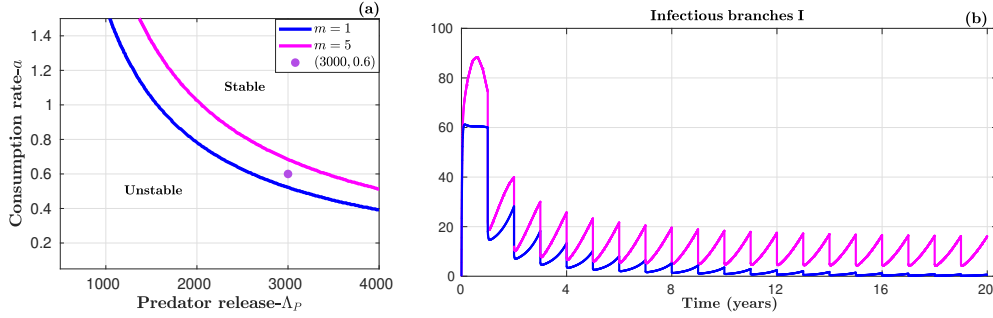


Figure 6: Impact of release frequency on the stability for low predator mortality rate $\mu_P = 0.003/\text{day}$. Subplot (a) presents the stable and unstable regions of the m-cPDFS of system (30) according to parameter pair (Λ_P, a) . The regions are separated by $\mathcal{R}_{c,m} = 1$ for $m = 1$ (blue curve) or $m = 5$ (magenta curve) releases/year. Subplot (b) shows the dynamics of infected leaves I for parameter values $(\Lambda_P = 3000$ predators, $a = 0.6$ spores/predator.day) corresponding to the purple dot in subplot (a). Remaining parameter values are given in Table 1 and initial conditions by (35).

431 mortality μ_p , the low and high values representing respectively a favourable
 432 and unfavourable environment for the predators.

433 Moreover, we consider that an initial predator release occurs at time 0
 434 with the usual initial conditions (22) for the other variables, so that

$$(S(0^+), I(0^+), U(0^+), P(0^+), F(0^+), B(0^+)) = (500, 0, 3000, \frac{\Lambda_P}{m}, 0, 0) \quad (35)$$

435 4.2.1. Impact on stability

436 Herein, we present the impact of the yearly release frequency on the m-
 437 cPDFS stability of controlled system (30), for the two contrasted predator
 438 mortality rates depicted in Figure 5. Only two release frequencies are con-
 439 sidered, $m = 1$ and $m = 5$ releases per year, as results obtained for $m = 5$
 440 also hold for higher frequencies.

441 *Case 1 – Less frequent is more effective (low mortality rate $\mu_P = 0.003/\text{day}$).*
 442 In subplot (a) of Figure 6, $\mathcal{R}_{c,1} = 1$ for $m = 1$ (blue curve) and $\mathcal{R}_{c,5} = 1$ for
 443 $m = 5$ (magenta curve) separate the instability (below) and stability (above)
 444 regions of the m-cPDFS of controlled system (30), when parameters (Λ_P, a)
 445 vary. The stability region is greater for $m = 1$ than for $m = 5$, that is when
 446 the yearly release frequency is lower.

447 Subplot (b) of Figure 6 illustrates the long term dynamics (over 12 years)
 448 of the infected leaves of system (30), with initial conditions (35), for pa-

