

An SEAIHRS–W Norovirus Transmission Model with Asymptomatic Shedding, Hospitalisation, Waning Immunity and An Environmental Reservoir: Analysis Via The Multistage Homotopy Analysis Method

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ABSTRACT

Norovirus is the leading cause of acute gastroenteritis worldwide, and its outbreaks in schools are sustained by person-to-person contact and by virus shed into the shared environment. We formulate a seven-compartment SEAIHRS–W model with asymptomatic infection, hospitalisation, waning immunity and an environmental reservoir. We prove positivity, boundedness and existence of a unique endemic equilibrium when $\mathcal{R}_0 > 1$, derive $\mathcal{R}_0$ by the next-generation matrix, and establish global stability of the disease-free equilibrium for $\mathcal{R}_0 < 1$ and of the endemic equilibrium in the lifelong-immunity limit by Lyapunov functions. The system is solved by the multistage homotopy analysis method (MHAM), whose convergence on each stage is proven by a Banach fixed-point argument, with an explicit analyticity radius of at least 0.441 days. Calibrated to the 2001 Derbyshire primary-school outbreak (492 pupils, 201 cases), the model gives $\mathcal{R}_0 = 6.21$ and an initial effective reproduction number of 3.99, with $R^2 = 0.990$ for cumulative cases. Against an RK45 benchmark over 60 days, the third-order MHAM attains a maximum relative error of $1.47 \times 10^{-3}\%$. Sensitivity analysis ranks environmental transmission, virus decay and shedding as dominant, supporting combined hygiene and cleaning interventions.

Keywords: Norovirus, Environmental transmission, Multistage homotopy analysis method, Basic reproduction number, Lyapunov stability.

How to cite this article: Adhil Basha D, Balaganesan P, Sengothai R, Renuka J, An SEAIHRS–W Norovirus Transmission Model with Asymptomatic Shedding, Hospitalisation, Waning Immunity and An Environmental Reservoir: Analysis Via the Multistage Homotopy Analysis Method. *Int J Drug Deliv Technol.* 2026;16(85s): 1047-1068; DOI: 10.25258/ijddt.16.85s.110

Source of support: Nil

Conflict of interest: None

1. Introduction

1.1. Epidemiology and public health burden

Norovirus causes an estimated one in five cases of acute gastroenteritis globally and is responsible for a large share of gastroenteritis-related hospitalisations and deaths, particularly among young children and older adults [1]. Global estimates cited in recent modelling work put the annual toll at about 685 million infections, including roughly 200 million in children under five, around 200,000 deaths and an economic burden near 60 billion US dollars from health-care costs and lost productivity [2]. In the United States alone, norovirus produces roughly 19–21 million illnesses, 56,000–71,000 hospitalisations and 570–800 deaths each year [3]. Most deaths occur

in older adults, while the largest number of medically attended cases occurs in young children [1, 3].

The disease is short and usually self-limiting. After a median incubation period of about 1.2 days [4], infected people develop vomiting, diarrhoea, nausea and abdominal cramps that usually resolve within a few days. Treatment is supportive, based on rehydration, and no licensed vaccine or specific antiviral is available [5]. Noroviruses are genetically diverse: they are classified into several genogroups, of which GI, GII and GIV infect humans, and genogroup II accounts for most human infections [5]. This diversity, together with immunity that wanes over several years [6], allows repeated infection throughout life and complicates vaccine design.

1.2. Transmission routes and outbreak settings

Norovirus spreads by the faecal–oral route through direct person-to-person contact, contaminated food and water, and contaminated surfaces. Very few virus particles are needed to cause infection, infected people can shed virus in faeces for weeks, and the virus can remain infectious on surfaces for up to about two weeks [5]. A substantial fraction of infections is asymptomatic. Asymptomatic carriers are less infectious than symptomatic cases, but they shed virus for longer and may therefore sustain transmission between visible outbreaks [7, 6].

These features make semi-closed settings especially vulnerable. Analysis of 7094 outbreaks reported to the US National Outbreak Reporting System during 2009–2017 found a median basic reproduction number of 2.75. Long-term care facilities had the highest setting-specific value, 3.35, and schools, colleges and universities had 2.92 [8]. Transmission was lowest in summer and peaked in autumn and winter [8]. In schools, projectile vomiting in classrooms contaminates shared spaces and exposes many children at once. Bayesian analysis of a 2001 primary-school outbreak in Derbyshire, England, showed that vomiting events were an important route of spread in addition to ordinary person-to-person contact [9]. The same outbreak is used in the present study to calibrate the model.

1.3. Mathematical models of norovirus

Mathematical models have been used to estimate transmissibility, the duration of immunity and the effect of control measures. A review by Gaythorpe et al. [7] found published estimates of $\mathcal{R}_0$ ranging from 1.1 to 7.2. Population-level estimates were generally close to 2, while estimates from individual outbreaks were higher and more variable. The review also noted that most population-level models use SEIR-type structures without explicit environmental compartments, and that asymptomatic infection and waning immunity deserved more attention. Simmons et al. [6] fitted a transmission model with asymptomatic infection to surveillance data from England and Wales and estimated that immunity lasts 4.1–8.7 years. This is far longer than the months suggested by early challenge studies.

The environmental route has been examined explicitly in a smaller number of studies. Towers et al. [10] fitted a model with direct and environmental transmission to daily incidence from a cruise-ship outbreak. They estimated a combined reproduction number of 1.6 and found that direct transmission between passengers predominated in that setting. Wang et al. [11] fitted a model with two transmission

routes to 23 school outbreaks in Jiangsu Province, China. They estimated mean effective reproduction numbers of 8.92 for person-to-person spread and 2.19 for water- or food-borne spread, and showed that isolating symptomatic cases sharply reduces peak incidence.

A second line of work has focused on mathematical structure and numerical schemes. Raezah et al. [12] formulated a fractional-order model with vaccination and asymptomatic carriers under the Atangana–Baleanu–Caputo operator and solved it with an Adams–Bashforth scheme based on Newton polynomials. Cui et al. [13] combined a fractal–fractional operator with stochastic perturbations in a model with vaccination. Song et al. [14] studied a stochastic model driven by Lévy noise in which water and food contamination are explicit compartments. Ahmad et al. [15] analysed a fractional model with transmission through contaminated food and water. Saleem et al. [2] analysed a fractional SEIR model with a Grünwald–Letnikov nonstandard finite-difference scheme. Alqudah et al. [16] studied a discrete fractional SEAIRF model with an environmental reservoir and used a neural network to predict its dynamics.

Several recent studies have combined analysis with control. Ndendya et al. [17] modelled vaccination, treatment and public-health education, computed the reproduction number by a graph-theoretic method and estimated parameters from US outbreak surveillance data for 2023–2024. Gogovi et al. [5] included environmental reservoirs, waning immunity and reinfection, proved global stability by Lyapunov functions and solved an optimal control problem. They found that the environmental transmission and shedding rates dominate the reproduction number and that hand hygiene is the most effective single intervention.

1.4. Semi-analytical solution methods

Most of the studies above rely on numerical schemes, which give accurate trajectories but no explicit expression for the solution. Semi-analytical series methods instead produce closed-form approximations that show how each parameter enters the local dynamics and that can be differentiated directly. The homotopy analysis method (HAM) of Liao [18] constructs a continuous deformation from an initial guess to the exact solution. It contains an auxiliary convergence-control parameter $\hbar$ that can be used to enlarge the region of convergence. With a suitable choice of $\hbar$, HAM reduces to the homotopy perturbation method and to the Adomian decomposition method. Sufficient conditions for convergence of HAM series were given by Odibat

[19]. The Laplace–Adomian decomposition method has been applied to several epidemic models, including Lassa fever and malaria [20, 21].

A known weakness of all single-interval series methods is that a truncated series is accurate only near its expansion point. Epidemic curves evolve over weeks, so a single series is usually inadequate over the whole outbreak. The multistage homotopy analysis method (MHAM) restarts the series on short successive intervals and so extends the solution over long horizons. Its practical value, however, depends on knowing how long each interval may be before the series stops converging. Existing applications usually choose the step length by trial and error and verify convergence only numerically.

1.5. Research gap and contributions

To our knowledge, no published norovirus model combines (i) asymptomatic infection with reduced infectiousness, (ii) hospitalisation with disease-induced death, (iii) waning immunity and (iv) an environmental reservoir, together with a semi-analytical solution whose convergence is proven rather than assumed. In addition, few norovirus models that include an environmental compartment have been calibrated to case data from a documented outbreak.

This paper addresses these gaps. Its main contributions are as follows.

1. We formulate a seven-compartment SEAIHRS–W model and prove positivity, boundedness and existence of a positively invariant region.
2. We derive $\mathcal{R}_0$ by the next-generation matrix and decompose it into direct and environmental contributions from asymptomatic and symptomatic carriers. We also obtain the endemic equilibrium explicitly.
3. We prove global stability of the disease-free equilibrium for $\mathcal{R}_0 < 1$ by a Lyapunov function. For the endemic equilibrium we prove global stability only in the lifelong-immunity limit ($\omega = 0$), by a Volterra-type

Lyapunov function; with waning immunity ($\omega > 0$) we establish local stability for the calibrated parameters.

4. We derive explicit MHAM solutions from zeroth to third order for every compartment. We prove convergence on each stage by a Banach fixed-point argument that yields a computable bound on the stage length.
5. We calibrate the model to the Derbyshire 2001 school outbreak, quantify uncertainty by parametric bootstrap, examine identifiability of the transmission routes and of the assumed environmental decay, test out-of-sample prediction, and validate the MHAM solution against an RK45 benchmark.
6. We quantify the sensitivity of $\mathcal{R}_0$, of the peak size and timing and of cumulative cases by Latin hypercube sampling with partial rank correlation, and evaluate combined hygiene and cleaning interventions.

1.6. Organisation of the paper

Section 2 presents the model and its assumptions. Section 3 establishes the basic properties, equilibria and reproduction number, and Section 4 analyses local and global stability. Section 5 develops the MHAM solution and its convergence theory. Section 6 describes the data, calibration and numerical methods, and Section 7 reports the results. Sections 8–10 discuss the findings, limitations and public health significance, and Section 11 concludes.

2. Model formulation

The population of a closed or slowly renewing setting is divided into susceptible S , exposed (latent) E , asymptomatic infectious A , symptomatic infectious I , hospitalised H and recovered R individuals, with total $N = S + E + A + I + H + R$. The compartment W measures infectious virus in the shared environment, in normalised contamination units. The flow diagram is shown in Fig. 1.

