

A Bistable Reaction-Diffusion Analysis of Tumour to Determine Oncolytic Virotherapy Outcome

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ABSTRACT Oncolytic viruses selectively replicate inside cancer cells and spread through tumour tissue as an infection front, so that the therapeutic outcome is set not only by how strongly the virus replicates but by whether and where that front advances, stalls, or collapses. We cast spatial oncolytic virotherapy as a bistable reaction–diffusion problem in which uninfected and infected tumour populations are coupled through infection and lysis, and we map the outcomes across geometry and parameter space. Linear analysis of the local kinetics shows that the tumour-free state is stable while the fully established tumour is unstable to viral invasion, so an infection front can be launched from a localized inoculation. Simulations in one dimension, on a planar disc, and in a radially symmetric spheroid reveal three sharply distinct outcomes: complete eradication, in which the front traverses the whole tumour; partial remission by wave pinning, in which the front stalls at a finite position and leaves a viable residual rim; and treatment failure, in which the infection dies out. A two-parameter regime map identifies the infection rate as the decisive control, with a finite window separating pinning at lower values from failure at higher values, whereas the bistability threshold of the tumour kinetics has almost no influence. The infected-cell spread rate behaves similarly, with eradication confined to an intermediate band. These results frame eradication as a tuned, geometry-dependent property of a travelling front and provide a compact map from measurable kinetic parameters to therapeutic outcome.

KEYWORDS

Oncolytic
virotherapy
Reaction–
diffusion
Wave pinning
Travelling waves
Bistability
Mathematical
oncology

INTRODUCTION

Oncolytic virotherapy turns a pathogen into a treatment: engineered or naturally tumour-selective viruses replicate inside cancer cells, lyse them, and release progeny that infect neighbouring tumour tissue. The approach has matured from concept to clinic, with replication-competent agents now approved or in advanced trials and a growing appreciation that their action combines direct oncolysis with the recruitment of antitumour immunity (Russell *et al.* 2012; Kaufman *et al.* 2015; Andtbacka *et al.* 2015). What distinguishes virotherapy from most systemic treatments is that its active agent is self-amplifying and spatially propagating. The therapeutic agent does not simply diffuse to a uniform concentration; it advances through the tumour as a front of infection, and the fate of that front, whether it sweeps through the whole mass, stalls part-way, or burns out, determines whether the outcome is eradication, partial remission, or failure.

This spatial character places oncolytic virotherapy squarely within the theory of reaction–diffusion systems and travelling waves. The propagation of an advantageous or invasive species through space as a front of fixed shape and speed is one of the oldest results in mathematical biology, and the distinction between monostable fronts, which always invade, and bistable fronts, which invade only above a threshold and can be brought to rest, is central to that theory (Fisher 1937; Aronson and Weinberger 1978; Volpert and Petrovskii 2009; Murray 2003). Bistable kinetics are especially relevant to cancer, where a tumour population can be stable at both low and high densities with an unstable threshold between them, and reaction–diffusion descriptions have been used to capture invasion, growth and treatment response across several malignancies (Gatenby and Gawlinski 1996; Swanson *et al.* 2003; Maini *et al.* 2004).

A particularly informative phenomenon in such systems is wave pinning, in which a propagating front decelerates and halts at a finite position rather than traversing the domain, producing a stable, spatially localized boundary between two states (Mori *et al.* 2008). Translated to therapy, a pinned infection front is precisely the signature of partial remission: the virus clears the tumour up to a critical radius and no further, leaving a viable rim that can regrow.

Manuscript received: 12 June 2026,

Revised: 8 July 2026,

Accepted: 15 July 2026.

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Mathematical models of oncolytic virotherapy have illuminated many of its determinants, from the basic conditions for tumour remission to the roles of viral replication, infected-cell clearance, dosing schedule, and immune interaction, and they have done so with formulations ranging from well-mixed ordinary-differential-equation systems to spatially explicit and multiscale descriptions (Wodarz 2001; Wu *et al.* 2001; Wein *et al.* 2003; Friedman *et al.* 2006; Jenner *et al.* 2018; Pooladvand *et al.* 2021). Spatial structure has repeatedly been shown to matter, since a virus that would clear a well-mixed culture may fail to penetrate a solid tumour, and immunomodulation can shift the balance between containment and spread (Kim *et al.* 2015; Eftimie and Eftimie 2018). What is still lacking is a transparent, outcome-oriented map that connects the measurable kinetic parameters of the infection to the qualitative therapeutic result through the lens of front dynamics, and that does so consistently across the geometries in which tumours are actually studied, from one-dimensional sections to planar lesions and three-dimensional spheroids.

