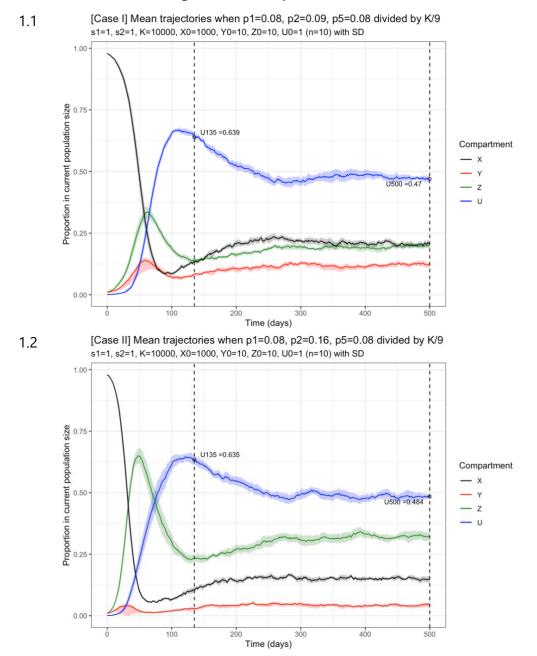
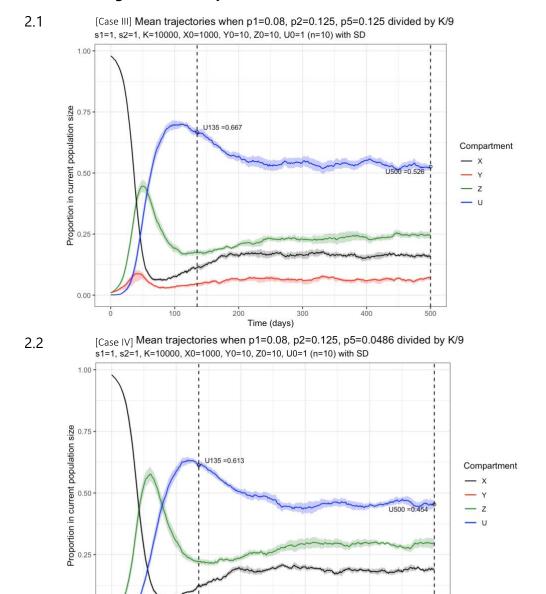
#### **Supplementary Figure**

Supplementary Figure 1.1 and 1.2: Progress curve of Rice Tungro Disease (RTD) from stochastic runs examining the effect of  $p_2$ 



The mean proportions of each host compartments: the susceptible (X), the RTBV-infected (Y), the RTSV-infected (Z) and the co-infected (U), colour-coded as in the legend, from 10 simulations were plotted with SD (shaded areas). The transmission-related parameters and initial conditions used are stated in the titles; the only difference was  $p_2$ : Case I used  $p_2$ = 0.09/(K/9) based on HC1996 paper, lower than  $p_2$ = 0.16/(K/9) based on BD2017 paper in Case II. They had similar U trajectories, although Z hit a higher peak in Case II.

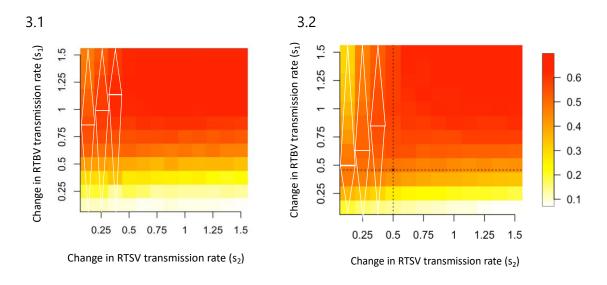
### Supplementary Figure 2.1 and 2.2: Progress curve of RTD dynamics from stochastic runs examining the effect of $p_5$



The mean proportions of each host compartments: the susceptible (X), the RTBV-infected (Y), the RTSV-infected (Z) and the co-infected (U), colour-coded as in the legend, from 10 simulations were plotted with SD (shaded areas). The transmission-related parameters and initial conditions used are stated in the titles; the only difference was  $p_5$ : Case III used  $p_5 = 0.125/(K/9)$  based on HC1996 paper, lower than  $p_5 = 0.0486/(K/9)$  based on BD2017 paper in Case IV. They had similar U trajectories, as well as other compartments.