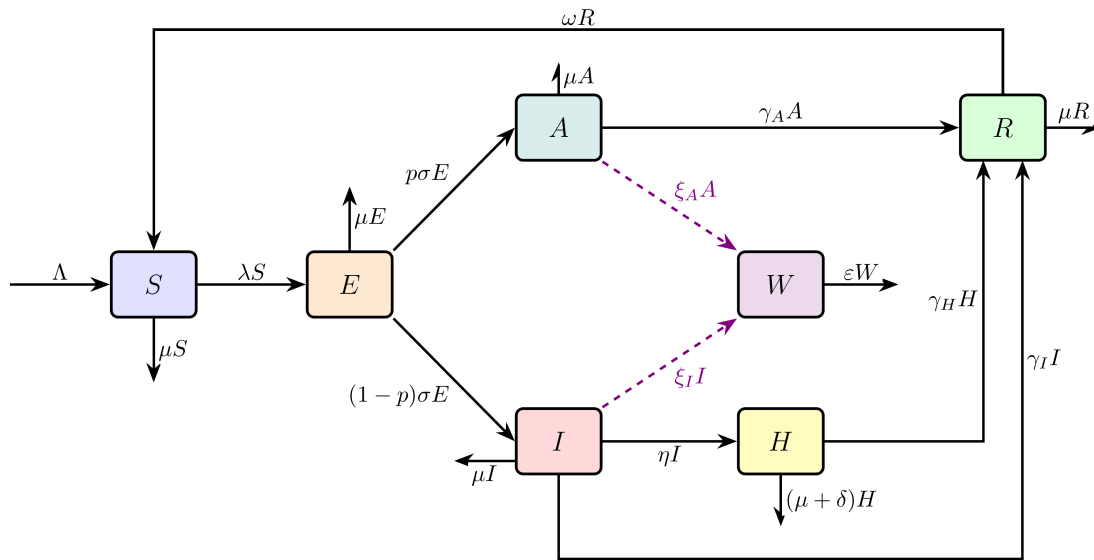


Fig. 1. Flow diagram of the SEAIHRS–W model (1)–(8). Solid arrows are transfers of individuals; dashed arrows are virus shedding into the environment. The force of infection is $\lambda = \beta(I + \kappa A) + \beta_W W$.

2.1. Assumptions

Assumption 1.

1. Mixing is homogeneous and transmission follows mass action, with force of infection $\lambda = \beta(I + \kappa A) + \beta_W W$, where $0 < \kappa < 1$ is the relative infectiousness of asymptomatic carriers.
2. A fraction p of exposed individuals becomes asymptomatic and $1 - p$ symptomatic after a mean latent period $1/\sigma$.
3. Only symptomatic cases are hospitalised; hospitalised patients are isolated and do not transmit, and experience disease-induced death at rate δ .
4. Infection confers immunity that wanes at rate ω .
5. Symptomatic and asymptomatic individuals shed virus into the environment at rates ξ_I and ξ_A ; environmental virus decays or is removed by cleaning at rate ε .

6. Recruitment Λ enters the susceptible class and all human classes exit at the natural rate μ .

2.2. Governing equations

Under Assumption 1 the dynamics are

$$\frac{dS}{dt} = \Lambda - \lambda S - \mu S + \omega R, \quad (1)$$

$$\frac{dE}{dt} = \lambda S - k_E E, \quad (2)$$

$$\frac{dA}{dt} = p\sigma E - k_A A, \quad (3)$$

$$\frac{dI}{dt} = (1 - p)\sigma E - k_I I, \quad (4)$$

$$\frac{dH}{dt} = \eta I - k_H H, \quad (5)$$

$$\frac{dR}{dt} = \gamma_A A + \gamma_I I + \gamma_H H - (\omega + \mu)R, \quad (6)$$

$$\frac{dW}{dt} = \xi_A A + \xi_I I - \varepsilon W, \quad (7)$$

$$\lambda = \beta(I + \kappa A) + \beta_W W, \quad (8)$$

with $k_E = \sigma + \mu$, $k_A = \gamma_A + \mu$, $k_I = \gamma_I + \eta + \mu$, $k_H = \gamma_H + \delta + \mu$ and non-negative initial data. The parameters are listed in Table 1.

Table 1. Parameters of model (1)–(8) for the Derbyshire school setting (rates per day).

Symbol	Description	Value	Source
Λ	Recruitment rate	μN_0 $= 0.01926$	Keeps $\Lambda/\mu = N_0 = 492$ [9]
μ	Natural exit rate	3.914×10^{-5}	70-year life span (assumed)
σ	Rate of leaving latency	$1/1.2$	Median incubation 1.2 d [4]
p	Asymptomatic fraction	0.30	[7]
γ_I	Recovery rate, symptomatic	$1/2$	[6]
γ_A	Recovery rate, asymptomatic	$1/10$	[6]
κ	Relative infectiousness of A	0.05	[6]
ω	Waning of immunity	$1/(5.1 \times 365)$	5.1-year immunity [6]
η	Hospitalisation rate	1.505×10^{-3}	0.3% of cases hospitalised [3]
γ_H	Discharge rate	$1/3$	Assumed (varied in Sect. 7.7)
δ	Disease-induced death in H	3.708×10^{-3}	1.1% in-hospital fatality [3]
ξ_I	Shedding by I	1	Normalisation of W
ξ_A	Shedding by A	$\kappa \xi_I = 0.05$	Scaled as κ [6]
ε	Environmental decay/cleaning	$1/3$	Assumed; surface survival of days [5]
β	Direct transmission rate	4.082×10^{-4}	Fitted (Sect. 6.1)
β_W	Environmental transmission rate	2.585×10^{-3}	Fitted (Sect. 6.1)
s_0	Initially susceptible fraction	0.643	Fitted (Sect. 6.1)

3. Basic mathematical properties

Theorem 1 (Positivity). *For non-negative initial data, the solution of (1)–(8) exists, is unique and remains non-negative for all $t \geq 0$; if $S(0) > 0$ then $S(t) > 0$ for all $t \geq 0$.*

Proof. The right-hand side of (1)–(8) is polynomial, hence locally Lipschitz, so a unique local solution exists. Each component is quasi-positive: when a state variable is zero and the others are non-negative, its derivative is non-negative ($\dot{S} = \Lambda + \omega R > 0$,

$\dot{E} = \lambda S \geq 0$, $\dot{A} = p\sigma E \geq 0$, $\dot{I} = (1-p)\sigma E \geq 0$, $\dot{H} = \eta I \geq 0$, $\dot{R} = \gamma_A A + \gamma_I I + \gamma_H H \geq 0$, $\dot{W} = \xi_A A + \xi_I I \geq 0$). Hence the non-negative orthant is positively invariant. Moreover $\dot{S} \geq -(\lambda(t) + \mu)S$, so $S(t) \geq S(0)\exp\{-\int_0^t (\lambda(\tau) + \mu) d\tau\} > 0$. Global existence follows from the bounds of Theorem 2.

Theorem 2 (Boundedness and invariant region). Let $\bar{\xi} = \max\{\xi_A, \xi_I\}$. The set

$$\Omega = \left\{ (S, E, A, I, H, R, W) \in \mathbb{R}_+^7 : N \leq \frac{\Lambda}{\mu}, W \leq \frac{\bar{\xi}\Lambda}{\mu\varepsilon} \right\} \quad (9)$$

is positively invariant and attracts every solution of (1)–(8).

Proof. Summing the human equations gives $\dot{N} = \Lambda - \mu N - \delta H \leq \Lambda - \mu N$. By Grönwall's inequality,

$$N(t) \leq N(0)e^{-\mu t} + \frac{\Lambda}{\mu}(1 - e^{-\mu t}), \quad (10)$$

so $N(0) \leq \Lambda/\mu$ implies $N(t) \leq \Lambda/\mu$ and $\limsup_{t \rightarrow \infty} N(t) \leq \Lambda/\mu$ in general. Then $\xi_A A + \xi_I I \leq \bar{\xi} N$, so $\dot{W} \leq \bar{\xi} N - \varepsilon W$, and the same

argument yields $W(t) \leq \bar{\xi}\Lambda/(\mu\varepsilon)$ whenever it holds initially, with the corresponding limsup bound otherwise. ■

3.1. Equilibria and basic reproduction number

The disease-free equilibrium (DFE) is $X_0 = (S_0, 0, 0, 0, 0, 0)$ with $S_0 = \Lambda/\mu$. Following van den Driessche and Watmough [22], take the transmitting compartments $x = (E, A, I, W)^T$ (H does not transmit and is omitted without loss). The matrices of new infections and of transfers at the DFE are

$$F = \begin{pmatrix} 0 & \beta\kappa S_0 & \beta S_0 & \beta_W S_0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} k_E & 0 & 0 & 0 \\ -p\sigma & k_A & 0 & 0 \\ -(1-p)\sigma & 0 & k_I & 0 \\ 0 & -\xi_A & -\xi_I & \varepsilon \end{pmatrix}. \quad (11)$$

Since $F = e_1 b^\top$ with $b = S_0(0, \beta\kappa, \beta, \beta_W)^\top$, the matrix FV^{-1} has rank one and $\mathcal{R}_0 =$

$\rho(FV^{-1}) = b^\top V^{-1} e_1$. Computing $V^{-1} e_1$ (Appendix A) gives

$$\mathcal{R}_0 = \frac{S_0\sigma}{k_E} \left[\frac{p}{k_A} (\beta\kappa + \frac{\beta_W \xi_A}{\varepsilon}) + \frac{1-p}{k_I} (\beta + \frac{\beta_W \xi_I}{\varepsilon}) \right] = \mathcal{R}_{dA} + \mathcal{R}_{dI} + \mathcal{R}_{eA} + \mathcal{R}_{eI}, \quad (12)$$

where the four terms are, respectively, the secondary infections produced directly by an asymptomatic or a symptomatic carrier and indirectly through the virus they shed.

Theorem 3 (Endemic equilibrium). System (1)–(8) has a unique endemic equilibrium X^* in the interior of Ω if and only if $\mathcal{R}_0 > 1$. It is given by

$$S^* = \frac{S_0}{\mathcal{R}_0}, \quad (13)$$

$$E^* = \frac{\Lambda(1-1/\mathcal{R}_0)}{k_E - \omega r}, \quad (14)$$

$$A^* = \frac{p\sigma}{k_A} E^*, \quad (15)$$

$$I^* = \frac{(1-p)\sigma}{k_I} E^*, \quad (16)$$

$$H^* = \frac{\eta}{k_H} I^*, \quad (17)$$

$$R^* = r E^*, \quad (18)$$

$$W^* = \frac{\xi_A A^* + \xi_I I^*}{\varepsilon}, \quad (19)$$

where

$$r = \frac{1}{\omega + \mu} \left[\frac{\gamma_A p \sigma}{k_A} + \frac{\gamma_I (1-p)\sigma}{k_I} + \frac{\gamma_H \eta (1-p)\sigma}{k_I k_H} \right]. \quad (20)$$

if $\Lambda(1 - 1/\mathcal{R}_0) > 0$, that is $\mathcal{R}_0 > 1$, and all other components are then positive. ■

4. Stability analysis

Table 2 summarises what is proven in this section. The distinction between the two immunity cases matters. Global stability of the endemic equilibrium is proven only when immunity is lifelong ($\omega = 0$). For the biologically relevant case of waning immunity ($\omega > 0$), only local stability is established, and only for the calibrated parameter values.

