Online Appendix

O.1. TIP Model Extension with Overnight Stays

To formally incorporate poultry staying overnight in the market, the model equations are adjusted to account for additional compartments and transitions. First, the study introduces the new input parameter $\phi \in [0,1]$, which denotes the fraction of poultry that remains in the market overnight, and the death rate d, which captures the fraction of infected poultry that do not survive to the next day.

The total number of infected and susceptible poultry present in the market at the beginning of each period t is now given by:

$$TI_t^{begin} = I_t^{in} + I_t^{market}, \tag{O.1}$$

$$TS_t^{begin} = S_t^{in} + S_t^{market}. \tag{O.2}$$

The infection mechanism then adjusts accordingly:

$$TI_t^{end} = \min\{(\beta \frac{TI_t^{begin}}{TI_t^{begin} + TS_t^{begin}} + E_t) \cdot TS_t^{begin} + TI_t^{begin}, TS_t^{begin} + TI_t^{begin}\}. \tag{O.3}$$

The outflows of infected and susceptible poultry now account for the fraction of poultry that remains in the market:

$$I_t^{out} = (1-\phi) TI_t^{end}, \tag{O.4}$$

$$S_t^{out} = (1-\phi) TS_t^{end}. \tag{O.5}$$

The outflow infection prevalence is then given by:

$$R_t^{out} = \frac{I_t^{out}}{I_t^{out} + S_t^{out}} = \frac{TI_t^{end}}{TI_t^{end} + TS_t^{end}}. \tag{O.6}$$

Meanwhile, the environmental risk continues to evolve as:

$$E_{t+1} = \min\{\gamma E_t + \alpha R_t^{out}, 1\}. \tag{O.7}$$

Finally, the fraction ϕ of poultry that stays overnight is updated as follows:

$$I_{t+1}^{market} = \phi(1-d) TI_t^{end}, \tag{O.8}$$

$$S_{t+1}^{market} = \phi TS_t^{end}. \tag{O.9}$$

This extended version of the TIP model can also incorporate stochastic variations in epidemiological parameters by considering the stochastic transmission rate $\hat{\beta}_t$, interaction rate $\hat{\alpha}_t$, and persistence rate $\hat{\gamma}_t$. Similarly, the fraction ϕ and death rate d can be subject to short-term stochastic perturbations, following the methodology outlined in §3.3.

O.2. Detailed Proofs

O.2.1. Theorem 1

The proof of Theorem 1 relies on several preliminary lemmas. These are outlined next with a discussion regarding their interpretation.

First, Equation (8) indicates that the outflow infection prevalence time series, which is the main outcome of interest, directly depends on the environmental risk time series. Thus, Lemma O.1 below characterizes the temporal evolution of the environmental risk time series.

Lemma O.1. *The environmental risk time series, E_t, follows the equation:*

$$E_{t+1} = \min\{(\gamma + \alpha(1-r)) \cdot E_t + \alpha r(1+\beta(1-r)), \gamma E_t + \alpha, 1\}. \tag{O.10}$$

Lemma O.1 indicates that the environmental risk E_t follows a piecewise first order recursion (Ouaknine and Worrell 2012), until it reaches the value 1, where all poultry exiting the market are infected, which will be helpful to further analyzing its temporal evolution. Next, Lemma O.2 provides a threshold condition on the environmental risk time series that is equivalent to the outflow infection prevalence time series, reaching the *'saturation'* level 1 and implying that all the poultry that exit the market are infected until the next market sanitation.

Proof of Lemma O.1: Substituting Equation (8) into Equation (9), which describes the update rule of the environmental risk over time, yields to:

$$E_{t+1} = \min\{\gamma E_t + \alpha \min\{(\beta r + E_t)(1-r) + r, 1\}, 1\}, \tag{O.11}$$

Which is equivalent to:

$$E_{t+1} = \min\{\gamma E_t + \alpha((\beta r + E_t)(1-r) + r), \gamma E_t + \alpha, 1\}. \tag{O.12}$$

Which leads to the result in Lemma O.1 after some algebraic manipulations □

Lemma O.2. *For each time period t, $E_t \geq 1 - \beta r$ if and only if $R_t^{out} = 1$.*

Proof of Lemma O.2 : The proof relies on Equation (8) and the following readily verified sequence of equivalence:

$$E_t \geq 1 - \beta r \iff E_t + \beta r \geq 1 \iff (E_t + \beta r)(1-r) + r \geq 1 \iff R_t^{out} = 1. \tag{O.13}$$

□

Lemma O.2 highlights the critical role of the threshold value $1 - \beta r$ in shaping the relationship between environmental risk and the outflow infection prevalence time series, which serves as the primary metric of interest. This threshold signifies that the market environment facilitates infection transmission

to all poultry that were not directly infected by incoming sick poultry. Furthermore, Lemmas O.3 and O.4 characterize the temporal evolution of the environmental risk time series when operating below this threshold.

Lemma O.3. *For each period t in the regime below* $1-\beta r$*, the environmental risk time series* E_t *could be expressed as:*

$$E_t = A(\gamma,\alpha,r)^t(E^{ref} - L(\beta,\gamma,\alpha,r)) + L(\beta,\gamma,\alpha,r) \tag{O.14}$$

With $A(\gamma,\alpha,r) = \gamma + \alpha(1-r)$ *and* $L(\beta,\gamma,\alpha,r) = \frac{r\alpha(1+\beta(1-r))}{1-\gamma-\alpha(1-r)}$.

Proof of Lemma O.3: As long as the environmental risk time series, E_t, is below $1-\beta r$, the expression in Equation (O.10) could be simplified as:

$$E_{t+1} = (\gamma + \alpha(1-r)) \cdot E_t + \alpha r(1+\beta(1-r)). \tag{O.15}$$

This expression is equivalent to a first order recurrent sequence (Andrica and Bagdasar 2020) in the form of $E_{t+1} = A \cdot E_t + B$, with $A = \gamma + \alpha(1-r)$ and $B = \alpha r(1+\beta(1-r))$. The set of parameters γ and α in the space of real numbers that could lead to $\gamma + \alpha(1-r) = 1$ is of probability measure 0 in $\mathbb{R}$ (Kingman and Taylor 2008, Halmos 2013), which implies that $A \neq 1$ almost surely in reality. Thus, the solution of such process could be expressed as $E_t = A^t(E^{ref} - L) + L$ where $L = \frac{B}{1-A}$. Consequently, as long as the environmental risk time series is below $1-\beta r$, it could be expressed in each period t as:

$$E_t = (\gamma + \alpha(1-r))^t(E^{ref} - \frac{r\alpha(1+\beta(1-r))}{1-\gamma-\alpha(1-r)}) + \frac{r\alpha(1+\beta(1-r))}{1-\gamma-\alpha(1-r)}. \tag{O.16}$$

