I. ANALYTICAL SOLUTION OF THE CONTACT-MEDIATED DYNAMICS MODEL

The following reduced model introduced in Eqs (3) – (4) represents dynamics of susceptible hosts, $S(t)$, and free viruses, $V(t)$:

$$\frac{dS}{dt} = -\phi SV(1 + \delta)$$  \hspace{1cm} (S.1)

$$\frac{dV}{dt} = -\phi SV$$  \hspace{1cm} (S.2)

This model can be solved analytically by observing that:

$$\frac{dS}{dV} = (1 + \delta)$$  \hspace{1cm} (S.3)

such that

$$S(t) = (1 + \delta)V(t) + \Omega$$  \hspace{1cm} (S.4)

where the integration constant, $\Omega$, can be solved using the initial conditions that $S(0) = S_0$ and $V(0) = V_0$:

$$\Omega = S_0 - (1 + \delta)V_0$$  \hspace{1cm} (S.5)

This two-dimensional system can be reduce to a one-dimensional system by substituting $S(t)$ as in Eq. (S.4), yielding:

$$\frac{dV}{dt} = -\phi V \left( (1 + \delta)V + \Omega \right)$$  \hspace{1cm} (S.6)

When $\Omega \neq 0$, then Eq. (S.6) can be solved by separation of variables yielding:

$$V(t) = \frac{M_0 \Omega e^{-\phi t t}}{1 - (1 + \delta)M_0 e^{-\phi t}}$$  \hspace{1cm} (S.7)

where $M_0$ is the initial population-level multiplicity of infection, i.e., the ratio $M_0 = V_0/S_0$. The solution for $V(t)$ is the basis for a complete description of the dynamics under the Quasi-steady state approximation:

$$S(t) = \frac{\Omega}{1 - (1 + \delta)M_0 e^{-\phi t}}$$  \hspace{1cm} (S.8)

$$V(t) = \frac{M_0 \Omega e^{-\phi t t}}{1 - (1 + \delta)M_0 e^{-\phi t}}$$

where, in addition:

$$C(t) = \frac{\phi S(t)V(t)}{k_f}$$

$$I(t) = V_0 - C(t) - V(t)$$

$$D(t) = (S_0 - S(t)) - (V_0 - V(t))$$  \hspace{1cm} (S.9)

When $\Omega = 0$, then Eq. (S.6) can be solved by integration:

$$S(t) = \frac{S_0}{1 + S_0 \phi t}$$

$$V(t) = \frac{V_0}{1 + V_0 \phi (1 + \delta)t}$$  \hspace{1cm} (S.10)

recalling that when $\Omega = 0$ then $(1 + \delta)V_0 = S_0$.

II. A MODEL WITH ENTRY-MEDIATED DORMANCY

Here we propose and analyze a model with viral entry-mediated dormancy. To incorporate viral entry mediated dormancy, we assume that upon viral entry into a cell, either the host become infected with probability $(1 - \tilde{p})$ or it fails to infect the host and host become dormant with probability $\tilde{p}$:

Susceptible $\frac{dS}{dt} = -k_+SV + (1 - \tilde{p})k_-C$

Complex $\frac{dC}{dt} = k_+SV - k_-C - k_fC$

Dormant $\frac{dD}{dt} = \tilde{p}k_-C + \frac{pk_-C}{k_fC}$  \hspace{1cm} (S.11)

Infected $\frac{dI}{dt} = (1 - \tilde{p})k_fC$

Free viruses $\frac{dV}{dt} = -k_+SV + k_-C$

where $\tilde{p}$, the probability of host being dormant after virus absorption, and $p$, the probability of contact mediated-host dormancy are free model parameters in the extended system. By QSSA (Quasi Steady State Assumption), we
can reduce the dimension of the system and by using control parameters in section II, we can rewrite the model as follows:

\[
\begin{align*}
\frac{dS}{dt} &= -\phi SV(1 + \delta) \\
\frac{dD}{dt} &= \phi SV(\check{p} + \delta) \\
\frac{dI}{dt} &= \phi SV(1 - \check{p}) \\
\frac{dV}{dt} &= -\phi SV
\end{align*}
\]