Table 2. Summary of the stability results and the conditions under which they hold.

Equilibrium	Condition	Result	Status
Disease-free	$\mathcal{R}_0 < 1$, any $\omega \geq 0$	Globally asymptotically stable	Proven (Theorems 4, 5)
Disease-free	$\mathcal{R}_0 > 1$	Unstable	Proven (Theorem 4)
Endemic	$\mathcal{R}_0 > 1$, $\omega = 0$	Globally asymptotically stable	Proven (Theorem 6)
Endemic	$\mathcal{R}_0 > 1$, $\omega > 0$	Locally asymptotically stable	Shown for calibrated parameters only (Proposition 1); global stability open

4.1. Local stability

Theorem 4 (Local stability of the DFE). *The DFE is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.*

Proof. Ordering the variables as $(S, H, R | E, A, I, W)$, the Jacobian at X_0 is block triangular. The non-transmitting block has

$$a_2 = k_E k_A + k_E k_I + k_A k_I + \varepsilon(k_E + k_A + k_I) - S_0 \beta \sigma \theta, \quad (23)$$

$$a_3 = \varepsilon(k_E k_A + k_E k_I + k_A k_I) + k_E k_A k_I - S_0 \beta \sigma [\varepsilon \theta + (1-p)k_A + p\kappa k_I] - S_0 \beta_W \sigma [(1-p)\xi_I + p\xi_A], \quad (24)$$

$$a_4 = k_E k_A k_I \varepsilon (1 - \mathcal{R}_0), \quad (25)$$

where the identity for a_4 was verified symbolically. If $\mathcal{R}_0 > 1$ then $a_4 < 0$, so P has a positive real root and the DFE is unstable. If $\mathcal{R}_0 < 1$, then $V - F$ is a

$$a_1 > 0, \quad a_3 > 0, \quad a_4 > 0, \quad a_1 a_2 a_3 > a_3^2 + a_1^2 a_4, \quad (26)$$

hold exactly when $\mathcal{R}_0 < 1$. All seven eigenvalues are then negative and the DFE is locally asymptotically stable. ■

For the calibrated parameters ($\mathcal{R}_0 = 6.21$), $a_3 = -0.585$ and $a_4 = -0.0726$, and the Jacobian at the DFE has the positive eigenvalue 0.417 d^{-1} . This is the initial exponential growth rate of the outbreak in a fully susceptible school.

$$(1, 2.109, 1.614, 0.4416, 0.03403, 2.895 \times 10^{-4}, 1.411 \times 10^{-5}, 5.519 \times 10^{-10}). \quad (27)$$

Its Hurwitz determinants are $\Delta_1 = 2.109$, $\Delta_2 = 2.963$, $\Delta_3 = 1.157$, $\Delta_4 = 3.81 \times 10^{-2}$, $\Delta_5 = 3.85 \times 10^{-6}$, $\Delta_6 = 5.36 \times 10^{-11}$ and $\Delta_7 = 2.96 \times 10^{-20}$, all positive. By the Routh–Hurwitz criterion all eigenvalues have negative real part; they are $-0.827 \pm 0.370i$, -0.337 , -0.116 , $-0.00150 \pm 0.0209i$ and -3.92×10^{-5} . ■

The slow complex pair $-0.0015 \pm 0.021i$ describes damped recurrent waves with a period of about $2\pi/0.021 \approx 300$ days, which arise from waning immunity. This is a statement about the long-run endemic state of a renewing population, not about the single outbreak studied below.

eigenvalues $-\mu$, $-k_H$ and $-(\omega + \mu)$. The transmitting block is $F - V$, whose characteristic polynomial is

$$P(\lambda) = \lambda^4 + a_1 \lambda^3 + a_2 \lambda^2 + a_3 \lambda + a_4, \quad (21)$$

with, writing $\theta = 1 - p + p\kappa$,

$$a_1 = k_E + k_A + k_I + \varepsilon, \quad (22)$$

non-singular M-matrix because $\rho(FV^{-1}) < 1$ [22]; therefore every eigenvalue of $F - V$ has negative real part. Equivalently, the Routh–Hurwitz conditions for the quartic,

Proposition 1 (Local stability of the endemic equilibrium, calibrated case). *For the parameters of Table 1, X^* is locally asymptotically stable.*

Proof. The characteristic polynomial of the 7×7 Jacobian at X^* has coefficients

4.2. Global stability

Theorem 5 (Global stability of the DFE). *If $\mathcal{R}_0 < 1$, the DFE is globally asymptotically stable in Ω .*

Proof. In Ω we have $S \leq N \leq S_0$. The transmitting subsystem can therefore be written as $\dot{x} = (F - V)x - f(x)$, where $f = ((S_0 - S)\lambda, 0, 0, 0)^T \geq 0$. Because V is a non-singular M-matrix, $V^{-1} \geq 0$, so the row vector $w^T = b^T V^{-1}$ is non-negative. Following the construction of Shuai and van den Driessche [23], consider

$$L(x) = w^T x = b^T V^{-1} x \geq 0. \quad (28)$$

Using $FV^{-1} = e_1 b^T V^{-1}$ we obtain $b^T V^{-1} F = \mathcal{R}_0 b^T$, and hence

$$\dot{L} = b^T V^{-1} (F - V)x - w^T f = (\mathcal{R}_0 - 1) b^T x - w^T f \leq (\mathcal{R}_0 - 1) b^T x \leq 0. \quad (29)$$

If $\dot{L} = 0$ then $b^\top x = S_0(\beta\kappa A + \beta I + \beta_W W) = 0$, so $A = I = W = 0$. Invariance of $A \equiv 0$ forces $p\sigma E \equiv 0$, so $E \equiv 0$. On $\{E = A = I = W = 0\}$ the remaining equations are linear: $\dot{H} = -k_H H$, $\dot{R} = \gamma_H H - (\omega + \mu)R$, $\dot{S} = \Lambda - \mu S + \omega R$. The only solution that remains in the compact set Ω for all $t \in \mathbb{R}$ is $H = R = 0$ and $S = S_0$. By LaSalle's invariance principle every solution in Ω converges to X_0 , and together with Theorem 4 the DFE is globally asymptotically stable. ■

$$\mathcal{L} = S^* g\left(\frac{S}{S^*}\right) + E^* g\left(\frac{E}{E^*}\right) + c_A A^* g\left(\frac{A}{A^*}\right) + c_I I^* g\left(\frac{I}{I^*}\right) + c_W W^* g\left(\frac{W}{W^*}\right), \quad (30)$$

with $c_A = (T_A + T_{WA})/(p\sigma E^*)$, $c_I = (T_I + T_{WI})/((1-p)\sigma E^*)$ and $c_W = T_W/(\varepsilon W^*)$. Put $x = S/S^*$, $y = E/E^*$, $a = A/A^*$, $i = I/I^*$ and $z = W/W^*$. Differentiating

$$\begin{aligned} \dot{\mathcal{L}} = & -\frac{\mu S^*(x-1)^2}{x} + T_A \left(3 - \frac{1}{x} - \frac{xa}{y} - \frac{y}{a}\right) + T_I \left(3 - \frac{1}{x} - \frac{xi}{y} - \frac{y}{i}\right) \\ & + T_{WA} \left(4 - \frac{1}{x} - \frac{xz}{y} - \frac{a}{z} - \frac{y}{a}\right) + T_{WI} \left(4 - \frac{1}{x} - \frac{xz}{y} - \frac{i}{z} - \frac{y}{i}\right). \end{aligned} \quad (31)$$

In each bracket the product of the subtracted terms equals one, so by the arithmetic–geometric mean inequality every bracket is non-positive. Hence $\dot{\mathcal{L}} \leq 0$, with equality only when $x = 1$ and $y = a = i = z$. The largest invariant subset of this set is $\{X^*\}$, because $x \equiv 1$ forces $\lambda = \lambda^*$ and then $y = 1$. LaSalle's principle gives global attractivity of $(S^*, E^*, A^*, I^*, W^*)$. The linear equations for H and R are then asymptotically autonomous and converge to H^* and R^* . ■

Remark 1. For $\omega > 0$ the return flow ωR couples the recovered class back into S , and the Volterra function above no longer yields a sign-definite derivative. Global stability of X^* for $\omega > 0$ is

$$(1-q)\mathcal{L}[\phi(t;q) - u_0] = q\hbar\mathcal{N}[\phi(t;q)], \quad \phi(t_k;q) = u_k, \quad (32)$$

where $q \in [0,1]$ is the embedding parameter and $\hbar \neq 0$ is the convergence-control parameter.

$$u_m(s) = \chi_m u_{m-1}(s) + \hbar \int_0^s \mathcal{R}_m(\tau) d\tau, \quad \mathcal{R}_m = u'_{m-1} - f_{m-1}, \quad (33)$$

where $\chi_1 = 0$, $\chi_m = 1$ for $m \geq 2$, and f_{m-1} is the coefficient of q^{m-1} in $f(\sum_j u_j q^j)$. For the bilinear incidence term this coefficient is the Cauchy

Theorem 6 (Global stability of the EE in the lifelong-immunity limit). Let $\omega = 0$ and $\mathcal{R}_0 > 1$. Then X^* is globally asymptotically stable in the interior of Ω .

Proof. When $\omega = 0$ the subsystem for (S, E, A, I, W) does not depend on (H, R) . Let $g(z) = z - 1 - \ln z \geq 0$, with equality only at $z = 1$. At X^* , split the incidence as $S^*\lambda^* = T_A + T_I + T_W$, with $T_A = \beta\kappa S^*A^*$, $T_I = \beta S^*I^*$ and $T_W = \beta_W S^*W^*$. Split T_W further as $T_{WA} + T_{WI}$ in proportion to $\xi_A A^*$ and $\xi_I I^*$. Define

along solutions, using the equilibrium relations $\Lambda = S^*\lambda^* + \mu S^*$, $k_E E^* = S^*\lambda^*$, $p\sigma E^* = k_A A^*$, $(1-p)\sigma E^* = k_I I^*$ and $\varepsilon W^* = \xi_A A^* + \xi_I I^*$, and collecting terms by transmission pathway gives

therefore not claimed here. For the calibrated parameters, local stability is established in Proposition 1.