To simplify the exposition, let $A(\gamma,\alpha,r) = \gamma + \alpha(1-r)$ and $L(\beta,\gamma,\alpha,r) = \frac{r\alpha(1+\beta(1-r))}{1-\gamma-\alpha(1-r)}$. It follows that:

$$E_t = A(\gamma,\alpha,r)^t(E^{ref} - L(\beta,\gamma,\alpha,r)) + L(\beta,\gamma,\alpha,r). \tag{O.17}$$

□

Lemma O.3 indicates that the environmental risk time series can be expressed as a geometric sequence with an affine shift, as long as it stays in the regime below $1-\beta r$. This property will become helpful to further characterize the temporal patterns of its trajectory as a function of input parameters. Lemma O.4, below, defines under which conditions on the input parameters, the environmental risk can exceed the $1-\beta r$ threshold.

Lemma O.4. *Starting from a reference value,* $0 \leq E^{ref} \ll 1-\beta r$*, the environmental risk time series* E_t *can exceed the threshold* $1-\beta r$ *only if* $A(\gamma,\alpha,r) > 1$ *or* $L(\beta,\gamma,\alpha,r) \geq 1-\beta r$.

Proof Lemma O.4: First, similar to the proof of Lemma O.3, it is valid to assume that $A(\gamma,\alpha,r)$ is almost surely not equal to 1. Additionally, by definition of $A(\gamma,\alpha,r)$ and $L(\beta,\gamma,\alpha,r)$, it follows that

$A(\gamma,\alpha,r)>1$ is equivalent to $L(\beta,\gamma,\alpha,r)\leq 0$. Subsequently, three possible cases are possible depending on the values of $A(\gamma,\alpha,r)$ and $L(\beta,\gamma,\alpha,r)$. Consider the following three mutually exclusive cases, including the first case when $A(\gamma,\alpha,r)>1$, the second case is when $A(\gamma,\alpha,r)\leq 1$ and $L(\beta,\gamma,\alpha,r)\geq 1-\beta r$, and the third case is when $A(\gamma,\alpha,r)\leq 1$ and $L(\beta,\gamma,\alpha,r)<1-\beta r$.

Now when $A(\gamma,\alpha,r)>1$, it follows that $L(\beta,\gamma,\alpha,r)\leq 0$, which results in that $(E^{ref}-L(\beta,\gamma,\alpha,r))\geq 0$. Thus, the component $A(\gamma,\alpha,r)^t(E^{ref}-L(\beta,\gamma,\alpha,r))$ risk increases exponentially, as the growth multiplier $A(\gamma,\alpha,r)$ is higher than 1, and could exceed $1-\beta r$.

Similarly, if $A(\gamma,\alpha,r)\leq 1$, it follows that E_t approaches the value $L(\beta,\gamma,\alpha,r)\geq 1-\beta r$, which implies that the environmental risk time series ultimately exceeds the threshold value $1-\beta r$.

In the third case when $A(\gamma,\alpha,r)\leq 1$ and $L(\beta,\gamma,\alpha,r)\leq 1-\beta r$, the environmental risk time series approaches the value $L(\beta,\gamma,\alpha,r)$ and remains below the threshold (see more details about this case in the proof below of Theorem 1). □

Notably, the condition $A(\gamma,\alpha,r)>1$ implies that the environmental risk time series is in an exponential growth state. Additionally, $A(\gamma,\alpha,r)$ involves epidemiologic parameters γ and α that are related to environmental infections, which underscores the role of the market environment in shaping infection spread. Lemma O.5 complements Lemma O.3, by characterizing the evolution pattern of the environmental risk time series once it exceeds the threshold $1-\beta r$.

Lemma O.5. *If, for some time period t, $E_t\geq 1-\beta r$, then in the following period t+1, $E_{t+1}\geq 1-\beta r$, i.e., for all $t'\geq t$, $E_{t'}\geq 1-\beta r$.*

Proof of Lemma O.5: First, it should be noted that $E_t\geq 1-\beta r$ implies that:

$$E_{t+1}=\min\{(\gamma+\alpha(1-r))\cdot E_t+\alpha r(1+\beta(1-r)),\gamma E_t+\alpha,1\}\geq\gamma(1-\beta r)+\alpha\geq(\gamma+\alpha)(1-\beta r). \quad \text{(O.18)}$$

In this setting, the first case above when $A(\gamma,\alpha,r)>1$ is equivalent to $\gamma+\alpha(1-r)>1$ which implies $(\gamma+\alpha)\geq 1$ since $0\leq r\ll 1$. This implies that:

$$E_{t+1}\geq(\gamma+\alpha)(1-\beta r)\geq(1-\beta r). \quad \text{(O.19)}$$

Similarly, considering the second case when $A(\gamma,\alpha,r)\leq 1$ and $L(\beta,\gamma,\alpha,r)\geq 1-\beta r$, it follows after some algebraic manipulation that:

$$L(\beta,\gamma,\alpha,r)\geq 1-\beta r \iff \gamma(1-\beta r)+\alpha\geq 1-\beta r. \quad \text{(O.20)}$$

Thus, Inequality (O.18) implies that E_{t+1} will also be higher than the threshold value $1-\beta r$. Subsequently, all the next environmental risk time series will remain above this threshold until sanitation. □

Lemmas O.2 indicates that the environmental risk time series exceeds the threshold $1-\beta r$, if and only if the outflow infection prevalence time series reaches the value 1, where all poultry exiting the market are infected. Lemma O.4 provides necessary conditions on the input parameters for the environmental risk time series to exceed the threshold $1-\beta r$, under a specific condition on $A(\gamma,\alpha,r)$. Lemma O.5 shows that this threshold is a saturation point in that once exceeded the threshold, the environmental risk time series will stay above it until the next sanitation, and so will the outflow infection prevalence time series at the value 1. The proof of the theorem leverages Lemmas O.1-O.5, and focuses on the temporal evolution of the environmental risk time series in the regime below the threshold $1-\beta r$.