This study provides such a map. We formulate spatial oncolytic virotherapy as a two-component bistable reaction–diffusion system in which uninfected and infected tumour populations are coupled through infection and lysis, and we analyse it as a travelling-front problem. We first establish, through linear analysis of the local kinetics, that the tumour-free state is stable while the fully established tumour is unstable to viral invasion, so that an infection front can be launched from a localized inoculation. We then simulate the full system in one dimension, on a planar disc, and in a radially symmetric spheroid, and we classify every outcome as eradication, pinning, or failure using an automated front criterion. Finally, we sweep the governing parameters and construct a two-parameter regime map that pinpoints which quantities control the outcome and how sharply.

The central finding is that eradication is not generic but tuned: it occupies a finite window of infection rate bounded below by pinning and above by failure, it depends similarly on the infected-cell spread rate, and it is almost independent of the bistability threshold of the tumour kinetics. Framing eradication as a tuned property of a travelling front yields a compact and clinically suggestive picture of when spatial virotherapy succeeds.

MODEL AND METHODS

Bistable reaction–diffusion model

We represent a tumour as a two-component excitable tissue in which the normalized density of uninfected tumour cells $u(\mathbf{x}, t)$ and of infected, virus-bearing cells $w(\mathbf{x}, t)$ evolve by local kinetics and spatial spread,

$$\frac{\partial u}{\partial t} = D_u \nabla^2 u + r u (1 - u) (u - a) - \beta u w, \quad (1)$$

$$\frac{\partial w}{\partial t} = D_w \nabla^2 w + \beta u w - \delta w + \rho w (1 - w). \quad (2)$$

The cubic term $r u (1 - u) (u - a)$ endows the tumour with bistable growth: the tumour-free state $u = 0$ and the saturated state $u = 1$ are both locally stable, separated by an unstable threshold at $u = a$ with $0 < a < 1$. Infection transfers mass from the uninfected to the infected compartment through the bilinear coupling $\beta u w$, infected cells are cleared at rate δ through lysis and degradation, and intratumoural viral amplification is represented by the logistic replication term $\rho w (1 - w)$. The parameters D_u and D_w are the motilities of the uninfected and infected populations; the strong inequality $D_w \gg D_u$ that typifies a spreading infection is what

permits a self-limiting, pinnable front. Unless stated otherwise we use the reference parameter set $D_u = 0.02$, $D_w = 0.15$, $r = 0.5$, $a = 0.25$, $\beta = 0.8$, $\delta = 0.3$ and $\rho = 0.2$, expressed in the dimensionless units of the model.

All domains carry no-flux (Neumann) boundary conditions, representing a confined tumour mass with no cell flux across its border,

$$\left. \frac{\partial u}{\partial n} \right|_{\partial \Omega} = \left. \frac{\partial w}{\partial n} \right|_{\partial \Omega} = 0. \quad (3)$$

Equations (1)–(2) were discretized with second-order central finite differences and advanced in time by an explicit Euler scheme, with the step size chosen to satisfy the diffusive stability condition $\Delta t \leq (\Delta x)^2 / [4 \max(D_u, D_w)]$. The one-dimensional domain used six hundred grid points on an interval of length thirty; the planar domain used a 200×200 grid; and the spheroid was solved in radial coordinates with the symmetry condition $\partial_r u|_{r=0} = 0$.

Local kinetics and invasion condition

Setting the spatial derivatives to zero reduces the model to the homogeneous kinetics

$$\dot{u} = r u (1 - u) (u - a) - \beta u w, \quad \dot{w} = \beta u w - \delta w + \rho w (1 - w), \quad (4)$$

whose fixed points and their linear stability determine whether an infection front can be initiated. The eigenvalues of the Jacobian at each biologically relevant equilibrium were computed to classify it as stable or unstable; an infection front exists when the saturated tumour state is unstable to the introduction of infected cells, that is, when $\beta u^* - \delta + \rho > 0$ at $u^* = 1$.