5. Multistage homotopy analysis method

5.1. Formulation

Write (1)–(8) as $\dot{u} = f(u)$ with $u = (S, E, A, I, H, R, W)^\top$. Partition $[0, T]$ into stages $[t_k, t_{k+1}]$ of length Δt , and let $s = t - t_k \in [0, \Delta t]$. On each stage, choose the auxiliary linear operator $\mathcal{L} = d/dt$, the auxiliary function $H(t) \equiv 1$ and the initial guess $u_0(s) \equiv u_k$, the MHAM value at t_k . With the nonlinear operator $\mathcal{N}[\phi] = \partial_t \phi - f(\phi)$, the zeroth-order deformation equation is

Expanding $\phi(t;q) = u_0 + \sum_{m \geq 1} u_m(s)q^m$ gives the m th-order deformation equations

product $\sum_{j=0}^{m-1} S_j \lambda_{m-1-j}$, with $\lambda_j = \beta(I_j + \kappa A_j) + \beta_W W_j$. The M th-order stage

approximation is $\Phi_M(s) = \sum_{m=0}^M u_m(s)$, and $u_{k+1} = \Phi_M(\Delta t)$ starts the next stage.

5.2. Closed-form solutions up to third order

Let F_X denote the right-hand side of (1)–(8) for compartment X evaluated at u_k :

$$F_S = \Lambda - \lambda_k S_k - \mu S_k + \omega R_k, \quad (34)$$

$$F_E = \lambda_k S_k - k_E E_k, \quad (35)$$

$$F_A = p\sigma E_k - k_A A_k, \quad (36)$$

$$F_I = (1-p)\sigma E_k - k_I I_k, \quad (37)$$

$$F_H = \eta I_k - k_H H_k, \quad (38)$$

$$F_R = \gamma_A A_k + \gamma_I I_k + \gamma_H H_k - (\omega + \mu) R_k, \quad (39)$$

$$F_W = \xi_A A_k + \xi_I I_k - \varepsilon W_k, \quad (40)$$

$$K_S = -(\lambda_k + \mu)G_S - S_k \ell_2 - 2F_S \ell_1 + \omega G_R, \quad (49)$$

$$K_E = \lambda_k G_S + S_k \ell_2 + 2F_S \ell_1 - k_E G_E, \quad (50)$$

$$K_A = p\sigma G_E - k_A G_A, \quad (51)$$

$$K_R = \gamma_A G_A + \gamma_I G_I + \gamma_H G_H - (\omega + \mu)G_R, \quad (54)$$

$$K_W = \xi_A G_A + \xi_I G_I - \varepsilon G_W. \quad (55)$$

Solving (33) successively gives, for every $X \in \{S, E, A, I, H, R, W\}$,

$$\lambda_k = \beta(I_k + \kappa A_k) + \beta_W W_k. \quad (41)$$

With $\ell_1 = \beta(F_I + \kappa F_A) + \beta_W F_W$, define the second-derivative quantities

$$G_S = -(\lambda_k + \mu)F_S - S_k \ell_1 + \omega F_R, \quad (42)$$

$$G_E = \lambda_k F_S + S_k \ell_1 - k_E F_E, \quad (43)$$

$$G_A = p\sigma F_E - k_A F_A, \quad (44)$$

$$G_I = (1-p)\sigma F_E - k_I F_I, \quad (45)$$

$$G_H = \eta F_I - k_H F_H, \quad (46)$$

$$G_R = \gamma_A F_A + \gamma_I F_I + \gamma_H F_H - (\omega + \mu)F_R, \quad (47)$$

$$G_W = \xi_A F_A + \xi_I F_I - \varepsilon F_W. \quad (48)$$

With $\ell_2 = \beta(G_I + \kappa G_A) + \beta_W G_W$, define the third-derivative quantities

$$K_I = (1-p)\sigma G_E - k_I G_I, \quad (52)$$

$$K_H = \eta G_I - k_H G_H, \quad (53)$$

$$X_0(s) = X_k, \quad (56)$$

$$X_1(s) = -\hbar F_X s, \quad (57)$$

$$X_2(s) = -\hbar(1 + \hbar)F_X s + \hbar^2/2 G_X s^2, \quad (58)$$

$$X_3(s) = -\hbar(1 + \hbar)^2 F_X s + \hbar^2(1 + \hbar)G_X s^2 - \hbar^3/6 K_X s^3. \quad (59)$$

Summing, the third-order MHAM solution on stage k is

$$X(t_k + s) \approx X_k - \hbar(3 + 3\hbar + \hbar^2)F_X s + \hbar^2(3 + 2\hbar)/2 G_X s^2 - \hbar^3/6 K_X s^3. \quad (60)$$

For $\hbar = -1$, (60) reduces to the third-order Taylor polynomial $X_k + F_X s + G_X s^2/2 + K_X s^3/6$, which is the homotopy perturbation (and Adomian) result for the same linear operator. The formulas (56)–(59) were checked against the numerical implementation to machine precision (difference below 1.4×10^{-17} at $\hbar = -0.8$).

5.3. Convergence

Lemma 1 (Analyticity radius via Banach's fixed-point theorem). *Let $b > 0$ and let $B = \{z \in \mathbb{C}^7: \|z - u_k\|_\infty \leq b\}$. Set $M_f = \sup_B \|f\|_\infty$ and $L = \sup_B \|Df\|_\infty$. If $0 < r < \min\{b/M_f, 1/L\}$, then*

the solution U of $\dot{U} = f(U)$, $U(t_k) = u_k$, extends analytically to the complex disc $|\tau| < r$ around t_k .

Proof. Let X_r be the set of functions φ that are continuous on the closed disc $D_r = \{\tau \in \mathbb{C}: |\tau| \leq r\}$, analytic in its interior, and satisfy $\|\varphi - u_k\|_\infty \leq b$. With the supremum norm X_r is complete, because uniform limits of analytic functions are analytic. Define $(\mathcal{T}\varphi)(\tau) = u_k + \int_0^\tau f(\varphi(\zeta)) d\zeta$, integrating along the segment $[0, \tau]$. Then $\|\mathcal{T}\varphi - u_k\| \leq rM_f \leq b$, so $\mathcal{T}$ maps X_r into itself. Moreover $\|\mathcal{T}\varphi - \mathcal{T}\psi\| \leq rL \|\varphi - \psi\|$ with

$rL < 1$, so $\mathcal{T}$ is a contraction. By Banach's fixed-point theorem $\mathcal{T}$ has a unique fixed point, which is analytic and coincides with $U(t_k + \tau)$ on the real segment. ■

Theorem 7 (Convergence of MHAM on each stage). Let r_k be a radius given by Lemma 1 at u_k , and let $c(\hbar) = |\hbar|/(1 - |1 + \hbar|)$ for $-2 < \hbar < 0$. If $c(\hbar)\Delta t < r_k$, then the series $\sum_m u_m(s)$ converges uniformly for $s \in [0, \Delta t]$ to the exact solution $U(t_k + s)$. Moreover there exist $C > 0$ and $\varrho > 1$ such that

$$\partial_t \phi = \tau(q) f(\phi), \quad \tau(q) = \frac{-q\hbar}{1-q(1+\hbar)}, \quad \phi(t_k; q) = u_k. \quad (62)$$

Its solution is the time-rescaled trajectory $\phi(t_k + s; q) = U(t_k + \tau(q)s)$, with $\tau(0) = 0$ and $\tau(1) = 1$. The $u_m(s)$ are therefore the Maclaurin coefficients in q of $g(q) = U(t_k + \tau(q)s)$. For $|1 + \hbar| < 1$, τ is analytic on $|q| < 1/|1 + \hbar|$, and for $|q| \leq \varrho$ we have $|\tau(q)| \leq |\hbar|\varrho/(1 - \varrho|1 + \hbar|)$. Since $c(\hbar)\Delta t < r_k$, continuity provides $\varrho > 1$ with $|\tau(q)|s < r_k$ for all $|q| \leq \varrho$ and $s \in [0, \Delta t]$. By Lemma 1, g is analytic on $|q| < \varrho$ and bounded there by $C = \|u_k\| + b$. Cauchy's estimate gives $\|u_m(s)\| \leq C\varrho^{-m}$, so the series converges geometrically at $q = 1$ to $g(1) = U(t_k + s)$, and summing the tail gives (61). For $\hbar \in [-1, 0)$ we have $c(\hbar) = 1$. For $\hbar \in (-2, -1)$ we have $c(\hbar) = |\hbar|/(2 + \hbar)$, and the condition becomes $|\hbar| < 2r_k/(r_k + \Delta t)$. ■

For the calibrated outbreak, evaluating Lemma 1 along the trajectory (optimising over b) gives $\min_k r_k = 0.441$ days and $\max_k r_k = 0.721$ days. With $\Delta t = 0.25$ days, Theorem 7 therefore guarantees convergence on every stage for $\hbar \in (-1.276, 0)$, which includes $\hbar = -1$ and the optimal values found below. The observed ratios $\|u_{m+1}(\Delta t)\|/\|u_m(\Delta t)\|$ on the first stage are 0.104, 0.117, 0.080, 0.044 and 0.049 for $m = 1, \dots, 5$. Their maxima over all 240 stages are 0.106, 0.117, 0.099, 0.079 and 0.075. These values are consistent with the geometric decay in (61).

$$\|U(t_k + s) - \Phi_M(s)\| \leq C \frac{\varrho^{-(M+1)}}{1-\varrho^{-1}}. \quad (61)$$

In particular, convergence holds for all $\hbar \in [-1, 0)$ when $\Delta t < r_k$, and for $\hbar \in (-2r_k/(r_k + \Delta t), -1)$.

Proof. With $\mathcal{L} = d/dt$, $H \equiv 1$ and a constant u_0 , (32) becomes $(1 - q - q\hbar)\partial_t \phi = -q\hbar f(\phi)$, that is,

6. Numerical simulation methodology

6.1. Data and calibration

We use the norovirus outbreak in a primary school and nursery in Derbyshire, England, in 2001. Of 492 children, 186 were absent with gastrointestinal symptoms, and 201 children had a recorded illness onset or absence over 28 days [9]. The date of illness onset was used where recorded, and otherwise the first day of absence, to build daily and cumulative onset series for days 0–28. The model was started with one symptomatic case, $I(0) = 1$, a susceptible fraction S_0 ($S(0) = S_0 N_0 - 1$), and the remaining pupils immune, $R(0) = (1 - S_0)N_0$. The immune pupils represent prior immunity and genetic resistance. Cumulative symptomatic onsets $C(t)$ were obtained by augmenting (1)–(8) with $\dot{C} = (1 - p)\sigma E$.

We estimated $(\mathcal{R}_0, S_0, \phi_W)$ by bounded trust-region least squares on cumulative onsets. Here $\phi_W = (\mathcal{R}_{eA} + \mathcal{R}_{eI})/\mathcal{R}_0$ is the environmental share of transmission, and β and β_W follow from $\mathcal{R}_0$ and ϕ_W . The share was bounded by $0 \leq \phi_W \leq 0.95$, so that at least 5% of transmission is by direct contact. All other parameters were fixed at the values in Table 1.

