Supporting information

S1 Table

S1 Table.	Overview of available mathematical models of spatial virus
spread	

load				
Model	Modelling formalism	Key assumptions	Predictions	Applications
HIV propagation	3D stochastic	Diffusion-limited	Excitable	Relevant for
in space and	cellular	propagation of	medium type	in vitro system
in time [1]	automata	HIV by infected	of behavior.	as the effect
		CD4+ cells	Stability- and	of CD8+ T
			geometry limited	T cell is not
			regimes of	considered
			viral spread.	
			Propagations	
			regimes (1)	
			travelling wave.	
			(2) chaotic	
			steady state.	
			(3) extinguishing	
			of infection	
VSV growth	Set of reaction-	2D radial	Estimated of	In vitro studies
and spread [2]	diffusion	symmetry.	the parameters	of spatially
	PDEs, ODEs,	infection of	of VSV-BHK	spreading viruses
	Integro-	kidney cells,	infection growth	over multiple
	differential	IFN response	and spread	generations
	PDEs	-	*	characterized
				by immuno-
				histochemical
				labeling and
				digital imaging
IAV infection of	2D square	Infection	Distribution	Target-cell
humans $[3,4]$	lattice	grows	of initially	limited
	cellular	locally	infected cells	resolution
	automation	around	has a great	of infection,
	[3]; two-	infected	impact on	sustainable
	dimensional	site by	the infection	spread of
	sheet of	spreading	dynamics,	infection as a
	hexagonally	from	the impact	propagating
	-tilled	cell-to-cell	of the local	wave, low
	epithelial		versus global	level $(2\% \text{ of})$
	cells [4]		regeneration	infected cells)
			rules, the	persistence of
			importance	infection [3]
			of occasional	
			jumps in	
			viral spread	
			to previously	
			uninfected	
			areas	

Model	Modelling	Key assumptions	Predictions	Applications
	formalism			
Multi-	System of	Sessile	A robust	Analysis of
compartment	ODEs for	target and	persistence	HIV and SIV
virus-target	basic	infected cells.	of infection	dynamics
cell-immune	virus-	Spatial	for intermediate	around the
cell dynamics	immune	coupling	dispersal	viral set point
with a local	dynamics	by nearest	rate; rejected	
random	at 21x21	neighborhood	the "dynamic	
spread of	site grid	dispersal	elimination"	
the virus [5]	_	of the virus,	paradigm	
		predator-prey		
		type of virus-		
		immune		
		dynamics		
Dynamics of	System of	Cell-to-cell	Existence of	Key role
HIV-1 infected	distributed	spread of HIV-1,	sustained	of latently
target cells [6]	DDEs	well-mixed	oscillations	infected
		system	of infection	cells in
			for typical	sustaining
			tissue culture	the infection,
			parameters	HIV persistence
				in the form
				of infective
				oscillations
Spatiotemporal	System of PDEs	2D surface	The speed of	Initial foci
dynamics of	describing	domain,	the pattern	of HIV infection
virus - target	infection,	purely	propagation,	can be established
cell system	replication,	Brownian	the spatial	as a result of
in HIV	diffusion and	motion of	frequency and	the virus-target
infection [7]	chemotaxis	cells,	amplitude	cell dynamics;
		bounded rate	of the density	immune system
		chemotactic	variation	response may
		movement	(hot spots)	give rise to
				pattern formation

Model	Modelling	Key assumptions	Predictions	Applications
	formalism			
Viral infection	System of	3D view of	Analytical	Areas of
in spherical	reaction-	infected	solutions	high viral
organs [8]	diffusion	organ with	showing	concentration
	equations	a spherical	non-uniform	in organs;
	with influx	shape,	distribution	lack of
	boundary	viruses and	of viruses	accessibility
	conditions	immune cells	and cells in	of some inner
	and radial	penetrate	the organ;	parts of the
	symmetry	organ at	the role of	organs for
		the surface and	diffusivity	the immune
		propagate	parameters;	cells and
		to the inner	temporal and	antiviral drugs
		domain	spatial profiles	_
			of the infection	
			evolution	
Virus growth	System	Cell-to-cell	Growth of	Quantitative
in vitro [9]	of master	mode of viral	larger foci	analysis of
	equations for	spread in	is best explained	the efficacy
	the evolution	the in vitro	by the birth	for blocking HCV
	of the number	culture; no	model;	cell-to-cell
	of infected	cell death;	growth of	spread when
	cell in foci	empirical	the smaller	targeting
	for HCV	density	foci is better	different
		function for	described by	host factors
		the time to	the boundary	
		infection of	model	
		the founding		
		cell in a focus		

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