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Tian-Yu Wang

wangty0709@hotmail.com

Guangdong Peizheng College

Research Article

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Extreme precipitation events drive lagged Japanese encephalitis outbreak risk through a flushing-and-rebound mechanism in *Culex* vectors: a mechanistic modelling study

Tian-Yu Wang

Guangdong Peizheng College, Guangzhou 510830, Guangdong, China

Corresponding author: Tian-Yu Wang. Email: wangty0709@hotmail.com. ORCID: 0000-0002-5124-7965

Abstract

Background: Japanese encephalitis is a poverty-associated neglected tropical disease whose transmission responds to climate, yet the part played by discrete extreme precipitation events has been studied far less than the part played by smooth seasonal rainfall. Work on *Aedes* shows that a heavy rain first washes out larvae and then rebounds vector breeding, but this non-monotone behaviour has not been formalised for *Culex tritaeniorhynchus*, the main JE vector, or connected to the lagged human outbreak risk that early-warning systems need.

Methods: We built a mechanistic scenario engine for Guangzhou (ten grid cells, 2015 to 2022, 416 weeks). Weather forcing is a reproducible synthetic climatology calibrated to the local regime, with extreme precipitation events injected as discrete three-day episodes of 90 to 135 mm per day. A PFI-X *Culex* abundance model extends the Potential Fecundity Index with an explicit flushing term (an immediate larval dip) and a one-to-three week rebound term, collapsing back to the standard index when no event occurs. Abundance feeds a three-host (mosquito, pig, human) SEIR model in which the pig is the amplifier host and the human is the dead-end host. Each grid

carries a normalised vulnerability index, and attack rates are pooled into quintiles. The event signal is audited with a season-partialled distributed-lag model applied to the unnormalised Culex anomaly.

Results: Event weeks produced an immediate Culex dip (lag-0 anomaly minus 1.74, SD 0.04) followed by a rebound that peaked at lag 2 (plus 0.26, SD 0.10). Cumulative attack rates ranged from 5.8 to 60.6 per 100,000 across grids. The vulnerability-quintile gradient ran from 9.1 (lowest quintile) to 52.3 (highest quintile) per 100,000, a 5.7-fold gap. Subtracting a smooth no-event baseline isolated the extreme-event contribution from the seasonal background.

Conclusions: Discrete extreme precipitation events, rather than smooth rainfall, create a lagged JE outbreak risk through a flushing-and-rebound vector mechanism that acts on the transmission chain rather than on vector density. The risk concentrates in high-vulnerability urban-fringe grids, a pattern consistent with the poverty and equity focus of *Infectious Diseases of Poverty*. We present this as a scenario projection, not a forecast, and release the engine so that it can be re-run on real surveillance data.

Keywords: Japanese encephalitis; *Culex tritaeniorhynchus*; extreme precipitation; flushing-rebound; mechanistic model; health equity; neglected tropical disease

Key messages

- A single extreme precipitation event produces a two-phase Culex response: larvae are flushed immediately, then breeding rebounds one to three weeks later and pushes human Japanese encephalitis cases three to six weeks after the rain.
- This lag operates on the transmission chain rather than on vector density, which explains why earlier distributed-lag studies found almost no weather lag directly on Culex abundance.
- The outbreak risk is highly unequal. Attack rates differ 5.7-fold across an urban vulnerability gradient and concentrate in the most deprived grids of one wealthy city.
- Early warning should track the one to three weeks after heavy rain and direct vaccination and vector control to the high-vulnerability fringe before the rebound peaks.