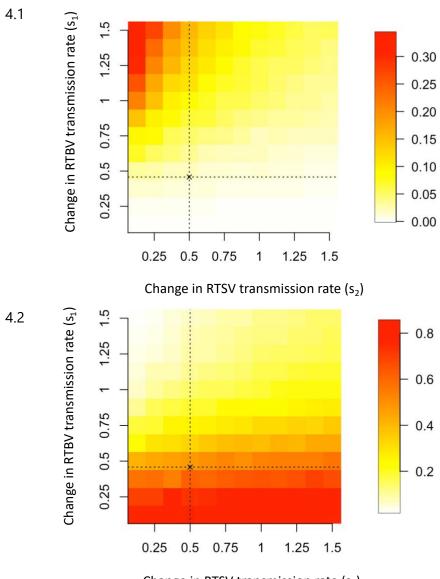
Time (days)

# Supplementary Figure 3.1 and 3.2: The two-way $s_1$ - $s_2$ scan outcome of the co-infected (U) at day 135 that can show the negative relationship between RTBV and RTSV.



 $s_1$  is the change in transmission rate of RTBV due to a source previously being infected by RTSV and  $s_2$  is the change in transmission rate of RTSV due to a source previously being infected by RTBV. Color represents the value of the proportion of RTD-affected host at day 135 (U135) as shown in the legend. The white triangles represent the gradient of the colour, with the base being the darkest tone and where the trend of colour change reversed. This reversal happened earlier with the lower  $s_2$ . The two different initial conditions were used with the same  $p_{1d}$ ,  $p_{2d}$ , and  $p_{5d}$ : Y0=Z0=10 and U0=1 in Fig 3.1 and Y0=Z0=U0=100 in Fig. 3.2, while X0=1000 in both. Regardless of the initial conditions, when  $s_2$  was low, increasing  $s_1$  increased U135 at first, but then caused it to drop (this can potentially be seen for all  $s_2$  if we were to increase the scanning range). It may mean that there was a threshold of the RTSV level in the system, above which RTBV cannot persist.

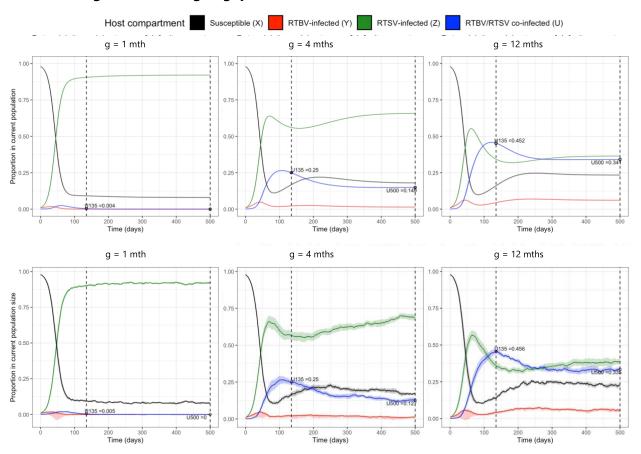
# Supplementary Figure 4.1 and 4.2: The two-way $s_1$ - $s_2$ scan outcome of the RTBV-infected (Y) and the RTSV-infected (Z) at day 135, respectively.



Change in RTSV transmission rate (s<sub>2</sub>)

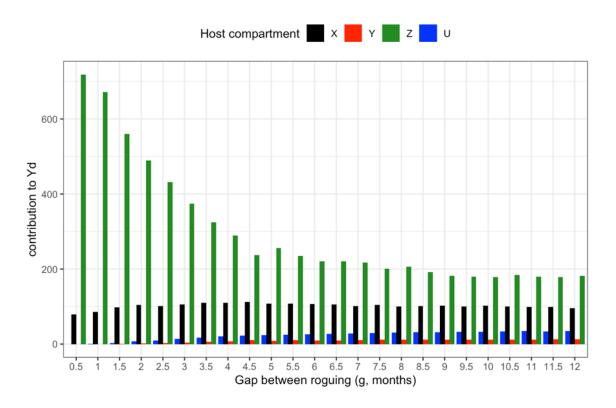
 $s_1$  is the change in transmission rate of RTBV due to a source previously being infected by RTSV and  $s_2$  is the change in transmission rate of RTSV due to a source previously being infected by RTBV. RTBV singly infected hosts (Y) can only increase when  $s_1$  is high enough. Similarly, RTSV singly infected hosts (Z) can only increase when  $s_2$  is high enough, but with a lower threshold. Also note the difference in the color scale; Z can increase to a larger proportion than Y. In almost all cases, Z dominated Y, well-reflected by 1) its low death rate due to little to no symptoms and 2) the higher transmission rate ( $p_2$ ) compared to RTBV transmission rate ( $p_1$ ). A cross (×) represents U135 proportion, given by this pair  $s_1$  and  $s_2$  equal to 0.458 and 0.500, respectively.