Proof of Theorem 1: The proof builds on the result of Lemma O.4 to characterize the temporal evolution of the environmental risk time series as a function of $A(\gamma,\alpha,r)$ and $L(\beta,\gamma,\alpha,r)$ in the regime below the threshold $1-\beta r$. Equation (8) implies that this directly determines the temporal dynamics of the outflow infection prevalence time series. The analysis follows by analyzing different cases:

Case 1 when $A(\gamma,\alpha,r)>1$: As long as $E_t \le 1-\beta r$, the environmental risk time series would follow Equation (O.14). Given that the growth multiplier $A(\gamma,\alpha,r)$ is higher than 1, the environmental risk time series evolves exponentially over time. If it reaches $1-\beta r$ before the end of the sanitation cycle, then the environmental risk above this threshold, which implies that the outflow infection prevalence is at the saturation point, where all the poultry that exit the market are infected.

Case 2 when $A(\gamma,\alpha,r)<1$ and $L(\beta,\gamma,\alpha,r)\ge 1-\beta r$: Under this case, it follows from Lemma O.4 that the environmental risk time series increases toward $L(\beta,\gamma,\alpha,r)$ until it potentially reaches the threshold of $1-\beta r$. Notably, the conditions of this case imply that, although $A(\gamma,\alpha,r)$ is lower 1, but still close to it. That is, the environmental risk time series grow at approximately linear rate. Indeed:

$$L(\beta,\gamma,\alpha,r)\ge 1-\beta r \iff \frac{r\alpha(1+\beta(1-r))}{1-\gamma-\alpha(1-r)} \ge 1-\beta r \tag{O.21}$$

$$\iff \gamma+\alpha(1-r)\ge 1-\frac{r\alpha(1+\beta(1-r))}{1-\beta r} \tag{O.22}$$

$$\iff A(\gamma,\alpha,r)\ge 1-\frac{r\alpha(1+\beta(1-r))}{1-\beta r}. \tag{O.23}$$

Based on the assumption that $r\ll 1$, this implies that the growth multiplier $A(\gamma,\alpha,r)$ is close to 1, which implies that the Taylor expansion of $A(\gamma,\alpha,r)^t$ (Pourahmadi 1984) will be a good approximation. Hence, as long as $(1-\gamma-(1-r)\alpha)\cdot t \ll 1$, the expression has the following approximate linear evolution:

$$A(\gamma,\alpha,r)^t=(\gamma+(1-r)\alpha)^t \sim 1-t(1-\gamma-(1-r)\alpha). \tag{O.24}$$

As long as the environmental risk time series is below $1-\beta r$, this could then be used to approximate Equation (O.14). It follows that the temporal evolution of the environmental risk time series is approximately captured by:

$$E_t \sim E^{ref} + t(1-\gamma-(1-r)\alpha)(L(\beta,\gamma,\alpha,r)-E^{ref}). \tag{O.25}$$

Thus, the resulting approximate growth rate is:

$$(1-\gamma-(1-r)\alpha)(L(\beta,\gamma,\alpha,r)-E^{ref}). \tag{O.26}$$

Meanwhile, Equation (O.23) is equivalent to:

$$1-\gamma-\alpha(1-r) \leq \frac{r\alpha(1+\beta(1-r))}{1-\beta r}. \tag{O.27}$$

Which ultimately implies the following inequation after multiplying by $(L(\beta,\gamma,\alpha,r)-E^{ref}) \geq 0$ on both sides:

$$(1-\gamma-(1-r)\alpha)(L(\beta,\gamma,\alpha,r)-E^{ref}) \leq \frac{r\alpha(1+\beta(1-r))}{1-\beta r}(L(\beta,\gamma,\alpha,r)-E^{ref}). \tag{O.28}$$

The assumption that the inflow infection prevalence r is significantly smaller than 1 implies that the right-hand side of Equation (O.28) would also be significantly lower than 1, so as would be the growth rate expressed in the left-hand side. Hence, the environmental risk time series has an approximate linear growth that is significantly lower than 1, and would then slowly increase until it reaches $1-\beta r$. Once it exceeds that value, it will remain higher than the threshold and the outflow infection prevalence will remain at saturation level 1. Note that is the analysis is still technically valid in the edge case $A(\gamma,\alpha,r)=1$, however this happens with an probability 0 in practice.

Case 3 when $A(\gamma,\alpha,r)<1$ **and** $L(\beta,\gamma,\alpha,r)<1-\beta r$: Under this case, the environmental risk time series will always remain below the threshold level $1-\beta r$ and therefore the outflow infection prevalence time series will never reach the saturation level 1. More specifically, there would be two different sub-cases depending on whether $L(\beta,\gamma,\alpha,r)$ is higher or lower than E^{ref}. When $L(\beta,\gamma,\alpha,r) \leq E^{ref}$, then it follows from Equation (O.10) that the environmental risk time series decreases towards the limit $L(\beta,\gamma,\alpha,r)$ which is negligibly small below E^{ref}. This implies that the market infectivity level is maintained at a relatively safe level as it would remain negligibly small.

On the other hand, when $L(\beta,\gamma,\alpha,r) \geq E^{ref}$, the environmental risk time series increases towards the limit $L(\beta,\gamma,\alpha,r)$. Now, note the following relationship between $A(\gamma,\alpha,r)$ and $L(\beta,\gamma,\alpha,r)$:

$$A(\gamma,\alpha,r) = 1 - \frac{r\alpha(1+\beta(1-r))}{L(\beta,\gamma,\alpha,r)}. \tag{O.29}$$

From Equation (8) it follows that Case 1 above corresponds to a state where the outflow infection prevalence time series grows exponentially over time, and Case 2 above corresponds to a state where it will have a low and approximately linear growth. That is, if the sanitation period T is sufficiently long, under Cases 1 and 2, the outflow infection prevalence time series will reach the saturation level 1. However, in the Case 1, this will occur significantly faster. In contrast, Case 3 above corresponds to a stable state in the sense that the outflow infection prevalence time series either evolves toward a relatively low and safe value or will have a relatively small growth over time but will never arrive at the saturation level. □

Proof of Lemma O.6: To derive these results, Equation (O.10) should be considered in the stochastic setting described in §3.3. Let $0 \leq t \leq T-1$ be a time within a sanitation cycle of length T, and let s indicate the season during which the cycle is happening. The evolution of the model's epidemiological parameters during the sanitation cycle follow Equations (12), (13), and (14) of the the stochastic TIP model.