Front tracking and outcome classification

In every geometry the wave-front position was defined as the location at which the uninfected-cell density crosses a fixed level, and its trajectory in time was recorded. Each simulation was then classified automatically into one of three outcomes. Eradication denotes a front that traverses the entire domain and drives the uninfected density below a small threshold everywhere. Pinning denotes a front that advances and then stalls at a stable interior position, leaving a viable residual tumour beyond it. Failure denotes the extinction of the infected population before substantial clearance, so that the tumour remains intact. To characterize the spheroid we additionally tracked the total tumour burden, the spatial integral of the uninfected density, as a scalar measure of therapeutic efficacy.

Parameter sweeps and regime mapping

We probed the determinants of outcome in two stages. One-dimensional sweeps varied each of the infection rate β , the bistability threshold a , and the infected-cell motility D_w in turn, classifying the outcome and, for pinned cases, recording the pinning position. We then constructed a two-parameter regime map over the (β, a) plane by running an ensemble of one-dimensional simulations and classifying each, which resolves the boundaries between eradication, pinning and failure and exposes which parameter actually controls the transition.

RESULTS

Local kinetics admit an invading infection front

The homogeneous kinetics possess the structure required for a launchable infection front. Three equilibria lie on the infection-free axis: the tumour-free state $(u, w) = (0, 0)$, the unstable growth

threshold at $(a, 0) = (0.25, 0)$, and the saturated tumour state $(1, 0)$. Linear analysis classifies the tumour-free state as stable, with both eigenvalues negative, while the saturated tumour state is unstable to infection, carrying a positive eigenvalue of 0.7 in the infected-cell direction that quantifies the invasion condition $\beta - \delta + \rho > 0$. Figure 1 shows the corresponding phase portrait, with the nullclines and the flow confirming that a small inoculation of infected cells into a saturated tumour grows and that the system is drawn toward the tumour-free state. This is the dynamical prerequisite for therapy: the established tumour is excitable to infection, and clearance is the stable end state once the front has passed.

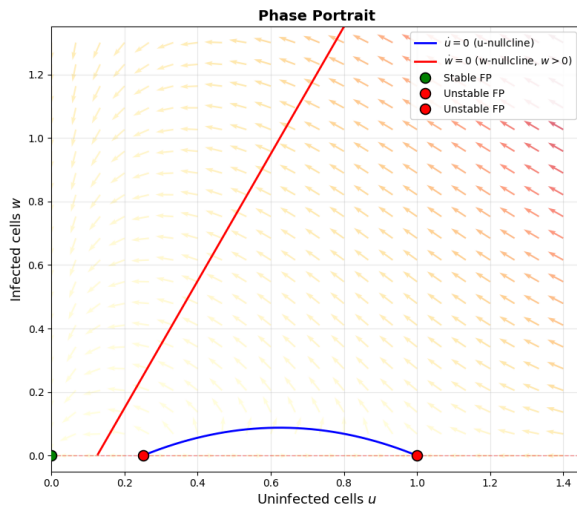


Figure 1 Phase portrait of the homogeneous kinetics, Eq. (4). The tumour-free state (green) is stable; the growth-threshold and saturated-tumour states (red) are unstable. The positive eigenvalue at the saturated state encodes the invasion condition that allows an infection front to be launched

One-dimensional front: propagation and eradication

At the reference parameters a localized viral inoculation at the left boundary launches an infection front that propagates into the saturated tumour at an essentially constant speed. Figure 2 shows the space–time evolution of both populations: the band of infected cells advances steadily across the domain while the uninfected density collapses behind it, so that the tissue passes from a fully established tumour to a cleared state in the wake of the front. The front position advances at a speed of approximately 0.6 in model units, reaching the far boundary at about forty-five time units, after which the residual infected population resolves and the tumour interface disappears entirely (Figure 3). The reference parameter set therefore yields complete eradication through a traversing travelling wave, the most favourable of the three outcomes.