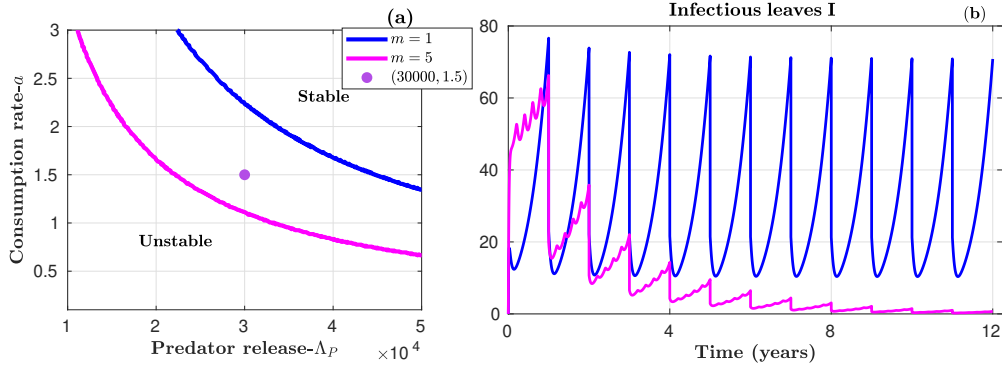


Figure 7: Impact of release frequency on the stability for high predator mortality rate $\mu_P = 0.1/\text{day}$. Subplot (a) presents the stable and unstable regions of the m-cPDFS of system (30) according to parameter pair (Λ_P, a) . The regions separated by $\mathcal{R}_{c,m} = 1$ for $m = 1$ (blue curve) or $m = 5$ (magenta curve) releases/year. Subplot (b) shows the dynamics of infected leaves I for parameter values $(\Lambda_P = 30000 \text{ predators}, a = 1.5 \text{ spores/predator.day})$ corresponding to the purple dot in subplot (a). Remaining parameter values are given in Table 1 and initial conditions by (35)

449 parameter values $\Lambda_P = 3000$ predators and $a = 0.6$ spores/predator.day, which
 450 correspond to the purple dot in subplot (a). As expected from subplot (a),
 451 CLR goes extinct for one release per year (blue curve), while it persists for
 452 five releases per year (magenta curve).

453 Therefore, if the predator mortality is very low, the best strategy for
 454 eradicating CLR is to release predators once a year at the beginning of the
 455 season, implying that releasing less frequently in larger quantity is the most
 456 effective. This is consistent with the control intensity proxy computed in
 457 equation (34), which is higher for lower frequencies.

458 *Case 2 – More frequent is more effective (high mortality rate $\mu_P = 0.1/\text{day}$).*
 459 Figure 7 is built as Figure 6, but for a high predator mortality. In subplot (a),
 460 as opposed to what is observed for low mortality, the stability region is greater
 461 for $m = 5$ than for $m = 1$, that is when the yearly release frequency is higher.

462 Subplot (b) is plotted with parameter values $\Lambda_P = 30000$ predators and
 463 $a = 1.5$ spores/predator.day, which correspond to the purple dot in sub-
 464 plot (a). As expected from subplot (a), CLR extinction is achieved for five
 465 releases per year but not for one release per year. infected leaves I be-
 466 have quite differently. For $m = 1$, I decrease at the beginning of each year
 467 and then increase sharply; indeed, the predator population is large at the

468 beginning of the year, but declines sharply due to the high mortality rate
 469 (see Figure 5(b)), so that CLR is not kept in check. By contrast, for $m = 5$,
 470 predators do not stay close to 0 for long so that no large increase of I occurs.

471 For high predator mortality, the best strategy to control CLR is to release
 472 predators five times per year, meaning that more frequent releases are better.
 473 This conclusion was not obvious, considering that the control intensity proxy
 474 computed in equation (34) is lower for more frequent releases.

475 4.2.2. *Impact on transient dynamics*

476 Herein, we still look at the impact of the yearly release frequency, for
 477 the same contrasted predator mortality rates, but we focus on the first six
 478 years. Four frequency values are considered, namely $m = 1, 2, 5, 50$ releases
 479 per year. We choose the predator parameter values corresponding to a stable
 480 m-cPDFS for all release frequencies considered.

481 *Case 1 – Less frequent is faster (low mortality rate $\mu_P = 0.003/\text{day}$).* Just
 482 as Figure 6(a), subplot (a) of Figure 8 displays the instability (below) and
 483 stability (above) regions of the m-cPDFS of controlled system (30). These
 484 regions are separated by $\mathcal{R}_{c,1} = 1$ for $m = 1$ release/year (blue curve), $\mathcal{R}_{c,2} =$
 485 1 for $m = 2$ releases/year (brown curve), $\mathcal{R}_{c,5} = 1$ for $m = 5$ releases/year
 486 (magenta curve) and $\mathcal{R}_{c,50} = 1$ for $m = 50$ releases/year (light blue curve).
 487 Subplot (a) confirms that for low predator mortality, the best strategy for
 488 controlling CLR is to release the predators once a year at the beginning of
 489 the season.

490 Figure 8 also shows the temporal evolution of infected leaves (subplot (b))
 491 and berries (subplot (c)), using initial conditions (35) and parameter values
 492 $\Lambda_P = 3000$ predators and $a = 0.8$ spores/predator.day. These parameter
 493 values, which correspond to the purple dot in subplot (a), ensure that the m-
 494 CPDFS is stable for all four values of the release frequency m . In subplot (b),
 495 we can observe that the disease is driven to extinction for $m = 1$ (blue curve).
 496 For $m = 5$ (magenta curve) and $m = 50$ (light blue curve), the number of
 497 infected leaves after six years is still notably greater than zero (less notably
 498 so for $m = 2$, brown curve). However, if we increase the number of years,
 499 the extinction will be observed for all values of m .