Similarly to Lemma O.3, as long as for any time t, $0 \leq \hat{E}_t \leq 1-\hat{\beta}_t r$, the environmental risk update would follow the dynamic:

$$\hat{E}_{t+1} = (\hat{\gamma}_t + \hat{\alpha}_t(1-r)) \cdot \hat{E}_t + \hat{\alpha}_t r(1+\hat{\beta}_t(1-r)). \tag{O.30}$$

The analogous expression of the outflow infection prevalence growth multiplier in the stochastic setting would then be $A(\hat{\gamma}_t, \hat{\alpha}_t, r)$, defined as:

$$A(\hat{\gamma}_t, \hat{\alpha}_t, r) = \hat{\gamma}_t + \hat{\alpha}_t(1-r) = \gamma + (1-r)\alpha + \sigma_\gamma W_t^\gamma + (1-r)\sigma_\alpha W_t^\alpha. \tag{O.31}$$

Hence, the compounded effect of the stochastic perturbations to the growth rate is $\sigma_\gamma W_t^\gamma + (1-r)\sigma_\alpha W_t^\alpha$. By independence of the Brownian processes W_t^γ and W_t^α and their linearity (Durrett 2019), this compounded effect can be equivalently described as a Brownian process:

$$\sigma_\gamma W_t^\gamma + (1-r)\sigma_\alpha W_t^\alpha = (\sigma_\gamma + (1-r)\sigma_\alpha) W_t. \tag{O.32}$$

where W_t is also a Brownian perturbation that follows a normal distribution $\mathcal{N}(0,t)$. □

O.2.2. Theorem 2

Theorem 2 defines the probability to transition into the exponential growth rate. It leverages, Lemma O.6 which characterizes the compound effect of the short-term perturbations (See Appendix O.2 for detailed proof).

Lemma O.6. *The compound effect of short-term Brownian perturbations on the TIP model's epidemiological parameters results in a Brownian perturbation of the environmental risk growth rate.*

Hence, using Lemma O.3, the probability to transition into the exponential growth rate is equal to the probability that the growth multiplier, $A(\hat{\gamma}_t, \hat{\alpha}_t, r)$, defined in §4.1, becomes higher than 1 during a sanitation cycle T.

Proof of Theorem 2: Starting from a stable or approximate linear growth state where $A(\hat{\gamma}_t, \hat{\alpha}_t, r) \leq 1$, the probability to transition to a state when $A(\hat{\gamma}_t, \hat{\alpha}_t, r) > 1$ depends on the compounded perturbations:

$$A(\hat{\gamma}_t, \hat{\alpha}_t, r) > 1 \iff \gamma + (1-r)\alpha + (\sigma_\gamma + (1-r)\sigma_\alpha)W_t > 1 \tag{O.33}$$

$$\iff W_t > \frac{1-\gamma-(1-r)\alpha}{\sigma_\gamma + (1-r)\sigma_\alpha}. \tag{O.34}$$

Define the threshold on the right-hand side of Equation (O.34), $g = \frac{1-\gamma-(1-r)\alpha}{\sigma_\gamma+(1-r)\sigma_\alpha}$, to be the gap that the compound perturbations need to reach for $A(\hat{\gamma}_t, \hat{\alpha}_t, r)$ to exceed 1 during the sanitation cycle T. This probability of an outbreak then corresponds to the probability that the compound perturbations exceed this threshold before the end of the sanitation cycle in T. This is equivalent to the probability that the first passage time of W_t above the threshold g occurs prior to T (Redner 2001). Formally, let τ_g be the first passage time of the perturbation W_t above the gap g, $\tau_g = \inf\{t \geq 0, W_t \geq g\}$. Hence, the probability of an outbreak is equal to $P(\tau_g \leq T)$. Note that this does not necessarily imply that the market remains in this state afterwards as the process could go back to a stable or approximate linear growth state. However, by symmetry of the Brownian evolution, once the process exceeds the gap, the probability that it stays at the exponential growth state after that is $\frac{1}{2}$. To further characterize this risk probability, the reflection principle of a Brownian motion (Jacobs 2010) is used in Equation (O.36) below to obtain the Theorem's expression of the risk of an outbreak:

$$P(\tau_g \leq T) = P(\max_{0 \leq t \leq T} W_T > g) \tag{O.35}$$

$$= 2P(W_T > g). \tag{O.36}$$

Where W_T follows a normal distribution $\mathcal{N}(0, T)$. □

O.3. Calibration of the TIP Model Parameters

This section describes the calibration procedure of the TIP model parameters with a comprehensive literature search on avian influenza epidemiological data. It provides estimates for the transmission rate, interaction rate, and persistence rate parameters from the literature. These estimates offer direct realistic numerical values or serve as reliable proxies for each parameter.

Transmission rate parameter. The transmission rate parameter β has been measured for the case of H5N1 in previous research. Experiments in (Spekreijse et al. 2011) estimate the virus transmission

to be 1.43 per day for poultry, meaning that each infected poultry transmits the disease to 1.43 other susceptible ones on average during one day. Similarly, results in (Bouma et al. 2009) suggest an estimate of 0.80 per day. Yet, these estimates significantly depend on the number of contacts, how close infectious and susceptible poultry are, among many other experimental settings. Thus, given the reported values, the study uses a transmission rate parameter β between 0.8 and 1.5 for its simulations.

Interaction rate parameter. The interaction rate parameter α is more challenging to estimate. No prior research has defined this parameter nor measured to what extent an environment becomes infectious due to viral shedding from infected poultry. However, the field study (Indriani et al. 2010) conducted tests for H5N1 in multiple environmental sites in markets where the disease is endemic, i.e., poultry are regularly infected. It found that 19% to 24% of the samples from environmental sites like poultry-processing tables, waste disposal areas, and cages tested positive for the virus. Similarly, the field study (Wang et al. 2017a) tested market environments where avian influenza disease is endemic and found that 39.7% of the environmental samples were contaminated. Hence, assuming such contaminated sites could potentially also infect susceptible poultry, these figures could serve as proxies for the virus's interaction rate with the environment. For low pathogenic disease strains, reduced interaction rates could be considered to reflect relatively lower levels of virus shedding and infectivity. Consequently, the study adopts an interaction rate parameter α ranging from 0.1 to 0.5 for its simulations.

Persistence rate parameter. More research has been done on the persistence of avian influenza in environmental sites such as animal pathogens, wastewater, or surfaces. Results in (Shortridge et al. 1998) show that H5N1 virus infectivity is maintained for at least 4 days in wet feces. Similar experiments in (Wood et al. 2010) show that avian influenza H5N1 virus can remain detectable on poultry feces, galvanized metal, or soil from 3 to 13 days depending on room temperature and humidity. Hence, given a percentage $P(detect)$ of remaining pathogens that enables the virus to be detected after n number of days (for e.g., 5% after 3 days), the persistence rate can be estimated as the following:

$$\rho^n \leq P(detect) \iff \rho \leq P(detect)^{\frac{1}{n}} \tag{O.37}$$

This provides an estimate of a persistence rate parameter ρ from 0.3 to 0.8.