The outcome is controlled by infection rate, not tumour threshold

Sweeping the governing parameters reveals a sharp organization of outcomes (Figure 4). Varying the infection rate β produces all three regimes in sequence: the infection fails at very low β , the front pins at an interior position over an intermediate band, and eradication is achieved once β is large enough, with the pinning position increasing monotonically with β before the transition to full

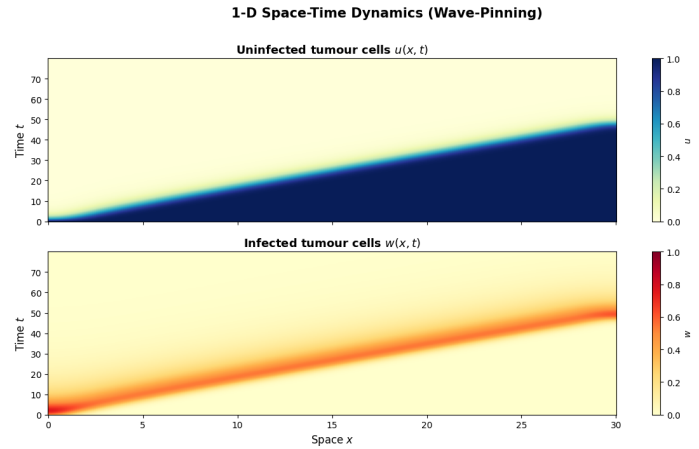


Figure 2 One-dimensional space–time dynamics at the reference parameters. Left: uninfected tumour cells $u(x, t)$, which are cleared in the wake of the advancing front. Right: infected cells $w(x, t)$, forming a travelling band that sweeps across the domain. The front advances at near-constant speed and reaches the far boundary by about forty-five time units, producing complete eradication

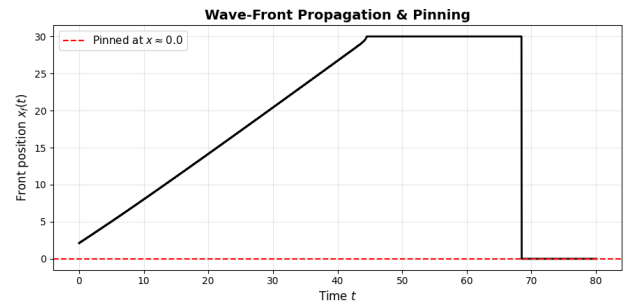


Figure 3 Front position over time at the reference parameters. The interface advances at constant speed to the far boundary, then collapses as the residual tumour is cleared, leaving no front, the signature of complete eradication

clearance. The infected-cell motility D_w behaves analogously, with eradication confined to an intermediate band; when viral spread is too slow the front pins, and when it is too fast the infection outruns and exhausts itself, and therapy fails. In sharp contrast, varying the bistability threshold a across its biologically admissible range leaves the outcome essentially unchanged, with eradication maintained throughout. The determinant of therapeutic success is thus the kinetics of infection and spread rather than the intrinsic growth threshold of the tumour.

The two-parameter regime map over the (β, a) plane confirms and sharpens this picture (Figure 5). The boundaries between failure, pinning and eradication run essentially vertically, that is, parallel to the a axis, so the outcome is set by β almost independently of the tumour threshold. Reading off the map, the infection fails below $\beta \approx 0.16$, the front pins for β between approximately 0.16 and 0.67 with a pinning position that grows steadily across this band, eradication holds for β between approximately 0.67 and 1.7, and the therapy fails again at the highest infection rates. The reference parameter set sits comfortably within the eradication region, consistent with the complete clearance observed in the simulations. The existence of an upper failure boundary is notable: an excessively aggressive infection is as counterproductive as an