500 Subplot (c) shows that the number of berries at the end of each year
 501 decreases when m increases. This confirms our previous result: the best
 502 strategy for low predator mortality is to few releases per year.

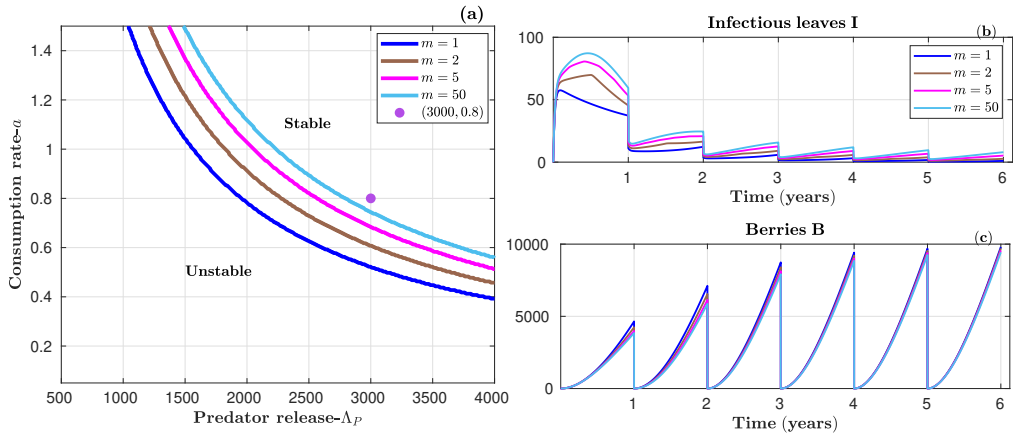


Figure 8: Impact of release frequency on the transient dynamics with low predator mortality. Subplot (a) presents the stable and unstable regions of the m -cPDFS of system (30) according to parameter pair (Λ_P, a) . The regions are separated by $\mathcal{R}_{c,m} = 1$ for $m = 1$ (blue curve), $m = 2$ (brown curve), $m = 5$ (magenta curve) or $m = 50$ (light blue curve) releases/year. Subplots (b) and (c) show the dynamics of infected leaves I and berries B for parameter values $(\Lambda_P = 3000$ predators, $a = 0.8$ spores/predator.day) corresponding to the purple dot in subplot (a). Remaining parameter values are given in Table 1 and initial conditions by (35).

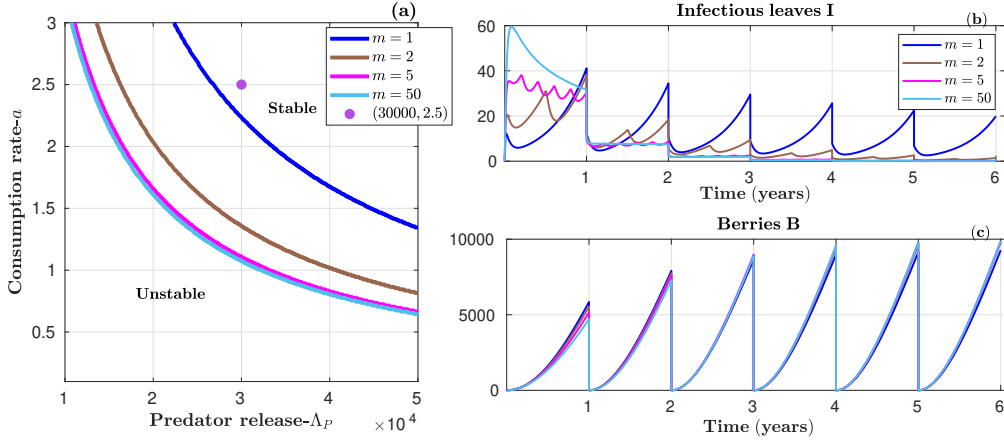


Figure 9: Impact of release frequency on the transient dynamics with high predator mortality. Subplot (a) presents the stable and unstable regions of the m -cPDFS of system (30) according to parameter pair (Λ_P, a) . The regions are separated by $\mathcal{R}_{c,m} = 1$ for $m = 1$ (blue curve), $m = 2$ (brown curve), $m = 5$ (magenta curve) or $m = 50$ (light blue curve) releases/year. Subplots (b) and (c) show the dynamics of infected leaves I and berries B for parameter values $(\Lambda_P = 30000$ predators, $a = 2.5$ spores/predator.day) corresponding to the purple dot in subplot (a). Remaining parameter values are given in Table 1 and initial conditions by (35).