## Supplementary Figure 5.1: Examples of progress curves from rogued situations with different length of inter-rogue gaps.



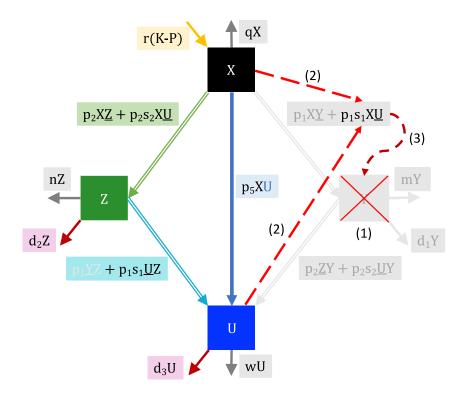
The first row was the trajectories from deterministic simulations and the second row showed the mean from 10 stochastic runs. There was no difference from both model for all gaps. Initial conditions used were X0=1000, Y0=10=Z0, U0=1. Parameters were default shown in Sup. Table 3.1 and Appendix 5. From left to right, the gap was 1, 4, and 12 months. As the gap was longer, the proportion of Z at day 135 markedly increase, U135 and Y135 decreased and X135 hovered around 0.10.

## Supplementary Figure 5.2: The yield contribution of each host compartment (based on *Yd*; equation (6)).



For all roguing gaps, RTSV-infected hosts (Z) contributed the most to the total yield (Yd) due to 1) its high abundance and 2) its high yield coefficient ( $c_2 = 0.78$ ), followed by the healthy hosts (X). The least contributing compartment was RTBV-infected hosts as it got rogued, leading to a low abundance and its low yield coefficient ( $c_1 = 0.46$ ), although this was still more than that of the co-infected (RTD-affected) hosts (U) ( $c_3 = 0.12$ ).

#### Supplementary Figure 6: Exemplified case of one-compartment extinction case.



- (1) RTBV-infected hosts (Y) become extinct, making to all transmission events contributed by the inoculum Y and demographic changes of Y) become non-feasible (shown by grey letters and boxes)
- (2) However, non-zero susceptible hosts (X) and the co-infected (U) can contribute to one pathway that leads to the production of Y
- (3) As a result, Y recovers from zero.

The likely recovery from zero of host compartments also applies to RTSV-infected (Z) and U compartments. This makes the simultaneous extinction of two or three compartments unlikely.

#### **Supplementary Table**

**Supplementary Table 1:** Probability of events and its consequence on the abundance of

each host compartment (plants per area).

each nost compartment (plants per alea).	Probability	Consequence**			
Event	(Divided by T*)	X	Y	Z	U
Planting of new susceptible plant	r(K-P)	+1	0	0	0
Death of the susceptible	qX	-1	0	0	0
Death of the RTBV singly infected	mY	0	-1	0	0
Death of the RTSV singly infected	nZ	0	0	-1	0
Death of the RTBV/RTSV doubly infected	wU	0	0	0	-1
Transmission of RTBV into the susceptible	$p_1(Y+s_1U)X$	-1	+1	0	0
Transmission of RTSV into the susceptible	$p_2(Z+s_2U)X$	-1	0	+1	0
Co-transmission of RTBV and RTSV into the susceptible	$p_5UX$	-1	0	0	+1
Transmission of RTBV into the RTSV singly infected	$p_1(Y+s_1U)Z$	0	0	-1	+1
Transmission of RTSV into the RTBV singly infected	$p_2(Z+s_2U)Y$	0	-1	0	+1
Roguing of the RTBV singly infected	$d_1Y$	0	-1	0	0
Roguing of the RTSV singly infected	$d_2Z$	0	0	-1	0
Roguing of the RTBV/RTSV doubly infected	$d_3U$	0	0	0	-1