Regime Diagram: Eradication vs. Pinning vs. Failure

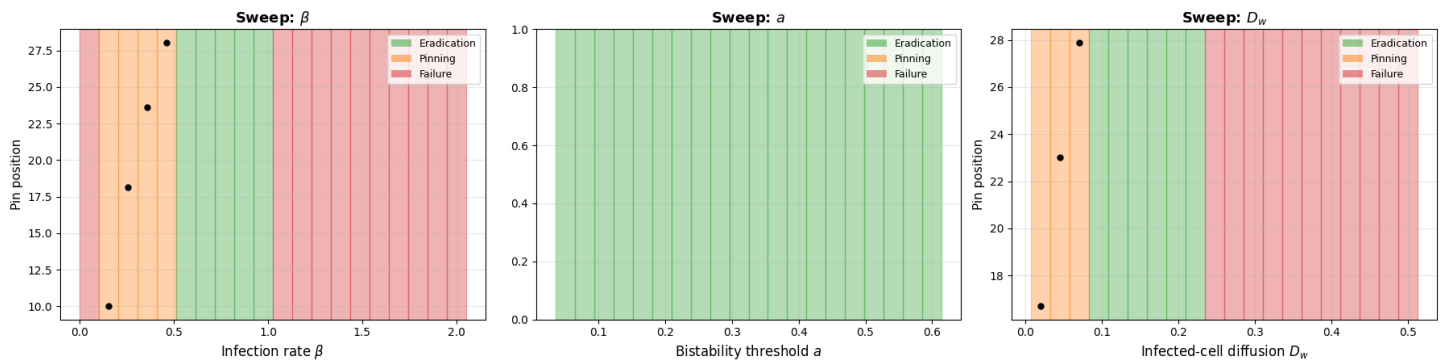


Figure 4 One-parameter outcome sweeps. Background colour marks eradication (green), pinning (orange) and failure (red); points show the pinning position where applicable. The infection rate β (left) and the infected-cell motility D_w (right) each admit an intermediate eradication window bounded by pinning and failure, whereas the bistability threshold a (centre) leaves the outcome unchanged

insufficient one, because it consumes the infected-cell population faster than the front can advance.

firms that, in the eradication regime, the geometry of the tumour does not rescue it from a sufficiently strong and sufficiently mobile infection.

DISCUSSION

The picture that emerges is that the success of spatial oncolytic virotherapy is best understood as a property of a travelling front, and that eradication is a tuned rather than a generic outcome. By formulating infection spread as a bistable reaction–diffusion process, we recovered the three therapeutically meaningful regimes, complete eradication, partial remission by pinning, and failure, as the three natural fates of such a front, and we showed that they are separated by sharp boundaries in parameter space. The most consequential result is the identification of the infection rate as the dominant control, with a finite eradication window bounded below by pinning and above by failure, and the near-irrelevance of the tumour’s intrinsic bistability threshold. The latter point is reassuring for translation: it implies that the outcome is governed by quantities one can in principle engineer or measure for the virus, the infectivity and the spread rate, rather than by a poorly constrained property of the host tumour kinetics.

The existence of wave pinning as an intermediate regime is biologically significant because it furnishes a clean mechanistic account of partial remission. A pinned front does not represent a failure of the virus to act but a balance in which clearance proceeds up to a critical radius and then halts, leaving a viable rim from which the tumour can regrow. That the pinning position increases with infection rate before giving way to full eradication suggests a graded, dose-like relationship between viral aggressiveness and the depth of clearance, up to the point at which the front breaks free and traverses the entire mass. Equally important is the upper failure boundary at high infection rate, which formalizes a counter-intuitive but recurring theme in virotherapy, that more aggressive infection is not always better: when infected cells are consumed faster than the front can propagate, the wave starves itself and the tumour is spared. Together these boundaries delimit a therapeutic window in the space of viral kinetics, and they argue for tuning rather than maximizing infectivity and spread.

These conclusions are consistent with, and extend, the existing modelling literature. Well-mixed analyses established the conditions under which a replicating virus can drive a tumour to remission, and spatial and multiscale models have repeatedly shown

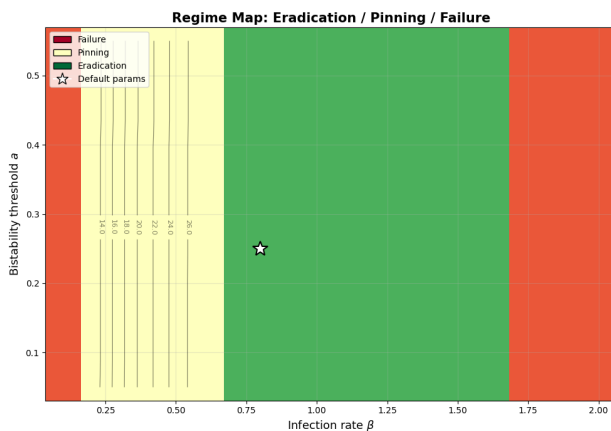


Figure 5 Two-parameter regime map over the (β, a) plane. Failure (red), pinning (yellow, with contours of pinning position) and eradication (green) are separated by near-vertical boundaries, showing that the infection rate β controls the outcome while the bistability threshold a is almost irrelevant. The star marks the reference parameters, which lie in the eradication region