503 *Case 2 – Best strategy depends on the horizon (high mortality rate $\mu_P =$*
 504 *0.1).* As expected for high predator mortality, in subplot (a) of Figure 9,
 505 the $\mathcal{R}_{c,m} = 1$ curves, delimiting the stable and unstable regions of the m -
 506 cPDFS, are in reverse order compared to Figure 8(a). However, we should
 507 note that $\mathcal{R}_{c,5} = 1$ (magenta curve) and $\mathcal{R}_{c,50} = 1$ (light blue curve)
 508 are almost indistinguishable, implying that both strategies are almost equivalent
 509 in terms of eradication success.

510 Subplots (b) and (c) of Figure 9 are built using parameter values $\Lambda_P =$
 511 30000 predators and $a = 2.5$ spores/predator.day, which correspond to the
 512 purple dot in subplot (a)). In subplot (b), we observe CLR extinction in all
 513 cases but for $m = 1$, for which extinction takes longer. In the latter case,
 514 the high mortality rate drives the predator population to very small values
 515 very quickly so that it is absent for most of the season, which slows down the
 516 control process.

517 To better explain the impact of the high predator mortality on berries
 518 depicted in subplot (c), we consider the number of berries at the end of
 519 each season and thus we obtain Table 2. The majority of parameter values

520 of the model are certainly assumed, but this qualitative study shows the
 521 effectiveness of the biocontrol of CLR using predator

Table 2: Berry production at the end of each season, for different yearly release frequencies and high predator mortality. Production values correspond to subplot (c) of Figure 9. The optimal production for each year is enhanced in bold.

	Releases per year			
	$m = 1$	$m = 2$	$m = 5$	$m = 50$
year 1	5845	5554	5131	4717
year 2	7906	7756	7600	7290
year 3	8663	8866	8985	8897
year 4	8987	9399	9568	9553
year 5	9159	9657	9793	9796
year 6	9270	9785	9876	9880

522 In terms of berry production, the best strategy from the first year to the
 523 second year is to release the predators once. From year 3 to year 4, the
 524 highest berry production occurs with $m = 5$ releases per year. From year
 525 5 and on, the production increases with the release frequency, which is the
 526 result expected in the case of high predator mortality. However, except for
 527 $m = 1$, the berry production numbers are then almost all identical since
 528 CLR is nearly eradicated. Therefore, the best release strategy depends on
 529 the time horizon considered.

530 4.2.3. Summary

531 From the four cases described above, we can conclude that the best predat-
 532 or release strategy, in terms of CLR control and berry production, strongly
 533 depends on predator characteristics, namely its mortality rate (μ_P) and its
 534 uredospore consumption rate (a). Both parameters determine the yearly
 535 released quantity of predators (Λ_P) needed to control CLR. Moreover, the
 536 mortality rate has an impact on the best predator release frequency (m). On
 537 the long run, more frequent is more effective for high predator mortality, and
 538 vice versa for low mortality. On the shorter run however, less frequent may
 539 also be better for high mortality.

540 5. Discussion

541 In this work, we built an original impulsive model of coffee leaf rust
 542 dynamics in a coffee plantation, in which the non production (dry) season

543 takes the form of an impulse, it is based in a previous study [43]. There are
544 few CLR models in the literature and they represent different geographical
545 scales, from the individual coffee bush to the country or even the continent.
546 Bebber et al. determined the germination and infection risk depending on
547 the climate in Colombia and neighbouring countries, based upon existing
548 experimental data [44]. In contrast to these static approaches, Vandermeer
549 et al. studied the interaction between the regional and local dynamics of CLR
550 model by representing the evolution of the proportion of infected bushes and
551 farms [45]. Vandermeer et al. also represented the CLR dynamics in a coffee
552 farm in Chiapas using an SI (susceptible–infected) epidemiological model of
553 the host and concluded that the network approach can be a useful way of
554 gaining qualitative insight about disease dynamics in space [46].