Moreover, for avian influenza viruses, poultry deaths can occur as early as 24–48 hours after infection (Takadate et al. 2023). This supports the study's approach that considers deaths only for the model where a fraction of poultry remain in the market overnight. The death rate one day d varies significantly depending on the strain's virulence, ranging from as low as 0.01 for low pathogenic strains (Spickler et al. 2008) to as high as 0.6 for highly pathogenic strains, particularly among unvaccinated poultry

(D.E. Swayne & D.L. Suarez 2000, Saczyńska et al. 2017). Based on these estimates, the study assumes a death rate of $d = 0.1$ when simulating the TIP model with overnight stay.

Furthermore, a Brownian perturbation with mean 0, a variance of 1, and a weight of 0.01 has been applied to each parameter to account for the environmental stochasticity affecting their baseline values while there is no market sanitation event. Similarly, the study accounts for seasonal effects on the persistence parameter with variations up to plus or minus 0.1 around its base value (See Appendix O.9 for more details on the calibration of seasonal effects). The weights of these perturbations and seasonal variations can be adjusted depending on the contextual characteristics of each market, such as variability of weather conditions, stringency of sanitation practices and market layout. Moreover, the study uses a Poisson distribution to simulate a stochastic inflow infection prevalence, with an average of 0.01 to account for relatively safe sourcing farms (see Appendix O.5 and O.7 for a comprehensive sensitivity analysis of the results with respect to each of the model parameters).

O.4. Analysis of Sanitation Stringency

The following analysis examines the importance of having stringent sanitation, i.e., a very low post-sanitation E^{ref}, with respect to sanitation frequency to control infection spread at the market level. Specifically, the study considers situations when sanitation could be imperfect, where the post-sanitation environmental risk can be E^{ref} significant as compared to the inflow infection prevalence r. To account for that, Equation (10) and (11), describing the evolution of the environmental risk, are adjusted as follows:

$$E_{t+1} = min\{\gamma E_t + \alpha R_t^{out}, 1\} \text{ if } t+1 \neq 0 \pmod{T}, \tag{O.38}$$

$$E_{t+1} = min\{E^{ref}, E_{t+1}\} \text{ if } t+1 = 0 \pmod{T}. \tag{O.39}$$

The study simulates the evolution of outflow infection prevalence for a highly pathogenic strain ($\beta = 1$, $\alpha = 0.4$, $\gamma = 0.8$) while considering different sanitation stringency levels, $E^{ref} = 0.01$ and $E^{ref} = 0.1$, and sanitation period $T = 7$ days and $T = 21$ days, using the stochastic TIP model. Figure O.1 presents the results over a period of 65 days.

For $T = 7$ days (top panel), frequent and stringent sanitation coupled with low environmental risk $E^{ref} = 0.01$ mitigates infection accumulation over time and maintains consistently low infection levels. However, imperfect sanitation, with a higher $E^{ref} = 0.1$ leads to periodic surges in infection prevalence between sanitation events. Thus, stringent sanitation is a necessary condition to effectively prevent infection spread although sanitation are conducted regularly.

For $T = 21$ days (bottom panel), longer sanitation intervals allow infection levels to rise significantly until all animals exiting the market get infected, for both levels of sanitation stringency $E^{ref} = 0.01$ and $E^{ref} = 0.1$. The main difference is that more stringent sanitation re-set outflow infections to a lower level at each sanitation cycle thereby delaying the time when all animals exiting the market get infected.

Overall, these results highlight that frequent sanitation is a first-order intervention for maintaining market safety, while the stringency of sanitation is a necessary condition to ensure its effectiveness.

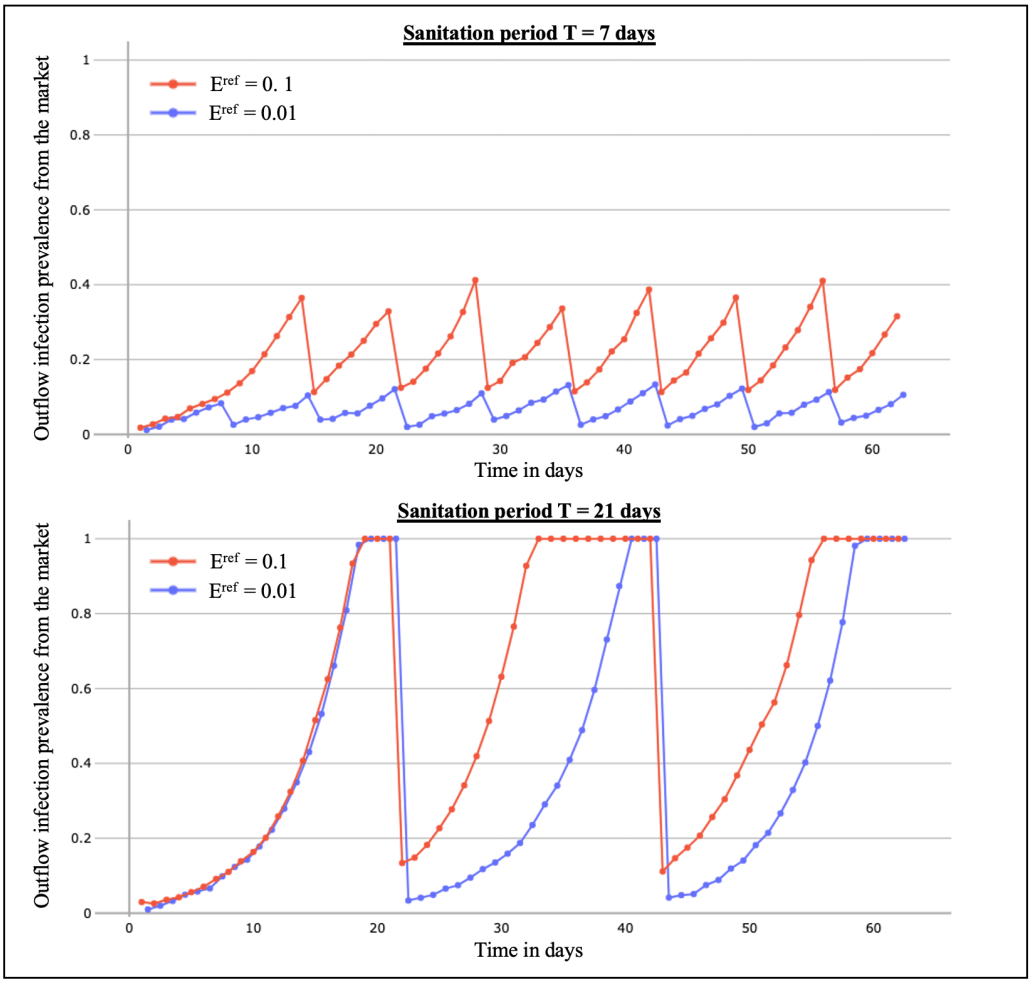
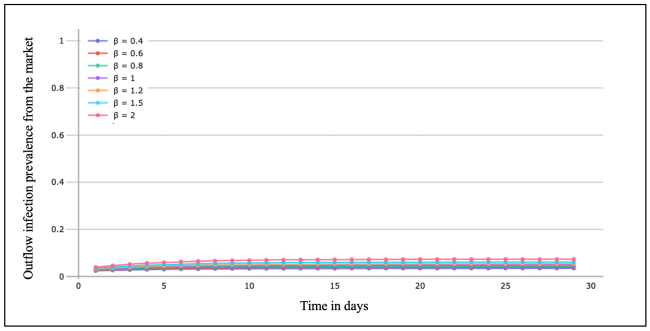