Planar and spheroid geometries

The same front dynamics govern higher-dimensional, clinically realistic geometries. On a planar disc, a central inoculation produces a radially expanding ring of infection that clears the tumour from the inside outward; at the reference parameters the clearing wave consumes the entire lesion, and both the uninfected and infected populations have essentially vanished by about thirty-five time units (Figure 6). The radially symmetric spheroid tells the same story quantitatively (Figure 7). The infection front advances outward to a maximal radius of about 12.5 around twenty-six time units and then collapses inward as the residual core is cleared, while the total tumour burden, after a brief and slight initial rise as the untreated periphery grows, falls in a sigmoidal decline to near zero by roughly thirty-five time units. The burden curve provides a compact efficacy readout that would translate directly to a spheroid assay, and its monotone decline after the transient con-

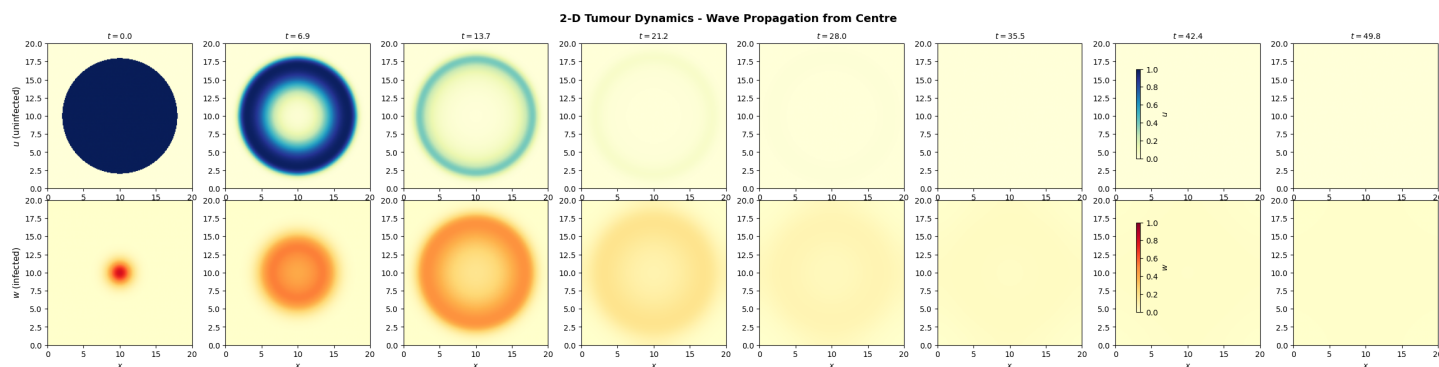


Figure 6 Two-dimensional dynamics from a central inoculation at the reference parameters. Top row: uninfected cells u ; bottom row: infected cells w , at successive times. A radially expanding infection ring clears the tumour from the centre outward, achieving complete eradication by about thirty-five time units

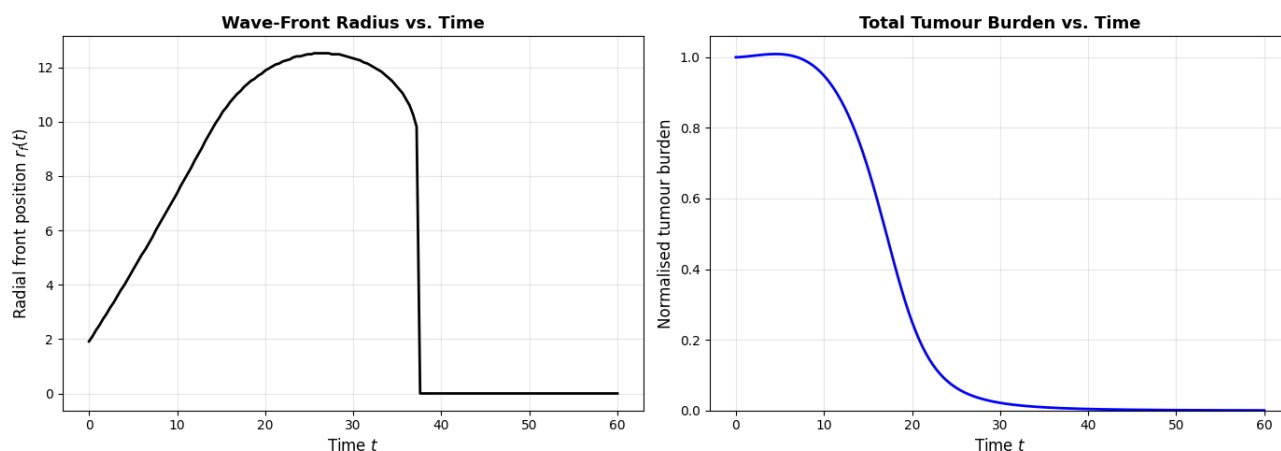


Figure 7 Radially symmetric spheroid at the reference parameters. Left: the infection front radius rises to a maximum of about 12.5 and then collapses as the core is cleared. Right: the total tumour burden declines sigmoidally to near zero by about thirty-five time units after a slight initial transient