555 The originality of our model, compared to the dynamical approaches
556 above, is the hybrid formalism that is particularly suited to describe the sea-
557 sonality of coffee and rust dynamics, as well as impulsive biocontrol. Floquet
558 theory was used to compute stability thresholds for the periodic disease-free
559 solution. The initial coffee leaf rust model was then coupled with the dynam-
560 ics of predators, as recommended by Zambolin [33] for alternative biocontrol
561 methods; this resulted in a controlled impulsive model. The semi-numerical
562 analysis of this controlled impulsive model allowed to conclude that predator-
563 based biocontrol can drastically reduce coffee leaf rust. Moreover, we studied
564 how successful biocontrol implementation depends on predator characteris-
565 tics, in particular its mortality.

566 We set the study in a framework where the yearly released quantity of
567 predators is fixed and only its release frequency varies. We deemed this
568 comparison relevant since, whatever the release strategy, the same yearly
569 budget may be allocated to biocontrol agents.

570 Low and high predator mortality rates were considered to represent dif-
571 ferent environmental situations and predator characteristics. High mortal-
572 ity occurs if the predator is a specialist parasite like *Mycodiplosis*[22], which
573 means that it only consumes CLR uredospores, or if it is a generalist predator
574 whose alternate food sources are absent; in both cases, the predator quickly
575 dies out in absence of CLR. Low mortality occurs for generalist predators
576 in a rich environment. Most predators of *H. vastatrix* in the literature have
577 alternative preys. For example, the *Lecanicillium lecanii* fungus is a CLR
578 hyperparasite that could be used for biocontrol [28, 47], and it is also a
579 pathogen of plant-parasitic nematodes [48]. Hence, both low and high mor-
580 tality situations could occur in the field.

581 For low mortality rates, the best strategy is to release the predators once
582 a year at the beginning of the production season. This strategy is efficient
583 because it is the one that ensures the largest average predator density over
584 the year, predators being always present because of the low mortality rate.
585 However, the cost of such a large release at the beginning of the production
586 season, on top of all the actions that need to be taken for coffee farming
587 at that same time (such as fertiliser and manure application, or trimming
588 of dry or excess leaves), can put too large a financial burden on the coffee
589 producer. This problematic represents a well-known trade-off between yield
590 and affordability in farming practices. Another potential problem is that
591 massively advising farmers to adopt these practices could cause an excessive
592 increase in the local demand of predators, and consequently a shortage in the
593 market. For both these reasons, a strategy considering two releases could be
594 recommended, even though it is less cost-efficient.

595 When the predator mortality rate is high, the best strategy depends on
596 the horizon. For the first year, the most efficient approach is to have a large
597 release at the beginning of the season in order to have an immediate mas-
598 sive impact. For the subsequent years, 5 and then 50 releases per year are
599 advocated in Table 2. However, there is little difference in berry production
600 between 5 and 50 releases in years 5 and 6. Moreover, the eradication success
601 is almost identical for both strategies in terms of yearly released quantity of
602 predators needed to control CLR (see Figure 9(a)). Considering the burden
603 and cost associated with having 50 interventions over the production season,
604 five yearly releases should be preferred to 50. Releasing predators twice a
605 year may appear slightly suboptimal in terms of eradication success. It is
606 however fairly close to the berry production achieved when releases occur five
607 times per year and it is less labour consuming. In terms of cost-effectiveness,
608 two releases per year might then be a good compromise. This result is com-
609 plementary to the results of Henk et al [22], which shows that the larvae of
610 some *Mycodiplosis* species can feed on spores of rust fungi and their frequency
611 play an important role.

612 In the present work, we have focused our attention on one family of
613 strategies: those that repeat themselves every year and are evenly spread
614 over the production season, with a first release at the beginning of each
615 year. Potential generalisations are numerous, but could not be considered
616 here. One could for example choose to change strategy every year; this would
617 seem to be a good idea if we look at Table 2, where we would choose to have
618 a large single release the first year, and potentially five releases per year after

619 that. One could also choose not to have a first release at the beginning of the
620 year, but later in the season, or to have a larger release at the beginning of the
621 year and then some smaller ones during the year. Finally, predators could
622 be released in reaction to peaks of uredospore densities in the plantation,
623 so as to maximise their impact; the latter approach however requires close
624 monitoring of the disease in the plantation. In addition to them potentially
625 being more efficient, these approaches have the advantage of not imposing
626 the full burden of acquiring predators at the beginning of the season.