<sup>\*</sup>Total rate (T)=  $r(K-P)+p_1(Y+s_1U)X+p_2(Z+s_2U)X+p_5UX+p_2(Z+s_2U)Y+p_1(Y+s_1U)Z+qX+mY+nZ+wU+d_1Y+d_2Z+d_3U$ 

<sup>\*\*</sup> Increase or decrease by 1 [plant][area]<sup>-1</sup> is represented by +1 or -1, respectively, and 0 means no change

**Supplementary Table 2**: Parameter value summary from HC1996 and BD2017 paper

Virus transmission events				Parameters
Virus that infects	Transition	Source	HC1996 paper (t is probabilities)	BD2017 paper (Each to be divided by current total host abundance)
Case1: +RTBV	X to Y or Z to U	Υ	$t_2 = 0.08$	Sigma = 0.08
Case2: +RTSV	X to Z or Y to U	Z	$t_1 = 0.16$	Beta = 0.09
Case3: +RTBV	X to Y or	U	$t_3 = 0.02$	Tau = $0.06$ (s <sub>1</sub> = $0.75$ )
	Z to U	- (s <sub>1</sub> = 0.25)		Lambda = $0.03 (s_1 = 0.375)$
Case4: +RTSV	X to Z or	U	$t_2 = 0.08$	Gamma = 0.01 (s <sub>2</sub> = 0.125)
	Y to U	-	$(s_2 = 0.5)$	Delta = 0.07 (s <sub>2</sub> = 0.875)
Case5: +both	X to U	U	t <sub>1</sub> = 0.16	Alpha = 0.035
	Other events		HC1996 paper	BD2017 paper
Host planting ra	te		-	r = 0.001
Maximum host a	abundance when bi	irth rate is zero	-	P <sub>0</sub> = 20000
Death rate of X			-	$q_0 = 0.008$
Death rate of Y		rate of Y		$q_1 = 0.009$
Death rate of Z		th rate of Z		q <sub>2</sub> = 0.0125
Death rate of U			-	q <sub>3</sub> = 0.025
Roguing efficiency for Y, Z, and U, respectively			0.7, 0.0, 1.0	-

<sup>\*</sup>Note that mean value of  $s_1 = 0.458$  and  $s_2 = 0.500$ 

### **Supplementary Table 3.1:** Fixed values of parameters used for comparative runs (Method 1.3; excluding \*) and parameterisation (Method 2; including \*)

Events or Constants	Parameter	Value
Host planting rate	r	0.001
Maximum host abundance when birth rate is zero	K	10000
Death rate of X	q	0.0080
Death rate of Y	m	0.0125
Death rate of Z	n	0.0090
Death rate of U	w	0.0250
*(RTBV transmission from RTBV-singly infected host (Y))	( <b>p</b> <sub>1</sub> )	(0.008/(K/9))

#### **Supplementary Table 3.2:** Ranges of non-fixed parameters for comparative runs

<b>Events or Constants</b>	Parameter	Range
<ul> <li>RTBV transmission from RTBV-singly infected host (Y)</li> <li>RTSV transmission from RTSV-singly infected host (Z)</li> <li>Co-transmission of RTBV and RTSV from RTBV/RTSV-doubly infected (U)</li> </ul>	$\begin{matrix}p_1\\p_2\\p_5\end{matrix}$	0.08/(K/9) – 0.16/(K/9)
<ul> <li>Change in transmission rate of RTBV due to a source previously being infected by RTSV</li> <li>Change in transmission rate of RTSV due to a source previously being infected by RTBV</li> </ul>	s <sub>1</sub> s <sub>2</sub>	0.125-2.00

#### **Supplementary Table 3.3:** Six sets of initial conditions used for comparative runs

Initial Condition Set ID	Х0	Y0	<b>Z</b> 0	U0
1	1000	1	1	0
2	1000	100	200	0
3	1000	200	100	0
4	1000	100	100	100
5	7500	100	100	100
6	7500	500	500	500

### **Supplementary Table 3.4:** The "extreme" parameter set that produced all-host out-of-range ratios (more than 1.5 or less than 0.5).