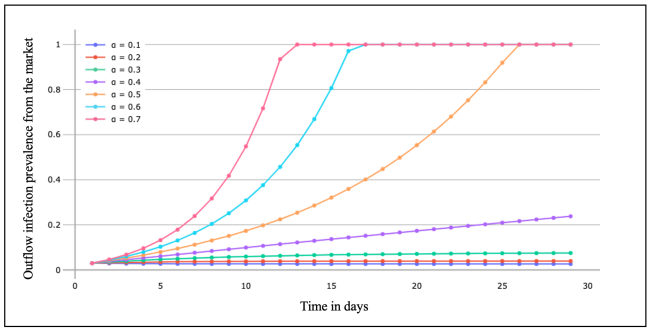
Figure O.1 **Outflow infection prevalence over time for different sanitation periods ($T = 7$ and $T = 21$ days) and sanitation stringency levels ($E^{\text{ref}} = 0.01$ and $E^{\text{ref}} = 0.1$).**

O.5. Sensitivity of Outflow Infection Prevalence Time Series to the TIP Model Parameters

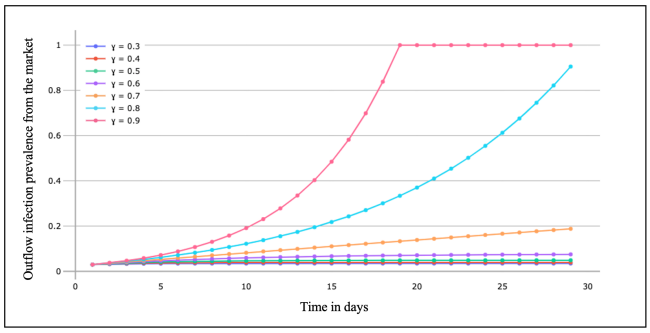
Figure O.2 the sensitivity of the outflow infection prevalence to each model parameter. The outflow infection prevalence is highly sensitive to the interaction rate parameter α and the persistence rate parameter γ, while variations in the transmission rate parameter β have a relatively minor effect. Since α and γ capture the role of the environment as a conduit for infections, this supports the paper's statement that the environment plays a central role in the spread and amplification of infections.



(a) Sensitivity to the transmission rate parameter β ($\alpha = 0.3, \gamma = 0.6$).



(b) Sensitivity to the interaction rate parameter α ($\beta = 1, \gamma = 0.6$).



(c) Sensitivity to the persistence rate parameter γ ($\beta = 1, \alpha = 0.3$).

Figure O.2 Sensitivity of outflow infection prevalence time series to the TIP model parameters

O.6. Sensitivity of Outflow Infection Prevalence Time Series to the Inflow Infection Prevalence

Figure O.3 presents a sensitivity analysis of the outflow infection prevalence time series for the TIP model with respect to the inflow prevalence, i.e. considering r = 0.01, r = 0.05, and r = 0.1. It specifically considers a highly pathogenic strain of the virus with the parameters $\beta = 1.5, \alpha = 0.5, \gamma = 0.6$. The results show that a higher inflow infection prevalence leads to a higher exponential growth of the outflow infection prevalence until saturation level 1.

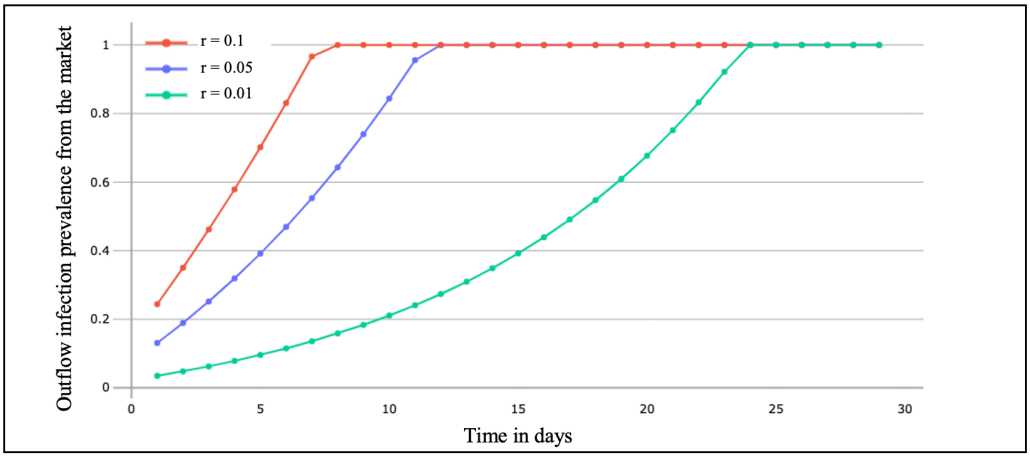
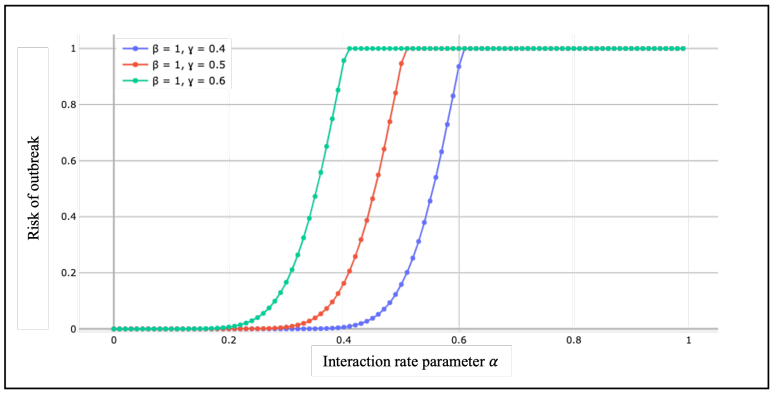