627 The continuous part of our model contains most elements that we deem
628 relevant, keeping it reasonably small and tractable. However, uredospore ger-
629 mination and mycelium formation depend on various environmental factors
630 [49], such as temperature [50]. Introducing meteorological data in our model
631 would allow for a better description of the day-to-day dynamics, but constant
632 parameters gives a fairly good representation of the average dynamics for our
633 purpose.

634 The hybrid model we developed is able to describe the dynamics of other
635 fungal diseases that attack leaves of perennial plants. It appears as a promis-
636 ing tool to explore the efficiency of biocontrol strategies based on the impul-
637 sive release of predators. The impulsive component of the model presented
638 in this work corresponds to plants that are harvested once a year; such is the
639 case in temperate climate or in tropical climate with dry and rainy seasons.
640 However, this model can be perfectly adapted to other countries without
641 such contrasted seasons, where there are more than one harvest per year. In
642 this case, several impulses can occur over the year, corresponding to partial
643 harvests only, and not seasonality.

644 To conclude, we were able to obtain analytical results on the qualitative
645 behaviour of our model without control. Compared to purely numerical anal-
646 yses, our semi-numerical analyses hold for large parameter regions. This is
647 particularly true for one of our main results, regarding the best predator re-
648 lease frequency: for a low predator mortality, one release per season is better
649 than five, and vice versa for high predator mortality. However, the minimum
650 yearly quantity of predators that need to be released to control the disease
651 depends on the model parameter values. Therefore, even if this research pro-
652 vides valuable insights on predator-based biocontrol, further research, most
653 notably around parameter calibration, would be needed to complete the step
654 from theory to practice. Furthermore, the conclusions that we have drawn
655 for CLR might not translate to other pathogen-plant pairs in other contexts,
656 beyond our analytical results; a full semi-analytical study and numerical sim-

657 ulations should be performed for each case.

658 **Acknowledgments**

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660 LIRIMA (<https://team.inria.fr/epitag/>)

661 **Appendix A. Parameter values**

662 We present in this appendix how parameter values presented in Table 1
663 were set.

- 664 • The maturation of flowers takes 180 days, which represents stage I +
665 stage II + stage III presented by Torres Castillo et al. [41], so the
666 maturation rate is $\theta = 1/180 \approx 0.0055/$ day.
- 667 • In the coffee tree, a node produces 30 flowers during the flowering period
668 [40, page 59]. Each node has 2 leaves, so a leaf corresponds to 15 flowers.
669 The production of flowers in the model is hence $\delta_S = 15/180 \approx 0.08$
670 flowers/leaf.day. We suppose that $\delta_I = 0.04 < \text{flowers/leaf.day}$ due to
671 the disease.
- 672 • Out of 100 flowers, 65 set fruit after pollination [40, page 28]. This
673 implies that $e^{-180\mu_F} = 0.65$, so $\mu_F = 2.4 \times 10^{-3}$ per day. We suppose
674 that the berries have the same mortality rate, so $\mu_B = 2.4 \times 10^{-3}$ per
675 day.
- 676 • Leaf mortality during the rainy season is fairly low. The rainy season
677 lasts 250 days. We assumed that leaves survive on average 194 days.
678 Then $\mu = 1/194 \text{ day} = 0.0034 \text{ day}^{-1}$.
- 679 • Leaf mortality during the dry season is naturally occurring, caused
680 by either the dry season or by man pruning the trees. We set the
681 leaf survival to the dry season to $\varphi_S = 0.7$ for susceptible leaves and
682 $\varphi_I = 0.4$ for infected leaves, branches with CLR-affected leaves being
683 preferentially targeted for pruning.
- 684 • Lesions form on the leaves 2 to 3 weeks after the spores germinate.
685 These lesions will directly cause the leaf to die; it may take longer, but
686 we have assumed that the average time is $(14+21)/2=18$ days. Then
687 $d = 1/18 \text{ days} = 0.056 \text{ day}^{-1}$.