Uncertainty and identifiability. Uncertainty was quantified in four ways. (i) A parametric bootstrap: 200 synthetic daily series were drawn from a negative binomial distribution with mean equal to the fitted daily incidence and dispersion $k = 1.47$, estimated from the daily residuals by the method of moments. The model was refitted to each cumulative series, and 95% intervals were taken as the 2.5th and 97.5th percentiles. (ii) A profile over ϕ_W on the grid

0, 0.1, ..., 0.9, 0.95, 0.99, refitting $\mathcal{R}_0$ and s_0 at each value. (iii) Refits with the environmental persistence $1/\varepsilon$ fixed at 1, 2, 3, 5, 7 and 14 days, to quantify how the estimates depend on this assumed parameter. (iv) An out-of-sample check, in which the model was fitted to days 0–20 only and used to predict days 21–28. Validation against further, independent outbreaks was not possible, because no other norovirus outbreak with openly available daily case data from a comparable setting was accessible to us.

6.2. Benchmark, software and tolerances

The benchmark is the adaptive Runge–Kutta 4(5) method (RK45) of SciPy’s *solve_ivp* [24], with relative and absolute tolerances of 10^{-12} (7376 function evaluations on $[0, 60]$). Its accuracy was checked against the eighth-order *DOP853* solver at 10^{-13} ; the two differ by at most 2.2×10^{-11} individuals, so benchmark error is negligible at the precision reported. MHAM was implemented with exact polynomial arithmetic in s (NumPy 2.4.4) using $\Delta t = 0.25$ days (240 stages) and, as a check, $\Delta t = 0.1$ days. The symbolic identity for a_4 and the closed forms (56)–(59) were verified in SymPy 1.14.0. All computations used Python 3.12 with SciPy 1.17.1, and figures were produced with Matplotlib 3.10.8 as vector PDF files. The optimal $\hbar$ minimises the averaged squared residual $E_M(\hbar) = \frac{1}{K} \sum_k \|\Phi_M'(s_k) - f(\Phi_M(s_k))\|_2^2$, evaluated at the midpoint of every stage on $[0, 30]$ [18]. Global

sensitivity of $\mathcal{R}_0$ was assessed with Latin hypercube sampling ($n = 5000$, seed 2026) of 13 parameters, each drawn uniformly on $\pm 25\%$ of its baseline, and partial rank correlation coefficients (PRCC) with t -tests [25]. The same procedure ($n = 2000$, seed 7) was applied to three outbreak outputs: the peak number of symptomatic cases, the time of that peak and the cumulative symptomatic cases by day 60. For these, 13 epidemiological parameters were varied with the initial conditions held at the fitted values.

7. Results

7.1. Calibration to the Derbyshire outbreak

The least-squares fit gives $\mathcal{R}_0 = 6.21$, $s_0 = 0.643$ and $\phi_W = 0.95$; the last value lies on the imposed upper bound. The corresponding rates are $\beta = 4.08 \times 10^{-4}$ and $\beta_W = 2.59 \times 10^{-3}$ per day. The initial effective reproduction number is $\mathcal{R}_{\text{eff}}(0) = s_0 \mathcal{R}_0 = 3.99$. For cumulative onsets the fit attains $R^2 = 0.990$ with a root-mean-square error of 7.8 cases (Fig. 2a). It predicts 207 cases by day 28 against 201 observed.

For daily onsets R^2 is only 0.374 (Fig. 2b). The daily series is dominated by reporting structure: absences accumulate on Mondays after weekends, and the school closure and two weekend cleanings are not represented in an autonomous model. The model captures the timing and overall size of the epidemic, not its day-to-day shape. This limitation is listed prominently in Section 9.

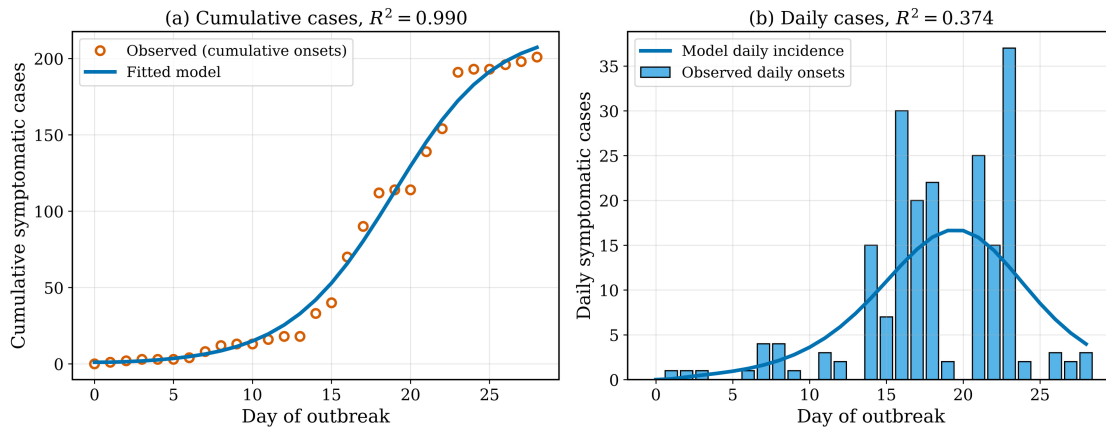


Fig. 2. Calibration to the Derbyshire 2001 school outbreak. (a) Observed cumulative symptomatic onsets and the fitted model, $R^2 = 0.990$. (b) Observed daily onsets and the model incidence, $R^2 = 0.374$.

The profile in Table 3 shows how the fit changes with the environmental share. The fit improves steadily as ϕ_W increases, but only from $R^2 = 0.985$ to 0.990, while the estimate of $\mathcal{R}_0$ rises from 2.42 to

6.21. The improvement is monotone, so the optimum is pushed to the bound rather than located in the interior. The data therefore do not identify ϕ_W independently. At the baseline, the contributions to

$\mathcal{R}_0$ in (12) are $\mathcal{R}_{dA} = 0.030$, $\mathcal{R}_{dI} = 0.280$, $\mathcal{R}_{eA} = 0.572$ and $\mathcal{R}_{eI} = 5.325$. These values follow from the bound on ϕ_W and should not be read as an estimate of how transmission actually divides between routes.

Table 3. Profile of the fit over the environmental share ϕ_W (cumulative onsets, days 0–28).

ϕ_W	$\mathcal{R}_0$	s_0	$\mathcal{R}_{\text{eff}}(0)$	SSE	R^2
0.00	2.42	0.864	2.09	2562	0.9848
0.25	3.01	0.794	2.39	2381	0.9858
0.50	3.81	0.733	2.79	2172	0.9871
0.75	4.93	0.680	3.35	1942	0.9884
0.95	6.21	0.643	3.99	1760	0.9895

7.2. Uncertainty and identifiability

Table 4 gives the bootstrap intervals and Fig. 3 summarises the identifiability analysis. The initial effective reproduction number is the best-determined quantity: $\mathcal{R}_{\text{eff}}(0) = 3.99$, with a 95% interval of 2.08–4.50. The basic reproduction number is much less certain (2.35–8.46), because it trades off against the susceptible fraction; the bootstrap correlation between $\mathcal{R}_0$ and s_0 is -0.71 . The environmental share is effectively unidentified. Its bootstrap interval spans the whole admissible range, 0.00–0.95, and 62.5% of the replicate fits end on the upper bound. The profile in Fig. 3a gives a narrower 95% likelihood set, $\phi_W \geq 0.8$. That set assumes independent Gaussian errors, which cumulative data violate, so it overstates the precision and the bootstrap interval should be preferred.

Table 4. Point estimates and 95% parametric-bootstrap intervals ($B = 200$, negative binomial observation noise).

Quantity	Estimate	95% interval
$\mathcal{R}_0$	6.21	2.35–8.46
$\mathcal{R}_{\text{eff}}(0) = s_0 \mathcal{R}_0$	3.99	2.08–4.50
s_0	0.643	0.419–1.000
ϕ_W	0.95 (bound)	0.00–0.95
β (d ⁻¹)	4.08×10^{-4}	2.87×10^{-4} – 4.45×10^{-3}
β_W (d ⁻¹)	2.59×10^{-3}	0– 3.53×10^{-3}

The assumed decay rate ε matters even more (Fig. 3c and Table 5). Refitting with mean persistence $1/\varepsilon$ between 1 and 14 days leaves the fit almost unchanged (R^2 between 0.988 and 0.990) but moves $\mathcal{R}_0$ from 3.81 to the search limit of 15, and $\mathcal{R}_{\text{eff}}(0)$ from 2.73 to 9.24. Longer persistence makes each

shedding event infectious for longer, so a higher $\mathcal{R}_0$ is needed to reproduce the same epidemic curve. $\mathcal{R}_0$ is therefore conditional on the assumed persistence, and the baseline value 6.21 should be read together with the assumption $1/\varepsilon = 3$ days.

Table 5. Refitted estimates for different assumed mean environmental persistence $1/\varepsilon$.^a At the upper search limit of the fit.

$1/\varepsilon$ (d)	$\mathcal{R}_0$	s_0	ϕ_W	$\mathcal{R}_{\text{eff}}(0)$	R^2
1	3.81	0.716	0.95	2.73	0.9879
2	5.06	0.666	0.95	3.37	0.9890
3 (baseline)	6.21	0.643	0.95	3.99	0.9895
5	8.32	0.624	0.95	5.19	0.9900
7	10.25	0.616	0.95	6.32	0.9901
14	15.00 ^a	0.616	0.94	9.24	0.9901

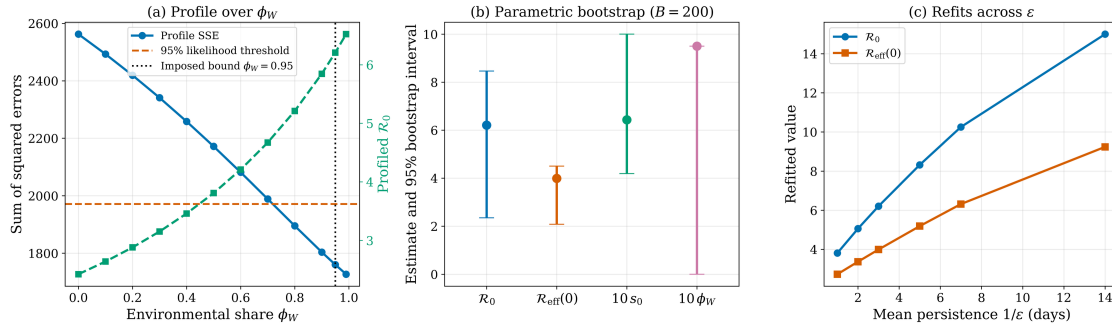


Fig. 3. Identifiability and uncertainty. (a) Profile sum of squared errors (left axis) and profiled $\mathcal{R}_0$ (right axis) against the environmental share ϕ_W ; the dashed line is the 95% likelihood threshold under independent Gaussian errors. (b) Point estimates with 95% parametric-bootstrap intervals. (c) $\mathcal{R}_0$ and $\mathcal{R}_{eff}(0)$ refitted for different assumed mean environmental persistence $1/\epsilon$.