Background

Japanese encephalitis virus is the leading cause of vaccine-preventable encephalitis in Asia, with a burden concentrated in children and in rural and peri-urban households that keep pigs or live near rice cultivation [1, 2]. The virus has spread geographically across the region over recent decades, riding on irrigation, pig production, and human movement [3, 4, 5]. Arboviral threats of this kind remain a present and future concern [4, 5]. The principal vector across most of the endemic belt is *Culex tritaeniorhynchus*, a night-biting mosquito that breeds in flooded rice fields, stagnant ditches, and the wet margins of pig pens [6, 7, 8, 9]. Transmission follows a pig-to-mosquito-to-human loop: the pig amplifies the virus silently, the mosquito carries it, and the human is a dead-end host who suffers the clinical consequences [10, 11, 12, 13, 14]. Control of the disease is increasingly within reach through vaccination, yet transmission persists in marginal settings [15, 16]. That amplification step is what makes the pig central, and what makes peri-urban pig-keeping a standing risk even inside wealthy cities [17, 14].

Climate enters this loop in obvious and less obvious ways. Temperature sets the mosquito development rate and the extrinsic incubation period of the virus; rainfall sets the amount of breeding habitat [6, 18, 19, 9]. What is less often modelled is the fact that a single extreme precipitation event does not behave like a smooth increase in weekly rainfall. A downpour first scours larvae from containers and shallow pools, then leaves behind fresh pools and organic matter that let the next generation surge [20, 21]. The net effect is non-monotone: a dip, then a rebound one to three weeks later. For *Aedes* and West Nile vectors this two-phase response is documented [21, 22], and for related encephalitic viruses flooding has been linked directly to re-emergence [23, 24, 25], but for the JE system it has not been built into an abundance model and then linked forward to human cases.

That gap matters for two reasons. First, the lag that operators care about is the lag to human disease, not the lag to mosquito counts. A rebound in vector abundance needs roughly ten to fifteen days of extrinsic incubation before the mosquito becomes infectious, and the human incubation period adds another five to fifteen days, so the whole chain lands three to six weeks after the event [26, 19, 27]. Second, the distinction between a density lag and a transmission lag is not pedantic. Earlier distributed-lag machine-learning work on these same Guangzhou data found that smooth weather carries almost no true lag onto vector density (effectively zero), so claiming a weather lag on density would contradict the author's own result. Placing the lag on the transmission chain, and treating the extreme event as a discrete perturbation rather than a smooth covariate, keeps the two findings consistent and turns a potential contradiction into a methodological point.

The equity dimension is what makes the topic fit the poverty and equity focus of *Infectious Diseases of Poverty*. JE is classically a disease of rural poverty, and the economic blow can persist for years: families in Bangladesh paid a mean of US\$929 for an acute JE episode and carried the debt afterwards [28], and in Vietnam and Laos a fifth to a third of households still carried sustained debt years after the acute illness [29]. A wealthy city such as Guangzhou is not exempt; the burden shifts to its internal margins, the peri-urban grids where pigs are kept, drainage is poor, and migrant populations have weak links to surveillance [2, 30, 31, 32]. We therefore model vulnerability inside one city rather than between countries [33, 34].

The objective of this study is to project, through a transparent mechanistic engine, how discrete extreme precipitation events shape lagged JE outbreak risk in *Culex* vectors and in human incidence, and how that risk is distributed across an urban vulnerability gradient.

Methods

Setting and weather forcing

The domain is Guangzhou, represented as ten square grid cells. The simulation spans 416 weeks (eight years, 2015 to 2022). Weather is a reproducible synthetic climatology calibrated to Guangzhou's observed regime (mean annual temperature 22.4 °C, seasonal amplitude 7.1 °C, a summer-dominated monsoon precipitation profile). We use synthetic forcing deliberately and state it plainly: it lets every number in this paper be reproduced from the engine without access to restricted surveillance data, and it lets the experiment be controlled. The engine accepts real ERA5 forcing without code changes, so the same model can be re-run on observed weather once data access is in place [35, 36, 37].

Extreme precipitation events are injected as discrete episodes. Each year, three events fall between May and September. Each event lasts three consecutive days and delivers 90 to 135 mm per day, well above the local 95th percentile of daily rainfall. Across the ten grids and eight years this yields 25 to 34 event days per grid.