$p_1$	$p_2$	$p_5$	$s_1$	$s_2$
0.09/(K/9)	0.13/(K/9)	0.16/(K/9)	1.7	0.7
initial set	X0	Υ0	Z0	U0
1	1000	1	1	0

## **Supplementary Table 4:** Host abundances at day 135 and index *Yd* (equation (6)) in the unrouged situation with the default parameters shown Appendix 4 (Table B)

	Host Abundances				Total Abundance	Index
Model	X135	Y135	Z135	U135	(P135)	Yd
Deterministic	112	52	97	307	568	248
Stochastic	121	49	94	294	558	252

The numbers are rounded to an integer. The units are plants per area

#### **Appendix**

#### Appendix 1: Construction of the death rate due to roguing $(d_i)$

The probability of detection ( $e_i$ ) of different infected compartments were assigned based on Holt & Chancellor (1996) (Sup. Table 1). We were interested in the effect of duration between roguing operation (g) on the yield. The roguing-related death rate ( $d_i$ ) took into account both parameters.

The death rate of each infected host due to roguing ( $d_i$ ) is the reciprocal of the average effective infectious period of that host which depends on roguing gap (g). The effective infectious period is equal to the duration between the round of rouging before the plant becomes infectious/symptomatic and the round that the host gets rogued on average (duration A) subtracted by the duration between the final round of rouging and the emergence of infectivity/symptom (duration B).

Duration  $\bf A$  is equal to the gap if the probability of detection ( $\bf e$ ) is 1.0 (no host can escape the roguing), but it gets longer than the gap as the probability decreases. The increase in the duration is by the factor of  $1/\bf e$  as the number of roguing rounds required for detecting hosts after it becomes infectious/symptomatic is also increased by this factor (when it is assumed to be geometric random variable, with the average  $1/\bf e$ ). For example, if 50% infected hosts get detected at each roguing ( $\bf e$  = 0.5), on average, it will take 2 rounds (1/0.5) of roguing to detect and rogue these hosts, meaning the duration  $\bf A$  is also doubled ( $\bf g/\bf e$  =  $\bf g$ /0.5). In our model, we ignored the consideration of the latent and incubation period, so the emergence of infectivity and symptoms were virtually at the same time (when the virus(es) got inoculated onto the host).

Duration  $\mathbf{B}$ , on average, is the half of the gap length ( $\mathbf{g}$ /2) when we assume the uniform distribution of the transition from X to U (or to Z) between rounds of roguing for each roguing gap ( $\mathbf{g}$ ). This requires us to ignore the potential change in the rates of the transition due to the change in the abundance of each host compartment between rounds of roguing. In other words,

we assume that X can become U (or to Z) at any time point with the identical probability within the gap and as time passes. Therefore, we obtained equation (5) as follow:

Death rate due to roguing 
$$(d_i) = \frac{1}{\text{Effective infectious period}} = \frac{1}{\text{Duration A - Duration B}} = \frac{1}{\frac{g}{e_i} - \frac{g}{2}}$$

#### Appendix 2: Normalisation of p<sub>1</sub> and the other transmission rates

Holt & Chancellor (1996, [8]) and Blas & David (2017, [9]) both considered plant and vector dynamics in their model. The variable representing the transmission of RTBV from RTBV singly infected (Y) was in the probability form in HC1996 ( $t_2 = 0.08$ ). In BD2017, the equivalent was sigma (sigma = 0.08), but transmission terms in their ODEs were divided by the total current population. Since our model did not considered vector dynamics, represented each compartment in the number, not proportion, (Fig. 1) and used the rates, not probability, to factor with each term in ODEs, we then need to change the absolute values 0.08 that they used in the models by dividing 0.08 by  $f(\mathbf{K})$ , where  $\mathbf{K}$  is the maximum number of hosts when the birth rate is zero  $(\mathbf{r}(\mathbf{K} - \mathbf{P}) = \mathbf{0})$ .

To obtain a reasonably large population at steady state,  $f(\mathbf{K})$  was determined based on the steady state value of the susceptible hosts  $(\frac{dX}{dt} = \mathbf{0})$ , when there is no disease  $(\mathbf{Y} = \mathbf{U} = \mathbf{Z} = \mathbf{0})$ , so  $\mathbf{P} = \mathbf{X} + \mathbf{Y} + \mathbf{Z} + \mathbf{U} = \mathbf{X})$ , which can be obtained as below:

$$\frac{dX}{dt} = r(K - P) - p_1(Y + s_1 U)X - p_2(Z + s_2 U)X - p_5 UX - qX = 0$$