7.3. Out-of-sample prediction

To test predictive ability within the outbreak, the model was fitted to days 0–20 only (Fig. 4). The training fit gives $\mathcal{R}_0 = 6.49$, $s_0 = 0.616$ and $\phi_W = 0.95$, with training $R^2 = 0.966$. For the held-out days 21–28 it predicts cumulative cases with a root-mean-square error of 10.8 cases and a mean absolute percentage error of 4.1%, and it predicts 199.3 cases by day 28 against 201 observed. This is a check on one outbreak only. It does not replace validation against independent outbreaks.

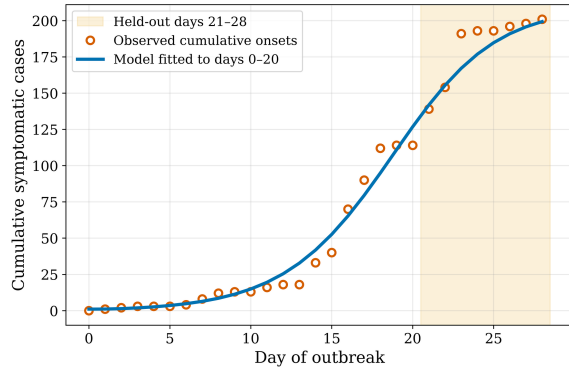


Fig. 4. Out-of-sample prediction. The model was fitted to cumulative onsets on days 0–20 and used to predict days 21–28 (shaded).

7.4. Outbreak dynamics and equilibria

Over 60 days the symptomatic class peaks at 31.2 cases on day 20.7. Environmental contamination peaks later, at 88.7 units on day 23.3, because virus persists after shedders recover. The hospital census peaks at 0.124 on day 23.1, that is, an expected 0.12 occupied beds in a population of 492 children. By day 60 the model gives 221 cumulative symptomatic cases (45% of pupils), and the susceptible pool has fallen to 13.6. The long-run endemic equilibrium of the renewing population is $S^* = 79.3$, $E^* = 0.284$, $A^* = 0.711$, $I^* = 0.331$, $H^* = 1.48 \times 10^{-3}$, $R^* = 411.3$ and $W^* = 1.10$. In that state most of the population is immune at any time, and a small reservoir of infection is maintained by waning immunity.

7.5. Accuracy of the MHAM solution

Fig. 5 compares the third-order MHAM solution with RK45 for every compartment. The curves are indistinguishable at plotting scale. The maximum absolute error over all compartments and times is 1.30×10^{-3} individuals, and the largest per-compartment relative error, $\max_t |u_{MHAM} - u_{RK45}| / \max_t |u_{RK45}|$, is 1.47×10^{-5} ($1.47 \times 10^{-3}\%$), attained in E . Table 6 lists values at selected times. At day 20, near the symptomatic peak, I differs from the benchmark by 1.1×10^{-4} cases.

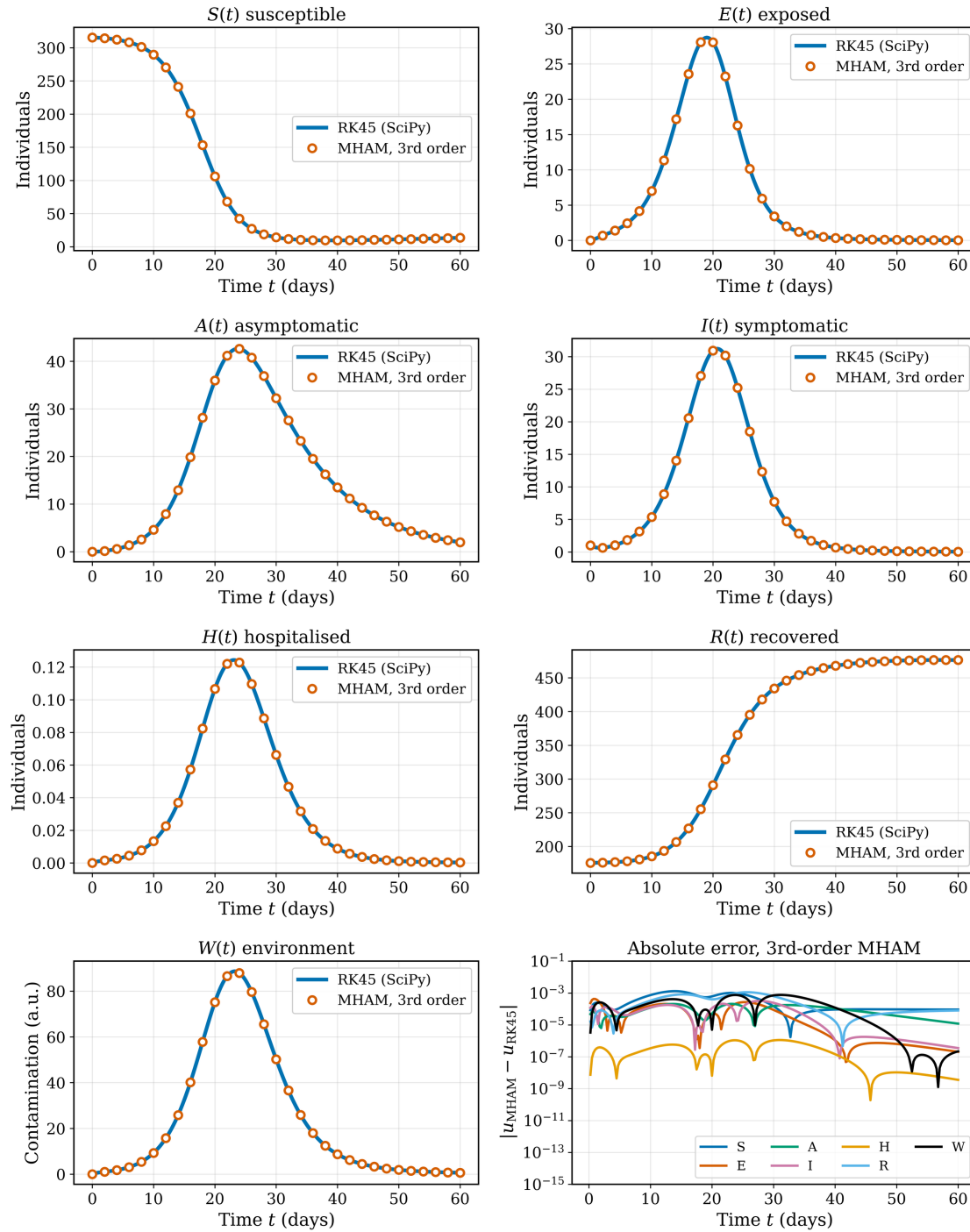


Fig. 5. Third-order MHAM ($\Delta t = 0.25$ d, $\hbar = -1$) and RK45 for each compartment of the calibrated model on $[0,60]$ days; the last panel shows the absolute error of each compartment.

Table 6. Third-order MHAM and RK45 values of S , I and W at selected times ($\Delta t = 0.25$ d, $\hbar = -1$).

t (d)	Compartment	MHAM	RK45	$ \Delta $
10	S	289.6129	289.6122	7.36×10^{-4}
	I	5.3597	5.3598	1.25×10^{-4}
	W	9.2713	9.2716	2.40×10^{-4}
20	S	106.1471	106.1465	6.15×10^{-4}
	I	30.9199	30.9200	1.11×10^{-4}
	W	75.1642	75.1642	4.95×10^{-6}
30	S	14.2628	14.2626	1.48×10^{-4}
	I	7.7182	7.7179	2.41×10^{-4}
	W	50.2096	50.2089	7.08×10^{-4}
45	S	10.2208	10.2209	9.57×10^{-5}
	I	0.2648	0.2648	1.77×10^{-6}
	W	3.8020	3.8019	1.85×10^{-5}
60	S	13.5940	13.5941	9.16×10^{-5}
	I	0.0420	0.0420	3.50×10^{-7}
	W	0.6079	0.6079	2.19×10^{-7}

Table 7 and Fig. 6 show how the error falls with the truncation order M . With $\Delta t = 0.25$ days, the maximum relative error falls from 6.25% at first order to 0.078% at second order, $1.47 \times 10^{-3}\%$ at third, $8.9 \times 10^{-5}\%$ at fourth and $1.2 \times 10^{-7}\%$ at sixth order. This is roughly one to two orders of

magnitude per additional term. Reducing the step to $\Delta t = 0.1$ days lowers the third-order error to $8.3 \times 10^{-5}\%$. These accuracies are demonstrated only for the calibrated parameters on $[0,60]$ days and for the stated steps; they are not claimed for other parameter regimes or horizons.

Table 7. Maximum absolute error (individuals) and maximum relative error (%) of MHAM ($\hbar = -1$) against RK45 on $[0,60]$ days.

Order M	Max. abs. $\Delta t = 0.25$ d	Max. rel. (%) $\Delta t = 0.25$ d	Max. abs. $\Delta t = 0.10$ d	Max. rel. (%) $\Delta t = 0.10$ d
1	1.17×10^1	6.25	4.69	2.48
2	1.65×10^{-1}	7.83×10^{-2}	2.70×10^{-2}	1.29×10^{-2}
3	1.30×10^{-3}	1.47×10^{-3}	8.52×10^{-5}	8.29×10^{-5}
4	2.55×10^{-5}	8.89×10^{-5}	5.89×10^{-7}	2.05×10^{-6}
5	1.04×10^{-6}	3.59×10^{-6}	1.01×10^{-8}	3.36×10^{-8}
6	6.00×10^{-8}	1.19×10^{-7}	2.57×10^{-10}	4.40×10^{-10}

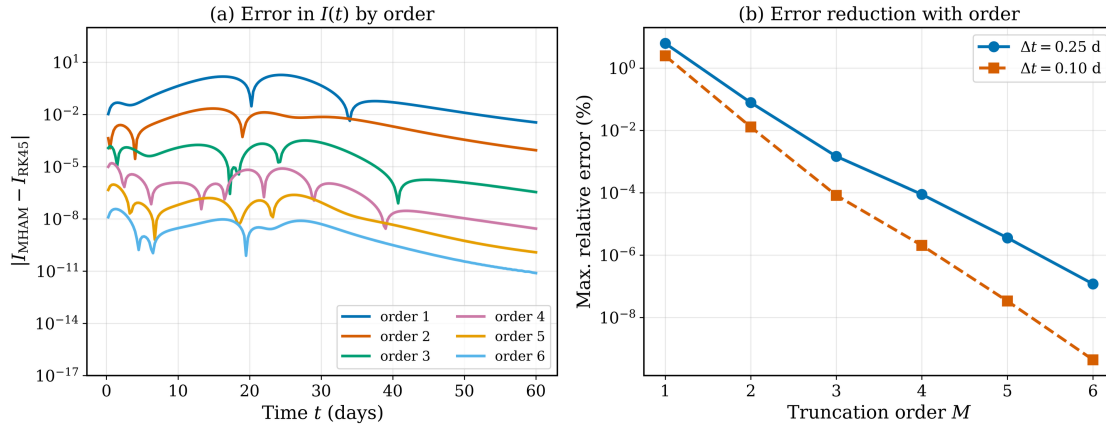


Fig. 6. Error reduction with increasing order. (a) Absolute error in $I(t)$ for orders 1–6 ($\Delta t = 0.25$ d). (b) Maximum relative error against order for two stage lengths.