that penetration and spread, not merely replication, govern solid-tumour outcomes (Wodarz 2001; Wu *et al.* 2001; Wein *et al.* 2003; Friedman *et al.* 2006; Paiva *et al.* 2009; Komarova and Wodarz 2010; Alzahrani *et al.* 2019). Our contribution is to organize these dependencies through the unifying language of front dynamics and wave pinning (Mori *et al.* 2008; Volpert and Petrovskii 2009), and to deliver an explicit, geometry-consistent map from kinetic parameters to outcome that holds from a one-dimensional section through a planar lesion to a three-dimensional spheroid. The robustness of the eradication outcome across geometries, together with the spheroid burden curve, points to a directly testable prediction for in vitro spheroid assays, in which the same intermediate-window behaviour in infectivity and spread should be observable.

Several simplifications bound the present analysis and define the next steps. The model tracks only uninfected and infected tumour cells and folds viral production into the infected-cell compartment; an explicit free-virus species with its own diffusion and decay would sharpen the description of spread and could shift the regime boundaries (Wu *et al.* 2001; Friedman *et al.* 2006). The kinetics are deterministic and homogeneous, omitting the tumour heterogeneity, stochastic inoculation, and physical barriers that shape real lesions, and, most importantly, the immune response is absent, even though immunomodulation is now understood to be central to virotherapy and can convert containment into clearance

or the reverse (Kaufman *et al.* 2015; Kim *et al.* 2015; Eftimie and Eftimie 2018; Jenner *et al.* 2018). Incorporating an immune compartment, an explicit virus field, anisotropic and heterogeneous tissue, and realistic dosing schedules are the natural extensions, as is calibration against spheroid and in vivo data so that the regime map can be placed on quantitative, patient-relevant axes. Within its scope, however, the analysis delivers a clear and useful message: spatial oncolytic virotherapy succeeds when a bistable infection front is tuned to traverse the tumour, and the parameters that tune it are those of infection and spread.

CONCLUSION

We have analysed spatial oncolytic virotherapy as a bistable reaction–diffusion front problem and mapped its outcomes across geometry and parameter space. Linear analysis showed that an established tumour is unstable to viral invasion while the tumour-free state is stable, so that an infection front can be launched and, if it traverses the tissue, can clear it. Simulations in one dimension, on a planar disc, and in a radially symmetric spheroid reproduced three sharply separated outcomes, eradication, pinning and failure, and a two-parameter regime map identified the infection rate as the decisive control, with a finite eradication window bounded by pinning below and failure above and with the tumour’s bistabil-

ity threshold almost irrelevant. Framing eradication as a tuned property of a travelling front, rather than as a generic consequence of viral replication, yields a compact and clinically suggestive map from measurable kinetic parameters to therapeutic outcome, and it provides a template for designing and interpreting spatial virotherapy in tumour spheroids and beyond.

Acknowledgments

The author thanks their respective institutions for support.

Availability of data and material

The simulation and analysis code that generated the datasets and figures of this study is available from the corresponding author on reasonable request.

Conflicts of interest

The author declares that there is no conflict of interest regarding the publication of this paper.

Ethical standard

The author has no relevant financial or non-financial interests to disclose.