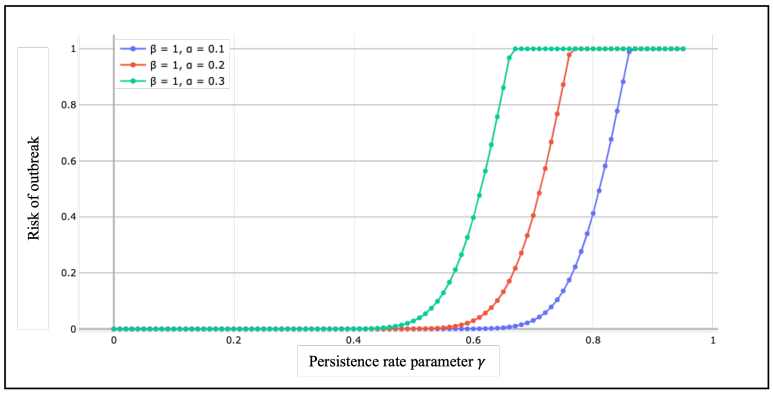
Figure O.3 **Sensitivity analysis of the outflow infection prevalence time series for the TIP model with respect to the inflow infection prevalence ($\beta = 1.5, \alpha = 0.5, \gamma = 0.6$).**

O.7. Sensitivity of the Risk of an Outbreak to the TIP Model Parameters

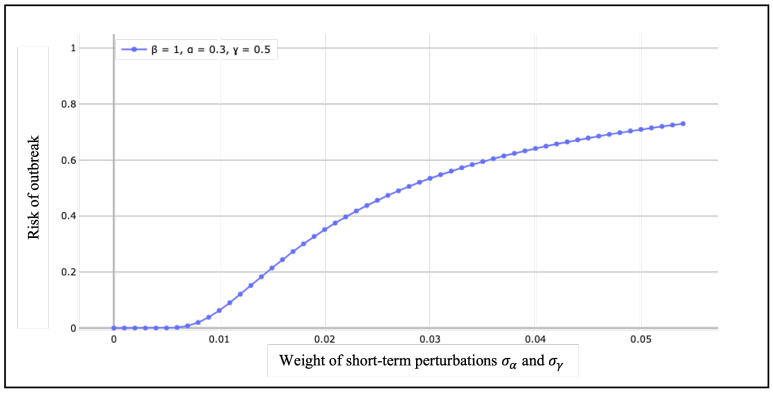
This section performs a sensitivity analysis on the risk of an outbreak measured by Theorem 2 with respect to the TIP model parameters. Specifically, Figures O.4a and O.4b and O.4c show the sensitivity of the risk of an outbreak with respect to the interaction rate α, persistence γ, and weights of perturbations $\sigma_\alpha = \sigma_\gamma$, respectively. Both figures demonstrate that the risk of an outbreak is highly sensitive to these parameters as relatively minor variations could transition this risk probability from almost 0 to 1. On the other hand, the weight of the short term perturbations of the reference parameters also affect this risk, in a rather logarithmic scale, as perturbation weights increase.



(a) Sensitivity to the interaction rate α with pre-set persistence rate γ (0.4, 0.5 and 0.6).



(b) Sensitivity of to the persistence rate γ with pre-set interaction rate α (0.1, 0.2 and 0.3).



(c) Sensitivity to the weight of short-term perturbations to the reference parameters values.

The perturbation weights σ_α and σ_γ are assumed equal and increasing together.

Figure O.4 Sensitivity of the risk of an outbreak under the deterministic TIP model parameters. The transmission rate parameter β does not influence this risk and has been set to 1.

O.8. Sensitivity of the Risk of an Outbreak to the Sanitation Period

Figures O.5 show how the risk of an outbreak varies with the sanitation period under different epidemiological parameter settings. The risk can rise from nearly zero to significant levels (e.g., above 10%) within a few days, depending on the parameters.

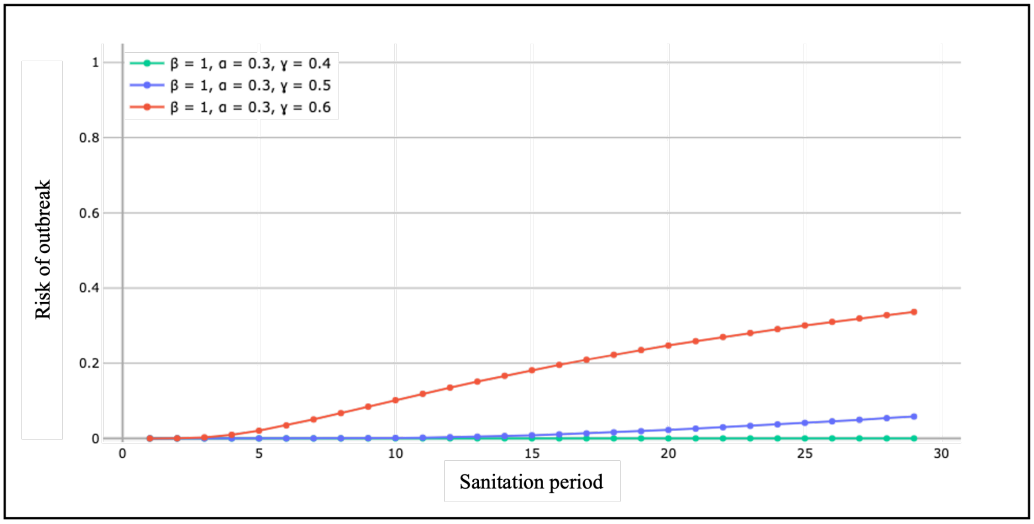


Figure O.5 Evolution of the risk of an outbreak with respect to the sanitation period for different parameter settings.