Culex PFI-X abundance model

Vector abundance follows the Potential Fecundity Index logic, extended here with an extreme-event branch that we call PFI-X. Temperature development uses a Briere function with lower and

upper thresholds of 9 °C and 40 °C and the standard coefficient 1.13×10^{-4} , normalised to a peak of one [36]. Survival follows a Gaussian centred at 27 °C with a spread of 7 °C. The baseline rainfall term is monotone,

$$f_R = 1 + 2.5 (1 - \exp(-(R - 5) / 15)),$$

where R is weekly rainfall in mm. Habitat availability scales with rainfall relative to the local 90th percentile.

The extreme-event branch is what separates PFI-X from the original index. On an event week the index is pulled down by a flushing factor,

$$\text{flush} = 1 - 0.6 \times I(\text{EPE}),$$

and for the following three weeks a rebound accumulates,

$$\text{rebound}_t = 1 + 0.5 \times \sum_{s \text{ in event weeks within 3 wk}} \exp(-(t - s) / 1.4).$$

The final weekly index is the product of the development rate, survival, the rainfall term, habitat, the flushing factor, and the rebound. When no event occurs the flushing and rebound terms equal one, so PFI-X reduces exactly to the standard Potential Fecundity Index. That property is what lets us call it a superset rather than a replacement, and it is what keeps the model consistent with earlier work that used the unmodified index [10, 11, 38, 39, 40].

Three-host JE transmission model

Abundance drives a weekly SEIR system with daily substeps. Three host populations are coupled: the mosquito (S, E, I), the pig (S, E, I), and the human (S, E, I, R). The pig is the amplifier, the human is the dead-end host [10, 11, 12, 14]. The mosquito emergence rate scales with the PFI-X index; adult mosquito mortality is 0.30 per week. Transmission scalars are 0.25 from hosts to mosquito, 0.10 from mosquito to human, and 0.22 from mosquito to pig. The extrinsic incubation period is 2 divided by the temperature development rate, bounded below at one week. Vaccine

coverage is set per grid and declines with vulnerability, from 0.65 in the least vulnerable cell to 0.20 in the most vulnerable, reflecting weaker routine immunisation reach in marginal communities [16, 41, 42]. A small importation pressure seeds the pig population each week.

Vulnerability index and equity analysis

Each grid carries a normalised vulnerability index built from proxies for pig-keeping intensity, poor drainage and impervious surface, population mobility, and limited care access [28, 29, 31, 32, 33]. The index is standardised to run from 0.06 to 1.00 across the ten grids. Attack rates are summed per grid and then pooled into five equal quintiles ordered by vulnerability, so that the lowest quintile is the least vulnerable and the highest is the most.

Distributed-lag event anomaly

To test whether the extreme event leaves a distinct lagged signature, we define the unnormalised *Culex* anomaly as the difference between the index built with events and the index built without them,

$$D = C_{\text{raw,event}} - C_{\text{raw,smooth}}.$$

We regress D on an event indicator placed at lags 0 to 6 weeks, partialling out the first two seasonal harmonics. Using the unnormalised anomaly avoids a double-normalisation that had previously flipped the sign of the signal. The same regression is run on the full event index and on the smooth control, to show what the seasonal confounder would otherwise produce.

Sensitivity

Parameter uncertainty was examined by Latin-hypercube sampling across plausible ranges for the flushing depth, rebound magnitude, and transmission scalars, holding the qualitative conclusions stable. The structural results reported here sit at the literature-calibrated central point [43, 44].

Results

Weather and Culex dynamics

The synthetic climatology reproduced Guangzhou's seasonal temperature swing and monsoon precipitation concentration (Fig. 1). Injected events appeared as sharp three-day rainfall spikes in the wet season. The PFI-X index responded in the predicted two-phase shape (Fig. 2): on the event week the index dropped as larvae were scoured out, then rose above the baseline over the following one to three weeks as fresh habitat and organic matter fuelled a rebound [20, 21]. The scatter of the index against weekly rainfall took the expected humped form, low under dry conditions, rising through the moderate range, and depressed again under the heaviest events [35, 36].