$$\frac{dX}{dt} = r(K - X) - qX = 0$$

$$rK - rX - qX = 0$$

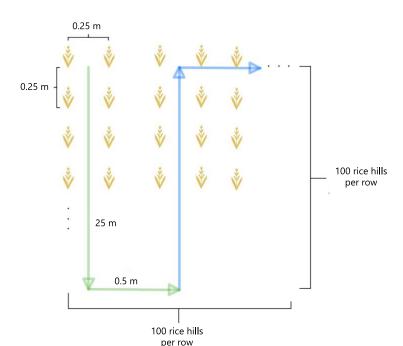
$$X = \frac{rK}{r + q}$$

This means that the disease-free steady state number of X depends on the values of  $\mathbf{r}$ ,  $\mathbf{K}$  and  $\mathbf{q}$ , and it equals to  $\mathbf{K}/9$  as we set  $\mathbf{r}=0.001$  and  $\mathbf{q}=0.008$  (Sup. Table 1); therefore,  $\mathbf{f}(\mathbf{K})$  should be equal to  $\mathbf{K}/9$ . With this decision, the size of  $\mathbf{p}_1$  was only reduced by the size of small long-term population (carrying capacity when birth and death rate are not zero). If we were to use  $\mathbf{K}$  as a

denominator, we would obtain too low incidence of RTD due to excessively reduced transmission rates, which is what can be seen from BD2017. This would not be suitable for further investigation of roguing effect.

#### **Appendix 3.1: Calculation of roguing distance for our model**

One rice hill (one plant in our investigation) is usually allowed to occupy 25x25 cm<sup>2</sup> area of land (the space between the hill is 25 cm in all directions) [6], and therefore, the maximum host capacity (*K*) of 10,000 host plants used in our model represents a square rice paddy with 2500x2500 cm<sup>2</sup> or 25x25 m<sup>2</sup> area (or 625 m<sup>2</sup>). For roguing operation, assuming walking along one line in the middle between two rows of rice allows the inspection of both rows, the staff only need to walk a 25.5-m distance to survey 2 rows, which amount to 50 times overall for 100 rows (Figure A). Therefore, they need to walk which equal to 1275 m or 1.28 km. Therefore, little human resource is needed, especially considering the fact that the host abundance was usually much lower than 10,000 in our model; a large portion of the paddy will not need the survey. However, a larger labour requirement will be applied for a larger-scale rice paddy.



path was drawn for the roguing survey of the first 2 rows of rice hills (100 hills per row, so each row is 0.25x100 = 25 m long). The blue path represented the survey distance of the second 2 rows. To complete the survey of a paddy with 100x100 (10,000) rice hills is, therefore, 25.5m x 50 = 1275 m.

#### Appendix 3.2: Infection age-dependent yield losses

Yield losses due to the infection of either or both RTBV and RTSV decreases as rice hosts get infected later after planting. Generally, the number of tillers (shoots that arise from the base of a grass plant) determined the yield, and it is negatively affected if rices reduce nutrients [7], which can be due to poor root development, induced by the infection of one of these viruses (with the most severe being due to the co-infection). Infected at the early stage from germination to tillering stage (vegetative phase), which lasts 45-55 days for Taichuang 1 (TN1) that are short-duration variety [7], the disease then can reduce the yield much more than infection in the later stage (from panicle initiation and to flowering phase).

The percentage yield loss of TN1 (Taichuang 1) varieties due to RTBV, RTSV, or coinfection [8] and calculated yield coefficients is shown in the **Table A** below:

Infection time (days	Yield loss				
after planting)	RTBV (Y)	RTSV (Z)	Both (U)		
7	83	27	95		
21	70	22	94		
35	35	20	90		
49	28	20	75		
Average yield loss	54	22	88		
Average yield	46	78	12		
Yield coefficient	$c_1 = 0.46$	<b>c</b> <sub>2</sub> = 0.78	<b>c</b> <sub>3</sub> = 0.12		

Note that since the yield reduction of RTBV/RTSV co-infected plants was reduced to only 5% if plants get infected at 60 days after planting [], we then disregard yield losses after 49 days, at which information can be found for all types of infection.

# Appendix 4: The strength of the influence of the change in transmision rate of each virus due to a source having the other virus on the U135 proportion.