- 688 • The values of parameters $\Lambda, \mu_U, \varphi_U, \varphi_P$, and K were assumed.
- 689 • The parameter values μ_P are chosen to have high and low mortality of
- 690 predator

691 Appendix B. Properties of the m-cPDFS

692 Appendix B.1. Expression of the m-cPDFS

693 Solving equation $\dot{P} = -\mu_P P$ for $t \in]\frac{jT}{m}, \frac{(j+1)T}{m}]$ we obtain

$$P(t) = P\left(\frac{jT^+}{m}\right) e^{-\mu_P(t-\frac{jT}{m})}. \quad (\text{B.1})$$

694 Using equation (B.1) and the second equation of (31) we have

$$P\left(\frac{(j+1)T^+}{m}\right) = P\left(\frac{jT^+}{m}\right) e^{-\mu_P \frac{T}{m}} + \frac{\Lambda_P}{m}.$$

695 For $j = 0$

$$P\left(\frac{T^+}{m}\right) = P(0^+) e^{-\mu_P \frac{T}{m}} + \frac{\Lambda_P}{m}.$$

696 For $j = 1$

$$\begin{aligned} P\left(\frac{2T^+}{m}\right) &= \left(P(0^+) e^{-\mu_P \frac{T}{m}} + \frac{\Lambda_P}{m}\right) e^{-\mu_P \frac{T}{m}} + \frac{\Lambda_P}{m}; \\ &= P(0^+) e^{-\mu_P \frac{2T}{m}} + \frac{\Lambda_P}{m} e^{-\mu_P \frac{T}{m}} + \frac{\Lambda_P}{m}. \end{aligned}$$

697 For $j = 2$

$$P\left(\frac{3T^+}{m}\right) = P(0^+) e^{-\mu_P \frac{3T}{m}} + \frac{\Lambda_P}{m} \sum_{i=0}^2 e^{-\mu_P \frac{iT}{m}}.$$

698 Generally, for all $j = 0, \dots, m-1$

$$\begin{aligned} P\left(\frac{jT^+}{m}\right) &= P(0^+) e^{-\mu_P \frac{jT}{m}} + \frac{\Lambda_P}{m} \sum_{i=0}^{j-1} e^{-\mu_P \frac{iT}{m}}; \\ &= P(0^+) e^{-\mu_P \frac{jT}{m}} + \frac{\Lambda_P}{m} \left(\frac{1 - e^{-\mu_P \frac{jT}{m}}}{1 - e^{-\mu_P \frac{T}{m}}}\right), \end{aligned}$$

699 in particular for $j = m - 1$, that is

$$P\left(\frac{(m-1)T^+}{m}\right) = P(0^+) e^{-\mu_P \frac{(m-1)T}{m}} + \frac{\Lambda_P}{m} \left(\frac{1 - e^{-\mu_P \frac{(m-1)T}{m}}}{1 - e^{-\mu_P \frac{T}{m}}} \right).$$

700 Using this latter expression and the third equation of (31), we obtain

$$P(T^+) = \varphi_P \left[P(0^+) e^{-\mu_P T} + \frac{\Lambda_P}{m} \left(\frac{e^{-\mu_P \frac{T}{m}} - e^{-\mu_P T}}{1 - e^{-\mu_P \frac{T}{m}}} \right) \right] + \frac{\Lambda_P}{m}.$$

701 The value for which $P(T^+) = P(0^+)$ is

$$P(0^+) = \frac{\Lambda_P}{m} \left[\frac{\varphi_P \left(e^{-\mu_P \frac{T}{m}} - e^{-\mu_P T} \right) + 1 - e^{-\mu_P \frac{T}{m}}}{(1 - \varphi_P e^{-\mu_P T}) \left(1 - e^{-\mu_P \frac{T}{m}} \right)} \right].$$

702 So we can conclude that, for $t \in]\frac{jT}{m}, \frac{(j+1)T}{m}]$, the m-cPDFS is given by $Z^T(t) =$
 703 $(S^T(t), 0, 0, P_{m,j}^T(t))$, with $S^T(t)$ given by equation (11) and

$$P_{m,j}^T(t) = \left[P(0^+) e^{-\mu_P \frac{jT}{m}} + \frac{\Lambda_P}{m} \left(\frac{1 - e^{-\mu_P \frac{jT}{m}}}{1 - e^{-\mu_P \frac{T}{m}}} \right) \right] e^{-\mu_P (t - \frac{jT}{m})}, \quad (\text{B.2})$$

704 $P(0^+)$ being defined above.