7.6. Convergence-control parameter

Fig. 7a plots $I(30)$ computed by MHAM against $\hbar$ for orders 2–4. The curves form a plateau around $\hbar = -1$ that widens with order and meets the benchmark value $I(30) = 7.718$. Outside this plateau the truncated series is inaccurate, as Theorem 7 predicts. The averaged residual (Fig. 7b) is minimised at $\hbar^* = -0.9998$, -0.9981 and

-0.9952 for orders 2, 3 and 4. At third order, $\hbar^*$ lowers the residual from 1.620×10^{-8} (at $\hbar = -1$) to 1.544×10^{-8} and the maximum relative error from $1.47 \times 10^{-3}\%$ to $1.40 \times 10^{-3}\%$. For this system, tuning $\hbar$ therefore gives only a marginal gain over the homotopy perturbation choice $\hbar = -1$. The main accuracy lever is the stage length.

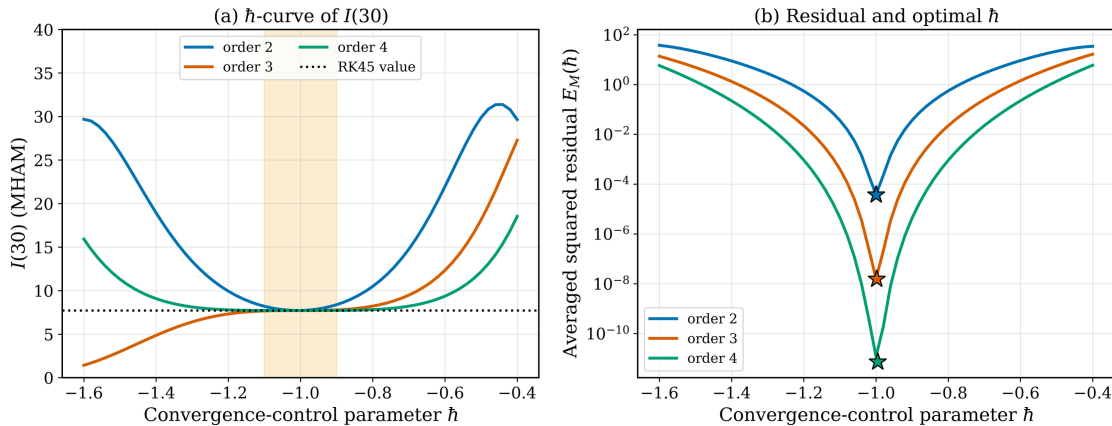


Fig. 7. (a) $\hbar$ -curves of $I(30)$ for MHAM orders 2–4; the shaded band marks $-1.1 \leq \hbar \leq -0.9$ and the dotted line is the RK45 value. (b) Averaged squared residual $E_M(\hbar)$; stars mark the optimal $\hbar^*$.

7.7. Global sensitivity of $\mathcal{R}_0$

Over the $\pm 25\%$ hypercube, $\mathcal{R}_0$ has mean 6.62 and a 95% range of 3.21–12.19; every sample exceeds one. The PRCCs (Fig. 8) identify three groups of parameters. The first group determines the size of the susceptible pool, Λ (+0.902) and μ (−0.901), through $S_0 = \Lambda/\mu$. The second is the environmental pathway: ε (−0.894), β_W (+0.892) and ξ_I (+0.868). The third is the duration of symptomatic shedding, γ_I (−0.882). The asymptomatic fraction p

has a moderate negative effect (−0.507), because asymptomatic carriers shed far less. The direct rate β (+0.109), ξ_A (+0.163) and γ_A (−0.199) are weak. The PRCCs for κ , η and σ are negligible, and those for κ ($p = 0.19$) and η ($p = 0.27$) are not significant. The discharge rate γ_H does not enter $\mathcal{R}_0$, so its assumed value does not affect the threshold.

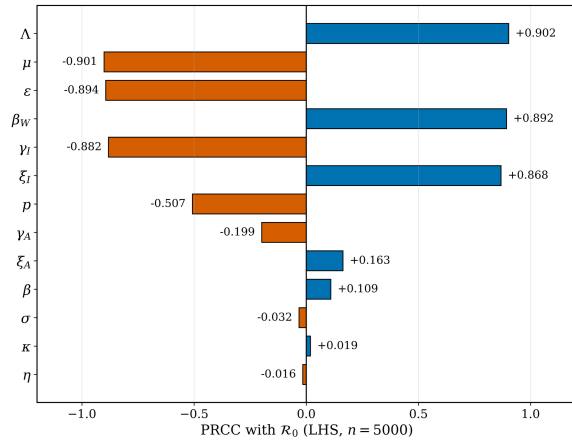


Fig. 8. PRCC of $\mathcal{R}_0$ from Latin hypercube sampling ($n = 5000$, each parameter varied by $\pm 25\%$).

7.8. Sensitivity of peak and cumulative cases

Fig. 9 extends the sensitivity analysis from $\mathcal{R}_0$ to three outbreak outputs. Over the $\pm 25\%$ hypercube the symptomatic peak ranges from 13.8 to 53.0 cases

(95% range), its timing from day 16.0 to day 31.1, and cumulative symptomatic cases by day 60 from 186 to 246. The peak size is governed mainly by the symptomatic recovery rate γ_I (-0.960), the environmental parameters β_W ($+0.892$), ξ_I ($+0.879$) and ϵ (-0.766), and the asymptomatic fraction p (-0.856). The timing of the peak depends on the same parameters with opposite signs, and additionally on the latent rate σ (-0.725). Cumulative cases are dominated by p (-0.971), because only symptomatic cases are counted. ϵ (-0.649), β_W ($+0.648$), γ_I (-0.607) and ξ_I ($+0.600$) also matter. For all three outputs the PRCCs of κ , η and γ_H are not significant at the 1% level, and γ_A is at most 0.08 in magnitude. ω is not significant for the peak size or timing and is weak ($+0.159$) for cumulative cases. The ranking agrees with that for $\mathcal{R}_0$. Because ϕ_W was fixed at its bound, however, these results describe the model as calibrated. They do not independently confirm that environmental transmission dominates.

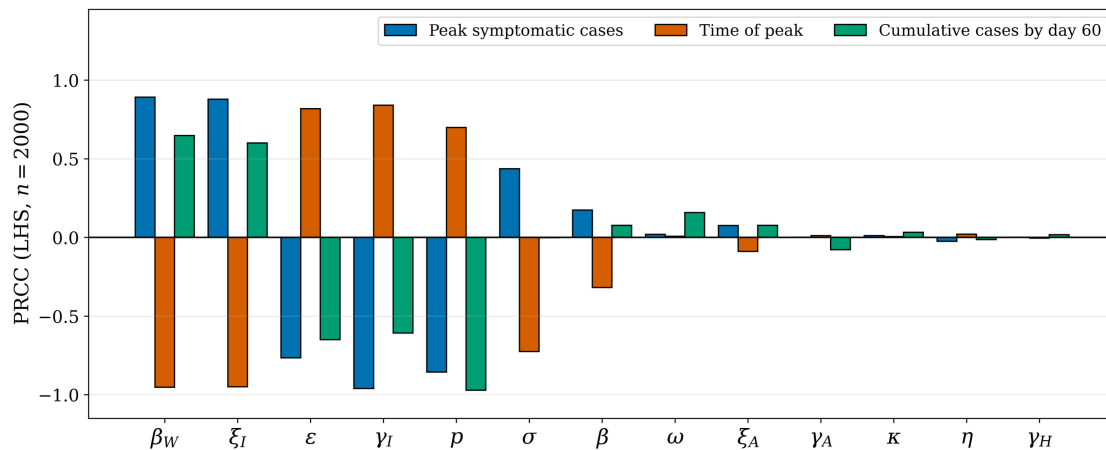


Fig. 9. PRCC of peak symptomatic cases, time of peak and cumulative symptomatic cases by day 60 (LHS, $n = 2000$, each parameter varied by $\pm 25\%$).

7.9. Hygiene and cleaning interventions

Fig. 10 shows cumulative symptomatic cases by day 60 when hygiene reduces both β and β_W by a fraction and cleaning multiplies the environmental decay rate ϵ . Without intervention the model gives 221 cases. A 30% reduction in transmission alone gives 209 cases ($\mathcal{R}_{\text{eff}} = 2.79$), and doubling ϵ alone gives 185 cases ($\mathcal{R}_{\text{eff}} = 2.10$). Combining the two gives 99 cases ($\mathcal{R}_{\text{eff}} = 1.47$). A 50% reduction together with three-fold faster virus removal brings $\mathcal{R}_{\text{eff}}$ to 0.73 and limits the outbreak to 3.4 cases. To reach $\mathcal{R}_{\text{eff}} < 1$ by cleaning alone, ϵ must be multiplied by 4.74. This multiplier drops to 3.09 with

30% hygiene improvement and to 2.11 with 50%. In this model the two measures act multiplicatively on the environmental pathway, so modest gains in each are more effective than a large gain in one.

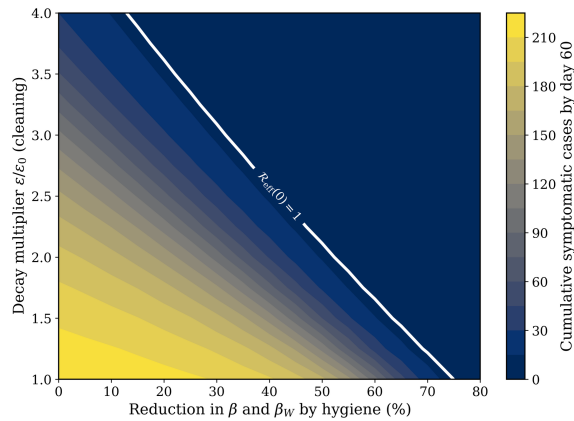


Fig. 10. Cumulative symptomatic cases by day 60 as a function of hygiene-driven reduction in β and β_W and of cleaning-driven increase in ϵ . The white curve is $\mathcal{R}_{\text{eff}}(0) = 1$.

7.10. Comparison with other methods and recent studies

Table 8 compares MHAM qualitatively with other approaches used for norovirus and related epidemic models. Table 9 places the present model among recent norovirus studies.