 $\mathbf{s_1}$  is change in transmission rate of RTBV due to a source previously being infected by RTSV) and  $\mathbf{s_2}$  is change in transmission rate of RTSV due to a source previously being infected by RTBV

 $\mathbf{p_1}$  is the transmission rate of RTBV from the RTBV-singly infected (Y) and  $\mathbf{p_2}$  is the transmission rate of RTSV from the RTSV-singly infected (Z), set at 0.08/(K/9) and 0.125/(K/9), respectively in the normal two-way  $\mathbf{s_1}$ - $\mathbf{s_2}$  scan (Fig. 7). The scan outcome with the swapped values of  $\mathbf{p_1}$  and  $\mathbf{p_2}$  switched the pattern seen in the normal scan (i.e. transpose the color matrix) (Fig. H). In contrast, when the death rates of Y and Z were swapped, there were no change in the colour pattern (not shown), further supporting that the depend on the relative values of  $\mathbf{p_1}$  and  $\mathbf{p_2}$  of the relative influence strength of  $\mathbf{s_1}$  and  $\mathbf{s_2}$ 

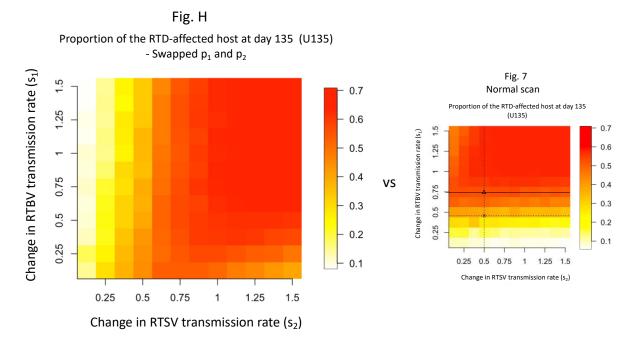


Figure H: two-way  $s_1$ - $s_2$  scan outcome using the transmission rate of RTBV ( $p_1$ ) and the transmission rate of RTSV ( $p_2$ ) equal to 0.125/(K/9) and 0.08/(K/9), respectively. The horizontal gradients were steeper than the vertical ones, reflecting the greater influence of  $s_1$  on U135 proportion compared to that of  $s_2$ , giving the opposite conclusion to the normal scan (Fig. 7)

**Appendix 5: Default values of transmission-related parameters to be used for roguing investigation.** Shown in the Table B below were the values that gave high incidence of RTD at day 135 and were in the ranges cited by HC1996 and BD2017 paper. They were used with fixed parameters (Sup. table 3.1) for roguing investigation.

Virus transmission event	Transition	Source	Parameter	Default values
+RTBV	X to Y or Z to U	Y	$p_1$	0.080/( <b>K</b> /9)
+RTSV	X to Z or Y to U	Z	$p_2$	0.125/( <b>K</b> /9)
+RTBV and RTSV	X to U	U	$p_{5}$	0.086/( <b>K</b> /9)
Change in				
Change in the transmission rat	$s_1$	0.750		
Change in the transmission rat	pared to from Z	$s_2$	0.500	

Similar to other parameter sets, the RTD dynamics with this parameter set was only subject to demographic stochasticity when either Y0 or Z0, or both were 1 (Figure B-D). Therefore, we kept the initial condition X0=1000, Y0=10=Z0, and U0=1 as the initial condition for further roquing investigation (Fig. 9).

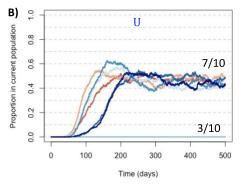
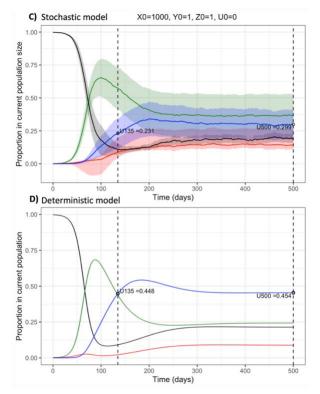


Figure B-D: Simulations using the default set of parameters for roguing and the initial condition Set 1 (X0=1000, Y0=1=Z0, and U0=0). B) Separate stochastic runs, showing 7/10 with the non-zero steady-state RTD-affected host (U). C) The mean trajectories of all 10 independent stochastic runs. A larger SD can be seen with these initial conditions compared Fig. 9. D) From the deterministic model.



U135 and U500 were higher than from stochastic runs due to the non-existence of the case with the extinction of U.