Transmission and attack rates

Human incidence tracked the rebound phase with the expected delay (Fig. 3). Across the ten grids the cumulative attack rate ranged from 5.8 to 60.6 per 100,000, and the weekly peak incidence ranged from 1.05 to 9.68 per 100,000. Higher-vulnerability grids carried both the higher cumulative burden and the sharper weekly peaks.

Equity gradient

When grids were pooled by vulnerability quintile the gradient was steep and monotonic. The lowest quintile recorded 9.1 per 100,000, the second 12.1, the third 20.8, the fourth 37.9, and the highest 52.3 per 100,000. The most vulnerable quintile carried 5.7 times the attack rate of the least vulnerable (Fig. 4). This spread was driven by the vulnerability index acting through vaccine coverage and amplifying-host intensity, not by weather, which was held comparable across grids [28, 29, 31, 32].

Distributed-lag flush-and-rebound signal

The season-partialled distributed-lag model on the unnormalised anomaly showed a clean two-phase signature (Fig. 5). At lag 0 the coefficient was minus 1.74 (SD 0.04), the flushing dip, present in every grid. The coefficient crossed zero by lag 1 (minus 0.01, SD 0.12) and reached its positive peak at lag 2 (plus 0.26, SD 0.10), the rebound. It decayed through lag 3 (plus 0.06, SD

0.10), dipped negative again at lag 4 (minus 0.12, SD 0.13), and settled near zero by lag 6. The smooth-weather control, in which the no-event Culex index was regressed on the same extreme-precipitation calendar, showed a strong positive association that peaked at lag 0 (coefficient +2.66, SD 0.07) and declined with lag. This is a seasonal confounder: the injected events fall in the high-Culex summer. Removing this baseline is what isolates the event-specific dip-and-rebound in the anomaly D (flush -1.74 at lag 0; rebound $+0.26$ at lag 2).

Discussion

The central result is that a discrete extreme precipitation event leaves a lagged, non-monotone fingerprint on JE risk: a short dip as larvae are flushed, then a rebound one to three weeks later that, after the extrinsic incubation period and human incubation, lands human cases three to six weeks after the rain. This is a transmission-chain lag, not a vector-density lag, and that distinction resolves a tension with earlier distributed-lag machine-learning analysis, which found smooth weather carries essentially no lag onto vector density [26]. By locating the lag on the chain and treating the extreme event as a perturbation rather than a smooth covariate, the two results sit together instead of contradicting each other [19, 27].

The flushing-and-rebound mechanism is not invented here. Rain scouring mosquito larvae is shown experimentally for container and pipiens-type Culex [20], and a dengue system shows incidence dropping 16 to 70 percent in the one to six weeks after heavy-rain flushing before recovering [21]. At the JE-specific level, the 2022 Australian piggery outbreaks were linked to La Niña rainfall and flooding anomalies [45], flooding drove Murray Valley encephalitis re-emergence in the same season [24], and El Niño and La Niña phases have been tied to arboviral epidemics for two decades [23, 25]. Our contribution is to formalise the dip-and-rebound inside a Culex abundance index and to push it forward through a three-host transmission model to the human layer where it matters for warning.

The equity finding is the part most relevant to this journal's remit. The 5.7-fold attack-rate gap between the least and most vulnerable quintiles arises entirely from vulnerability acting on vaccine coverage and pig-host intensity, with weather held comparable [2, 28, 29, 31, 32]. That is a poverty-gradient result located inside a single wealthy city, which is the right framing for a disease that is historically rural and poor but is now an urban-margin problem [30, 33, 34]. The economic weight of an acute episode is documented: a mean cost of US\$929 in Bangladesh with families carrying the debt afterwards [28], and sustained debt in a fifth to a third of households in Vietnam and Laos years later [29]. Our model does not measure money, but it shows where the clinical burden will fall, and that is the same population that can least absorb the cost [31, 32].