LITERATURE CITED

- Alzahrani, E. O., A. A. Asiri, M. A. El-Khadra, and Y. Kuang, 2019 Mathematical modeling of oncolytic virotherapy. *Mathematical Biosciences* **312**: 69–80.
- Andtbacka, R. H. I., H. L. Kaufman, F. Collichio, T. Amatruda, N. Senzer, *et al.*, 2015 Talimogene laherparepvec improves durable response rate in patients with advanced melanoma. *Journal of Clinical Oncology* **33**: 2780–2788.
- Aronson, D. G. and H. F. Weinberger, 1978 Multidimensional nonlinear diffusion arising in population genetics. *Advances in Mathematics* **30**: 33–76.
- Eftimie, R. and G. Eftimie, 2018 Tumour-associated macrophages and oncolytic virotherapies: a mathematical investigation into a complex dynamics. *Letters in Biomathematics* **5**: S6–S35.
- Fisher, R. A., 1937 The wave of advance of advantageous genes. *Annals of Eugenics* **7**: 355–369.
- Friedman, A., J. P. Tian, G. Fulci, E. A. Chiocca, and J. Wang, 2006 A mathematical model for glioma growth and virotherapy treatment. *Mathematical Biosciences and Engineering* **3**: 531–549.
- Gatenby, R. A. and E. T. Gawlinski, 1996 A reaction-diffusion model of cancer invasion. *Cancer Research* **56**: 5745–5753.
- Jenner, A. L., C.-O. Yun, P. S. Kim, and A. C. F. Coster, 2018 Mathematical modelling of the interaction between cancer cells and an oncolytic virus: insights into the effects of treatment protocols. *Bulletin of Mathematical Biology* **80**: 1615–1629.
- Kaufman, H. L., F. J. Kohlhapp, and A. Zloza, 2015 Oncolytic viruses: a new class of immunotherapy drugs. *Nature Reviews Drug Discovery* **14**: 642–662.
- Kim, P. S., J. J. Crivelli, I.-K. Choi, C.-O. Yun, and J. R. Wares, 2015 Quantitative impact of immunomodulation versus oncolysis with cytokine-expressing virus therapeutics. *Mathematical Biosciences and Engineering* **12**: 841–858.
- Komarova, N. L. and D. Wodarz, 2010 ODE models for oncolytic virus dynamics. *Journal of Theoretical Biology* **263**: 530–543.
- Maini, P. K., D. L. S. McElwain, and D. I. Leavesley, 2004 Traveling wave model to interpret a wound-healing cell migration assay for human peritoneal mesothelial cells. *Tissue Engineering* **10**: 475–482.
- Mori, Y., A. Jilkin, and L. Edelstein-Keshet, 2008 Wave-pinning and cell polarity from a bistable reaction-diffusion system. *Biophysical Journal* **94**: 3684–3697.
- Murray, J. D., 2003 *Mathematical Biology II: Spatial Models and Biomedical Applications*. Springer, third edition.
- Paiva, L. R., C. Binny, S. C. Ferreira Jr, and M. L. Martins, 2009 A multiscale mathematical model for oncolytic virotherapy. *Cancer Research* **69**: 1205–1211.
- Pooladvand, P., C.-O. Yun, A.-R. Yoon, P. S. Kim, and F. Frascoli, 2021 The role of viral infectivity in oncolytic virotherapy outcomes: a mathematical study. *Mathematical Biosciences* **334**: 108520.
- Russell, S. J., K.-W. Peng, and J. C. Bell, 2012 Oncolytic virotherapy. *Nature Biotechnology* **30**: 658–670.
- Swanson, K. R., C. Bridge, J. D. Murray, and E. C. Alvord Jr, 2003 Virtual and real brain tumors: using mathematical modeling to quantify glioma growth and invasion. *Journal of the Neurological Sciences* **216**: 1–10.
- Volpert, V. and S. Petrovskii, 2009 Reaction-diffusion waves in biology. *Physics of Life Reviews* **6**: 267–310.
- Wein, L. M., J. T. Wu, and D. H. Kirn, 2003 Validation and analysis of a mathematical model of a replication-competent oncolytic virus for cancer treatment. *Cancer Research* **63**: 1317–1324.
- Wodarz, D., 2001 Viruses as antitumor weapons: defining conditions for tumor remission. *Cancer Research* **61**: 3501–3507.
- Wu, J. T., H. M. Byrne, D. H. Kirn, and L. M. Wein, 2001 Modeling and analysis of a virus that replicates selectively in tumor cells. *Bulletin of Mathematical Biology* **63**: 731–768.

How to cite this article: Vargas-Cabrera, L., and Munoz-Pacheco, J. M. A Bistable Reaction-Diffusion Analysis of Tumour to Determine Oncolytic Virotherapy Outcome. *Computers and Electronics in Medicine*, 3(2), 156–161, 2026.

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