**Appendix 6:** Progression curve from stochastic simulations using the "extreme" set of parameters (Sup. Table 3.4) but varied initial conditions.

Since X0 (the initial susceptible) and U0 (the initial co-infected) has no effects on stochasticity, in this investigation, they were kept at 1000 and 0, respectively, while Y0 (the initial RTBV-infected) and Z0 (the initial RTSV-infected) were varied to search for the minimum number of initial Y and Z that allow the resistance to demographic stochasticity. The result showed that if either Y0 or Z0 was more than 1 (Fig. G), all 10 stochastic runs followed the same trajectories. If one of them started off with 1 (Fig. E, F), the trajectories diverged, and some extinction occurred. As expected, if Z0>Y0, the extinction, if ever occurred, happened with Y (Fig. E) and Z persisted, and vice versa (Fig. F).

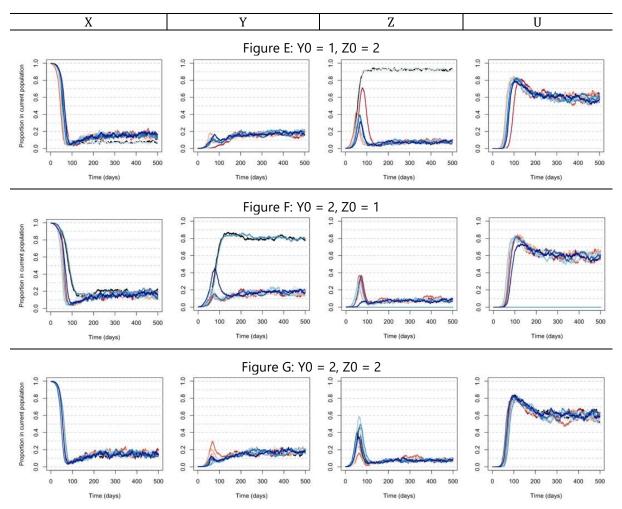
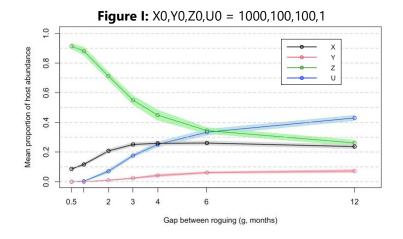


Figure E-G: the separate trajectories form 10 independent stochastic runs using  $\mathbf{p_1}$ ,  $\mathbf{p_2}$ ,  $\mathbf{p_5}$ ,  $\mathbf{s_1}$ ,  $\mathbf{s_2} = 0.09/(K/9)$ , 0.13/(K/9), 0.16/(K/9), 1.7, 0.7, respectively. X0=1000 and U0=0 in all cases, Y0 and zz0 varied as in the title. **Appendix 7:** Lengthening the inter-rogue gaps initially increase the proportion of susceptible host (X) at day 135 (harvesting day), regardless of the initial conditions. The

increase in X also does not result from the disproportionate decrease in X compared to the decrease in overall population. The abundance of X at day 135 were genuinely increased.



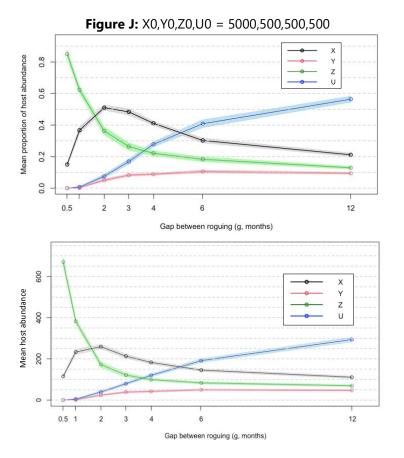


Figure I: The mean proportion of each host compartment at day 135. The initial conditions shown at the top of the graph. X135 proportion increases when the gap was increased from 0.5 to 3 months, similar to the initial condition that had much lower Y0 and Z0 (10 each) used in the main investigation. The increase was independent of Y0 and Z0.

Figure J: The initial conditions shown at the top of the two graphs. The graph above shows the mean proportion of each host compartment at day 135. X135 proportion increased when the gap was increased from 0.5 to 2 months, similar to the initial condition that had much lower YO and Z0 (10 each) used in the main investigation. The increase was also independent of X0 and U0. The graph below shows the mean number of each host compartment at day 135. X135 number increased when the gap was increased from 0.5 to 2 month, similar to its mean proportion.