Table 8. Literature-based comparison of solution approaches.

Method	Output	Convergence control	Convergence result used
MHAM (this work)	Piecewise closed-form polynomials	Yes ($\hbar$), with $\hbar$ -curve and residual	Banach-based analyticity radius; explicit step condition (Theorem 7)
HPM / ADM	Series (MHAM with $\hbar = -1$)	No	Special case of Theorem 7
Laplace–Adomian decomposition [20, 21]	Series via Laplace transform and Adomian polynomials	No	Existence/uniqueness by fixed-point arguments
NSFD scheme [2]	Discrete numerical values	Not applicable	Positivity and boundedness of the scheme
Adams–Bashforth / Newton polynomial [12]	Discrete numerical values	Not applicable	Schauder and Banach fixed-point theorems

Table 9. Comparison with recent (2023–2026) norovirus modelling studies.

Study	Model features	Analysis / solution	Data
Wang et al. 2023 [11]	SEIAQRW, human and water/food routes	ODE fitting; mean $\mathcal{R}_{\text{eff}}$ 8.92 (person-to-person), 2.19 (water/food)	23 school outbreaks, Jiangsu
Raezah et al. 2023 [12]	Fractional (ABC), vaccination, asymptomatic carriers	Adams–Bashforth–Newton scheme	Simulation only
Song et al. 2024 [14]	Stochastic, Lévy noise, water and food contamination, vaccination	Extinction/persistence threshold	Simulation only
Ndendya et al. 2025 [17]	Vaccination, treatment, education	Graph-theoretic $\mathcal{R}_0$, Routh–Hurwitz	CDC CaliciNet 2023–24, least squares
Saleem et al. 2025 [2]	Fractional SEIR	Lyapunov, GL-NSFD scheme	Assumed parameters
Alqudah et al. 2025 [16]	Discrete fractional SEAIRF with environment	Picard–Lindelöf, neural network	Simulated data
Gogovi et al. 2026 [5]	SAIR with environment, waning immunity, reinfection	Lyapunov, optimal control, cost-effectiveness	Literature parameters
Present study	SEAIHRS–W: asymptomatic, hospitalisation, waning, environment	Lyapunov; MHAM with convergence proof; RK45 validation; LHS-PRCC	Derbyshire 2001 school outbreak, $R^2 = 0.990$

8. Discussion

The model brings asymptomatic infection, hospitalisation, waning immunity and environmental persistence into one framework that can be analysed rigorously. The threshold $\mathcal{R}_0$ splits into four pathway terms, which makes it clear which interventions act on which route. The global results give the usual dichotomy when immunity is lifelong on the timescale of interest: extinction for $\mathcal{R}_0 < 1$ and a globally attracting endemic state for $\mathcal{R}_0 > 1$. With waning immunity only local stability of the endemic state was shown, and the damped oscillations in that case point to recurrent seasons in a renewing population.

The fitted reproduction number should be interpreted with care. Three results together show that the Derbyshire data constrain some quantities well and others hardly at all. The initial effective reproduction number is fairly well determined (3.99; 95% interval 2.08–4.50), and the model reproduces the size and timing of the outbreak, including out of sample. The split between direct and environmental transmission is not identified: the least-squares estimate sits on the imposed bound, and the bootstrap interval covers the whole admissible range. Finally, $\mathcal{R}_0$ itself depends on the assumed environmental persistence, rising from 3.81 to 15 as $1/\varepsilon$ increases from 1 to 14 days, with no change in fit quality. The data are therefore compatible with environment-dominated transmission, which is plausible for an outbreak in which vomiting in classrooms was documented. They do not establish it, and the environmental conclusions of this paper should be read as conditional on the calibration assumptions. Separating the two routes would require additional information, such as environmental sampling, timing of vomiting events relative to cases, or data from several outbreaks with different cleaning regimes.

On the numerical side, the multistage formulation removes the main weakness of single-interval series solutions: their loss of accuracy far from the expansion point. Restarting the series every quarter day keeps each stage inside the proven convergence radius, and third order already gives errors of about 10^{-3} individuals. The explicit third-order formulas show how each parameter enters the local solution, which a purely numerical integrator does not. Because the optimal $\hbar$ differs from -1 by less than 0.5% and improves accuracy only marginally, the advantage of MHAM here lies mainly in its stage-wise convergence guarantee and residual diagnostics rather than in large gains from tuning $\hbar$.

9. Limitations

The following limitations should be kept in mind when interpreting the results.

1. *Single-outbreak calibration.* All estimates come from one school outbreak of 201 cases. The out-of-sample check (Section 7) tests prediction within this outbreak only, and the parameter estimates may not generalise to other schools, other settings or other norovirus strains. Validation against independent outbreaks is the most important next step.
2. *Weak identifiability of the transmission routes.* The environmental share ϕ_W lies on its imposed bound, and its bootstrap interval spans 0–0.95. The data do not separate direct from environmental transmission, and statements about their relative importance are conditional on the calibration.
3. *Dependence on the assumed environmental decay rate.* ε is assumed, and $\mathcal{R}_0$ changes by a factor of about four across plausible values of $1/\varepsilon$ (Table 5), while $\mathcal{R}_{\text{eff}}(0)$ changes by a factor of about three. The natural exit rate and the hospital discharge rate are also assumed; the former has negligible effect over an outbreak, and the latter affects none of the fitted quantities.
4. *Poor fit to daily cases.* The model reproduces cumulative cases well ($R^2 = 0.990$) but daily cases poorly ($R^2 = 0.374$). The daily series is shaped by weekend reporting, the school closure, two weekend cleanings and case exclusion, none of which is represented in the autonomous model.
5. *Homogeneous mixing.* The school is treated as a single well-mixed population. A real school has classrooms, teachers, age groups and distinct contact networks, and vomiting events expose mainly the children in one room. A classroom-structured or network model would be more realistic, although substantially more complicated to analyse.
6. *Global stability with waning immunity.* Global stability of the endemic equilibrium is proven only for $\omega = 0$. For $\omega > 0$ only local stability is shown, and only for the calibrated parameters.

7. *Scope of the numerical accuracy claims.* The MHAM error levels hold for the calibrated parameters, the 60-day window and the stated step sizes only.

10. Public health significance

The sensitivity and scenario results agree that, in a school outbreak of this kind, virus removal from the environment and shortening the period in which symptomatic children shed in shared spaces are the dominant levers. This supports current outbreak guidance, which advises excluding children until 48 hours after symptoms resolve, prompt cleaning of vomiting incidents, and chlorine-based disinfection of shared surfaces. The model adds a quantitative point: hygiene and cleaning reinforce each other, and a 50% cut in transmission combined with three-fold faster virus removal was sufficient in the model to prevent the outbreak. Because the relative weight of the environmental route is not identified from these data, the effect of cleaning in particular may be overstated, whereas the benefit of hand hygiene and exclusion, which act on both routes, is more robust. Hospitalisation in a primary-school population is rare, and the model's hospital burden was a small fraction of one bed. The hospitalisation component becomes relevant when the framework is applied to care homes and older populations. There, norovirus vaccines now in clinical development could be added as a vaccinated class, and their value compared with environmental control.

11. Conclusion

We developed and analysed an SEAIHRS–W model of norovirus with asymptomatic shedding, hospitalisation, waning immunity and an environmental reservoir. We derived its reproduction number and endemic equilibrium explicitly, and proved global stability of the disease-free equilibrium for $\mathcal{R}_0 < 1$ and of the endemic equilibrium in the lifelong-immunity limit. The model was solved by a multistage homotopy analysis method with explicit third-order formulas and a Banach-based convergence theorem that gives a computable step condition. Calibrated to a school outbreak ($R^2 = 0.990$ for cumulative cases), the model gives $\mathcal{R}_{\text{eff}}(0) = 3.99$ (95% interval 2.08–4.50) and $\mathcal{R}_0 = 6.21$ (2.35–8.46), and it predicts the final size

$$V^{-1}e_1 = \left(\frac{1}{k_E}, \frac{p\sigma}{k_E k_A}, \frac{(1-p)\sigma}{k_E k_I}, \frac{1}{\varepsilon} \left(\frac{\xi_{AP}\sigma}{k_E k_A} + \frac{\xi_I(1-p)\sigma}{k_E k_I} \right) \right)^T. \quad (\text{A.1})$$

The next-generation matrix $K = FV^{-1} = e_1(b^T V^{-1})$ has a single non-zero row, so its only non-zero eigenvalue is its $(1,1)$ entry:

of the outbreak out of sample. The third-order MHAM solution agrees with RK45 to a maximum relative error of $1.47 \times 10^{-3}\%$ over 60 days. Within the calibrated model, environmental transmission, virus decay and the duration of symptomatic shedding dominate the threshold and the size and timing of the peak. The data from one outbreak cannot, however, separate environmental from direct transmission, and $\mathcal{R}_0$ depends strongly on the assumed virus persistence. Calibration to several outbreaks, ideally with environmental sampling, is needed before these conclusions can be generalised.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgment

The authors thank Academy of Maritime Education and Training (AMET) University for research facilities and support.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Data availability

The outbreak data are publicly available in the *outbreaks* data package (dataset *norovirus derbyshire 2001 school*, originally described by O'Neill and Marks). The complete Python code needed to reproduce every number, table and figure in this paper is provided as a supplementary archive: the model and MHAM solver, the calibration, bootstrap, profile and out-of-sample analyses, the RK45 benchmark and the sensitivity analyses, together with the data file and a README. The authors will deposit the same archive in a public repository (Zenodo) with a permanent DOI on acceptance.

Appendix A. Next-generation matrix derivation

Because V in (11) is lower triangular, its inverse is obtained by forward substitution. Its first column is

$$\mathcal{R}_0 = b^T V^{-1} e_1 = S_0 \left[\beta \kappa \frac{p\sigma}{k_E k_A} + \beta \frac{(1-p)\sigma}{k_E k_I} + \frac{\beta_W}{\varepsilon} \left(\frac{\xi_A p\sigma}{k_E k_A} + \frac{\xi_I (1-p)\sigma}{k_E k_I} \right) \right], \quad (\text{A.2})$$

which rearranges to (12). The left eigenvector of $V^{-1}F$ for the eigenvalue $\mathcal{R}_0$ is b^T , which is the

weight used in the Lyapunov function of Theorem 5. The Jacobian of the transmitting block at the DFE is

$$F - V = \begin{pmatrix} -k_E & \beta \kappa S_0 & \beta S_0 & \beta_W S_0 \\ p\sigma & -k_A & 0 & 0 \\ (1-p)\sigma & 0 & -k_I & 0 \\ 0 & \xi_A & \xi_I & -\varepsilon \end{pmatrix}, \quad (\text{A.3})$$

and expanding $\det(\lambda I - (F - V))$ gives the coefficients a_1 – a_4 of (21).

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