The solution of the system can be found analytically:

\[
\begin{align*}
S(t) &= \frac{\Omega}{1 - (1 + \delta)M_0e^{-\phi t}} \\
D(t) &= \frac{\delta + \check{p}}{1 + \delta} (S_0 - S(t)) \\
I(t) &= \frac{1 - \check{p}}{1 + \delta} (S_0 - S(t)) \\
V(t) &= \frac{M_0\Omega e^{-\phi t}}{1 - (1 + \delta)M_0e^{-\phi t}}
\end{align*}
\]

where \( \Omega = S_0 - (1 + \delta)V_0 \) and \( M_0 = V_0/S_0 \). These solutions hold so long as \( \Omega \neq 0 \). When \( \Omega = 0 \), similar to the original model, we obtain

\[
\begin{align*}
S(t) &= \frac{S_0}{1 + S_0\phi t} \\
V(t) &= \frac{V_0}{1 + V_0\phi(1 + \delta)t}
\end{align*}
\]

The system dynamics have qualitatively different behaviors for \( \Omega > 0 \) and for \( \Omega < 0 \) (see Table S.1).

Analysis of this system yields different qualitative behavior with respect to the same critical parameter \( \Omega \) as in the original system, namely the asymptotic dynamics exhibit the following dichotomy: either a host-depletion or a virus-depletion regime (Table S.1). As before, despite low MOI, \( M_0 \ll 1 \), we find that all of the hosts can undergo rapid dormancy, if viral contact-mediated dormancy probability, \( p \) is relatively large. However when viral contact-mediated dormancy is unlikely, \( p \to 0 \) in the system, then viral entry mediated dormancy leads to high dormancy only if the probability \( \check{p} \) is sufficiently large and \( V_0 \geq S_0 \). Consider the case, \( V_0 \ll S_0 \) and \( \delta \to 0 \) and \( \check{p} \to 1 \), such that most viruses enter cells and then induce dormancy. In this limit, \( \Omega > 0 \) and \( D = V_0 \). Therefore, only as many hosts can become dormant as there are viruses.

The analysis here implies that under low MOI, large scale dormancy cannot be explained with viral entry mediated dormancy as the only mechanism causing dormancy in the cell population. The current model neglects initiation of infection and cell-cell communication mediated by viral entry. Enhancements in dormancy due to those mechanisms cannot be ruled out by the current analysis.

### III. MODEL PARAMETERIZATION AND ANALYSIS GIVEN HOST GROWTH, DEATH AND RECOVERY

We parameterize the model in Eqs. (10) based on experiments from [10]. First, host-growth in the absence of viruses suggests \( r = 0.3 \text{ hrs}^{-1} \) and \( K = 9 \times 10^8 \text{ cells/ml} \). Next, the virus decay rate is estimated from free virus decay time series as \( d \approx 0.087 \text{ hrs}^{-1} \). Observations that dormant cells recover 48-72 hrs after initial virus exposure, inform our use of intervals of \( \gamma \in [\frac{1}{2}, \frac{1}{4}] \text{ hrs}^{-1} \). Finally, we assume that exposure to a (de-activated) virus comes with a potential cost, and vary the rate at which dormant and infected cells die via the free parameter, \( \mu \), assuming an approximately 50% chance of non-recovery. We present a series of analyses of the variation in dormancy induction given combinations of death and recover rates in Figure S.1.
FIG. S.1: Variation in dormancy induction given host demographic dynamics. Maximum dormant cell ratio w.r.t. enhancement factor $\delta$ a) when $\mu = 1/48$, and $\gamma = 1/36$; b) when no recovery ($\gamma = 0$), but cell birth/death and viral decay; c) when no cell death ($\mu = 0$) and no recovery ($\gamma = 0$), but others; d) when no cell death ($\mu = 0$), no recovery ($\gamma = 0$), no viral decay ($d = 0$); e) when no cell death ($\mu = 0$), no recovery ($\gamma = 0$), no viral decay ($d = 0$), no cell birth ($r = 0$) (Model 2). In (a), despite host demography (birth/death processes), infected cell virus clearance, dormant cells recovery and viral decay, contact mediated dormancy provides large scale dormancy given that viruses are present at low relative titer.