O.9. Seasonality of the Risk of an Outbreak

Additionally, the study analyzes the effect of seasonality on the risk of an outbreak. It denotes by δ the amplitude of the seasonal fluctuations (e.g., $\delta = 0.1$) and by t_{month} the day of the year at the start of each month (e.g., $t_{\text{January}} = 0$, $t_{\text{February}} = 31$). This allows the persistence rate parameter to fluctuate over time as: $\gamma_{\text{month}} = \gamma + \delta \cdot cos(\frac{2\pi t_{month}}{365})$, where γ is the reference value of the persistence rate parameter. The study considers fluctuations of amplitude 0.1, and seasonal variations every month, which can be adjusted depending on the region. Using these monthly seasonal effects, Figure O.6 presents the evolution of the risk of an outbreak over a year.

O.10. Sample Paths for Supply Chain Intervention Simulations

Figure O.7 shows four representative sample paths of the outflow infection prevalence over a single sanitation cycle for each combination of sanitation period and supply chain structure. In the consolidated network (100 farms supplying one market), trajectories grow smoothly and rapidly, often reaching saturation before the end of the cycle, especially when $T = 30$ days. In the deconsolidated network (100 farms supplying 20 markets), trajectories are more variable, with long periods of low or zero prevalence punctuated by sharp increases. These sharp increases are driven by the arrival of infected

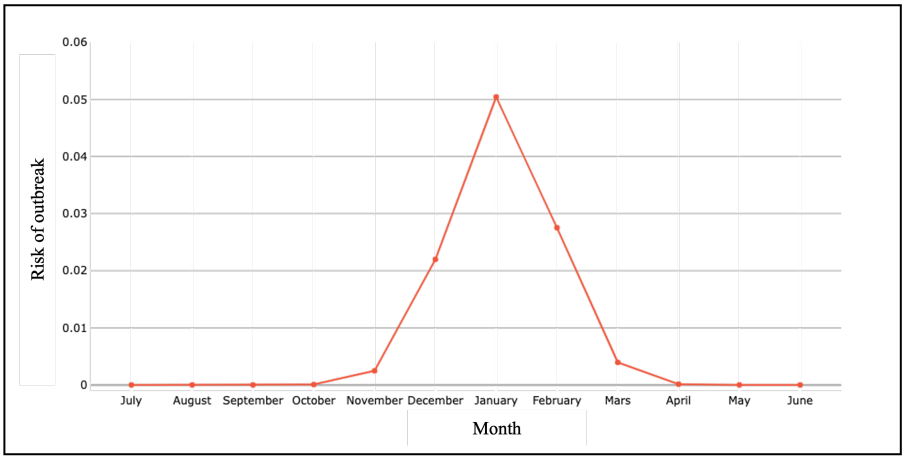


Figure O.6 **Evolution of the risk of an outbreak over a year, for the parameters $(\alpha = 0.3, \gamma = 0.5)$ and sanitation period of 7 days.**

batches, which represent a significant proportion of the overall inflow because markets are smaller in a deconsolidated supply chain structure. These examples illustrate how supply chain structure and sanitation frequency jointly shape the within-cycle dynamics underlying the end-of-cycle prevalence distributions in Figure 7.

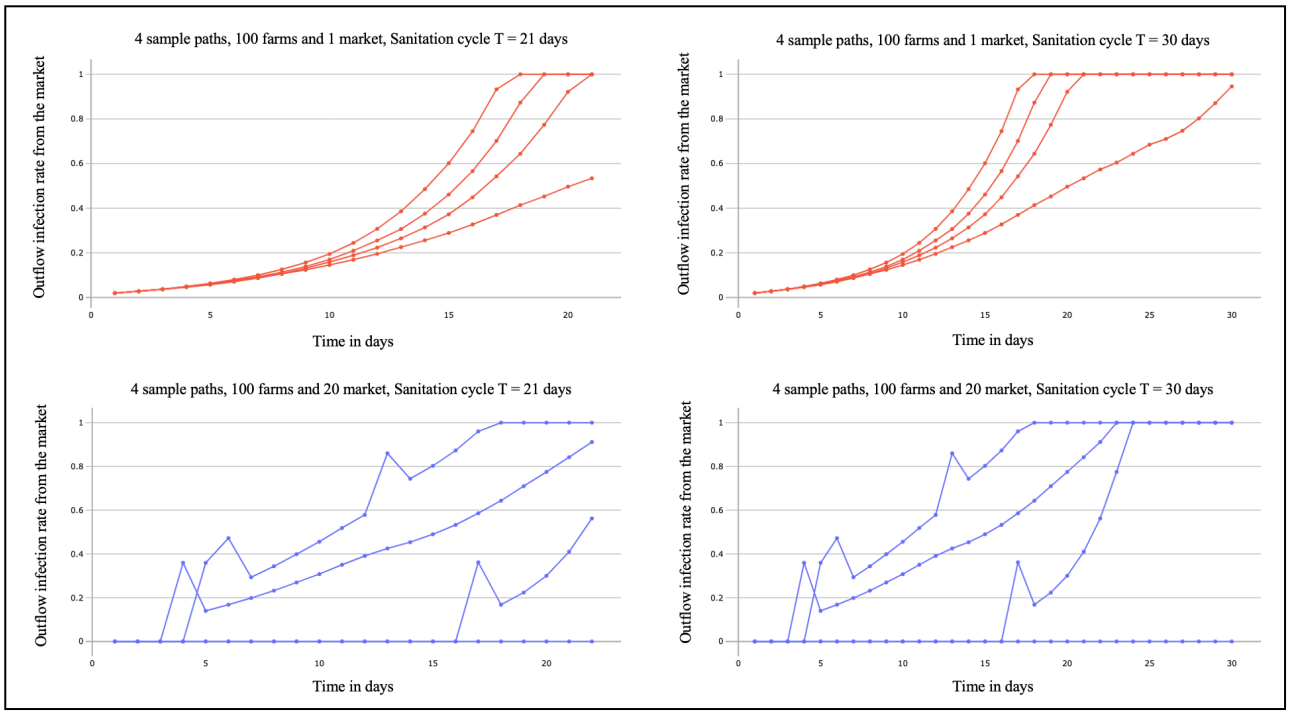


Figure O.7 **Representative sample paths of outflow infection prevalence over a sanitation cycle under two supply chain structures (consolidated: 100 farms–1 market; deconsolidated: 100 farms–20 markets) and two sanitation periods ($T = 21$, $T = 30$ days), with one randomly chosen farm infected at each time t and a highly pathogenic winter virus ($\beta = 1.5$, $\alpha = 0.5$, $\gamma = 0.6$).**