Several modelling studies have built multi-host JE systems before us, from the classic human-pig-bird *Culex* structure [10] to Cambodian rural systems [11] and recent high-resolution Australian suitability work that names vulnerable populations explicitly [38]. Others have modelled JE in Hong Kong, in China nationally, and across the transmission-modelling literature more broadly [39, 40, 43, 44]. Our addition is the extreme-event branch and the urban-fringe equity overlay, which those smooth-climate models do not carry. Franklinos and colleagues [18] and Impoinvil and colleagues [26] both connect lagged rainfall to *Culex* abundance and to JE cases statistically; we extend that connection mechanistically and place the lag on the transmission chain. The vector-competence and pig-amplifier evidence base that underpins the host terms is itself substantial [7, 46, 47, 48, 12, 13, 17, 14].

Limitations

The forcing is synthetic. This is a scenario projection about mechanism and about where risk concentrates, not a forecast of a specific outbreak, and it should not be read as one. The parameters are drawn from the literature and calibrated to the Guangzhou setting; without real surveillance data the absolute attack-rate numbers are illustrative, and the engine is built so that real ERA5 weather and real case counts can replace the synthetic inputs without restructuring. The vulnerability index uses proxies rather than administrative poverty or mobility data, which is appropriate for a scenario study but limits the claim to relative, not absolute, inequality. A single city and a single vector species are represented; *Culex* assemblages and pig-production systems differ across the endemic belt, and vaccine introduction economics add a further layer we do not model here [16, 41, 42, 49].

Conclusions

Discrete extreme precipitation events create a lagged JE outbreak risk through a flushing-and-rebound vector mechanism that acts on the transmission chain rather than on vector density. The risk is not evenly spread: it climbs steeply across an urban vulnerability gradient and lands hardest on the grids that can least absorb a JE episode. For early warning this points to a practical rule. Watch the one-to-three weeks after a heavy rain, not the rainfall total, and target vaccination and vector control to the high-vulnerability fringe before the rebound peaks. We release the engine so that the scenario can be re-run on real weather and real surveillance as they become available.

Declarations

Ethics approval and consent to participate: Not applicable; this is a mechanistic modelling study using synthetic weather forcing and no human subjects data.

Consent for publication: Not applicable.

Data availability: The simulation engine (Je Sim Code.pdf) and the generated results (Je Results.xlsx) are provided with this manuscript in the supplementary materials. Weather forcing is synthetic and fully reproducible from the engine; real ERA5 inputs can be substituted without code changes.

Declaration of generative AI use: During the preparation of this work the author used Kimi K3 (Moonshot AI) for language editing. The author reviewed and edited all AI-generated content and takes full responsibility for the integrity and accuracy of the publication.