705 *Appendix B.2. Expression of the yearly average number of predators*

706 The yearly average number of predators for the m-cPDFS is computed
 707 over the interval $[0, T]$ as follows:

$$g(m) = \int_0^T P^T(t) dt = \sum_{j=0}^{m-1} \int_{\frac{jT}{m}}^{\frac{(j+1)T}{m}} P_{m,j}^T(t) dt.$$

708 Replacing $P_{m,j}^T(t)$ by its expression in equation (32), we obtain

$$\begin{aligned} g(m) &= \sum_{j=0}^{m-1} \int_{\frac{jT}{m}}^{\frac{(j+1)T}{m}} \left[P(0^+) e^{-\mu_P \frac{jT}{m}} + \frac{\Lambda_P}{m} \left(\frac{1 - e^{-\mu_P \frac{jT}{m}}}{1 - e^{-\mu_P \frac{T}{m}}} \right) \right] e^{-\mu_P (t - \frac{jT}{m})} dt; \\ &= \sum_{j=0}^{m-1} \left[P(0^+) e^{-\mu_P \frac{jT}{m}} + \frac{\Lambda_P}{m} \left(\frac{1 - e^{-\mu_P \frac{jT}{m}}}{1 - e^{-\mu_P \frac{T}{m}}} \right) \right] \int_{\frac{jT}{m}}^{\frac{(j+1)T}{m}} e^{-\mu_P (t - \frac{jT}{m})} dt; \\ &= \sum_{j=0}^{m-1} \left[P(0^+) e^{-\mu_P \frac{jT}{m}} + \frac{\Lambda_P}{m} \left(\frac{1 - e^{-\mu_P \frac{jT}{m}}}{1 - e^{-\mu_P \frac{T}{m}}} \right) \right] \left[\frac{1 - e^{-\mu_P \frac{T}{m}}}{\mu_P} \right]. \end{aligned}$$

709 Replacing $P(0^+)$ by its expression in equation (33), we obtain

$$\begin{aligned}
g(m) &= \sum_{j=0}^{m-1} \left[\frac{\Lambda_P}{m} \left(\frac{\varphi_P \left(e^{-\mu_P \frac{T}{m}} - e^{-\mu_P T} \right) + 1 - e^{-\mu_P \frac{T}{m}}}{(1 - \varphi_P e^{-\mu_P T}) \left(1 - e^{-\mu_P \frac{T}{m}} \right)} \right) e^{-\mu_P \frac{jT}{m}} \right] \\
&\quad + \sum_{j=0}^{m-1} \left[\frac{\Lambda_P}{m} \left(\frac{1 - e^{-\mu_P \frac{jT}{m}}}{1 - e^{-\mu_P \frac{T}{m}}} \right) \right] \left[\frac{1 - e^{-\mu_P \frac{T}{m}}}{\mu_P} \right], \\
&= \frac{\Lambda_P}{m\mu_P} \sum_{j=0}^{m-1} \left[\frac{\varphi_P \left(e^{-\mu_P \frac{T}{m}} - e^{-\mu_P T} \right) + 1 - e^{-\mu_P \frac{T}{m}}}{(1 - \varphi_P e^{-\mu_P T})} e^{-\mu_P \frac{jT}{m}} + 1 - e^{-\mu_P \frac{jT}{m}} \right], \\
&= \frac{\Lambda_P}{m\mu_P} \left[\left(\frac{\varphi_P \left(e^{-\mu_P \frac{T}{m}} - e^{-\mu_P T} \right) + 1 - e^{-\mu_P \frac{T}{m}}}{(1 - \varphi_P e^{-\mu_P T})} \right) \left(\frac{1 - e^{-\mu_P T}}{1 - e^{-\mu_P \frac{T}{m}}} \right) \right. \\
&\quad \left. + m - \frac{1 - e^{-\mu_P T}}{1 - e^{-\mu_P \frac{T}{m}}} \right], \\
&= \frac{\Lambda_P}{m\mu_P} \left[\left(\frac{(\varphi_P - 1)e^{-\mu_P \frac{T}{m}}}{(1 - \varphi_P e^{-\mu_P T})} \right) \left(\frac{1 - e^{-\mu_P T}}{1 - e^{-\mu_P \frac{T}{m}}} \right) + m \right].
\end{aligned}$$

710 Simplifying this expression, we finally obtain

$$g(m) = \left(\frac{-\Lambda_P(1 - \varphi_P)(1 - e^{-\mu_P T})}{\mu_P(1 - \varphi_P e^{-\mu_P T})} \right) \frac{1}{m(e^{\mu_P \frac{T}{m}} - 1)} + \frac{\Lambda_P}{\mu_P}.$$

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