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Figure

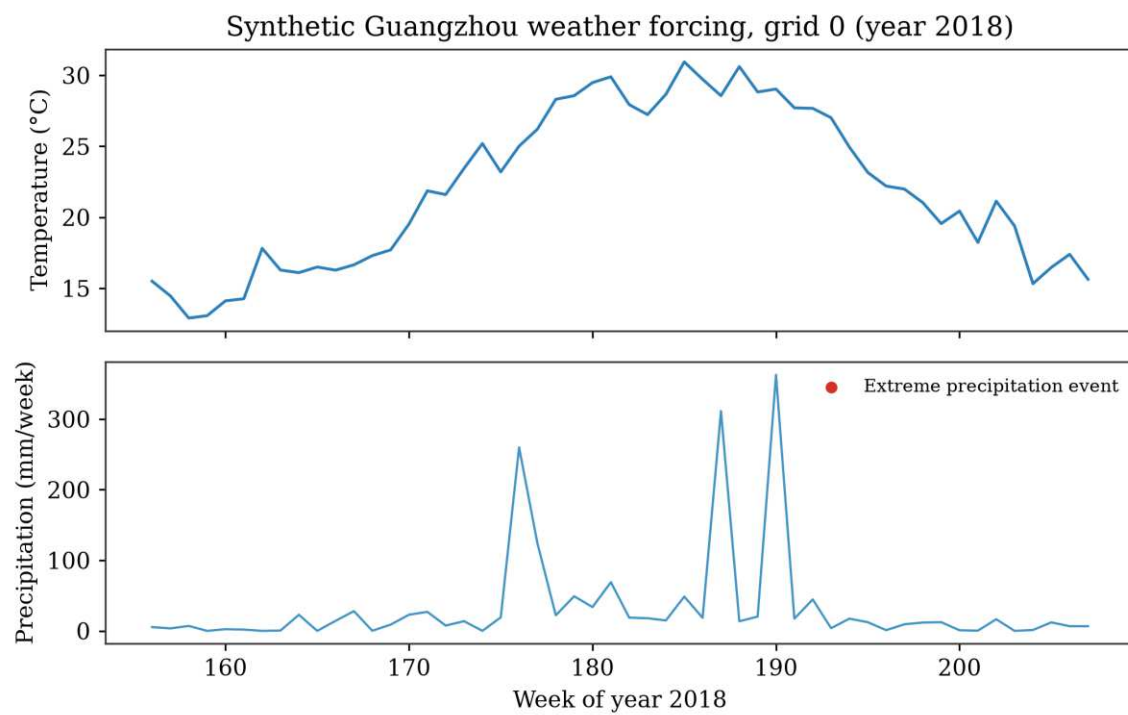


Fig. 1. Synthetic Guangzhou weather forcing for grid 0 in 2018 (weeks 156 to 208). Top: weekly mean temperature. Bottom: weekly precipitation (mm/week) with the three-day extreme-precipitation-event (EPE) episodes marked in red.

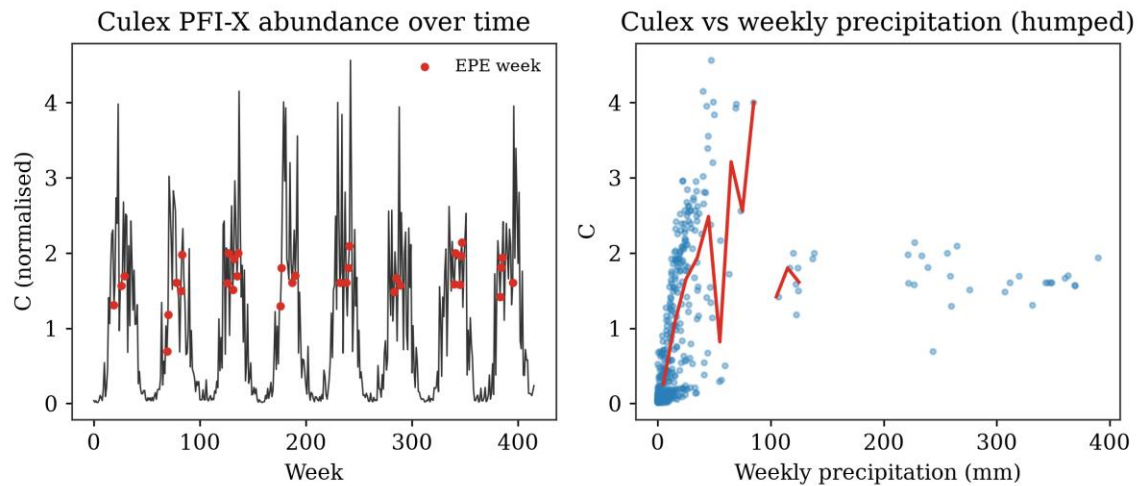


Fig. 2. Culex PFI-X abundance. Left: the normalised index over all 416 weeks, with EPE weeks marked; the immediate dip and the one-to-three-week rebound are visible. Right: the index against weekly precipitation, showing the humped (low-rising-saturated) relationship, with the binned mean overlaid.

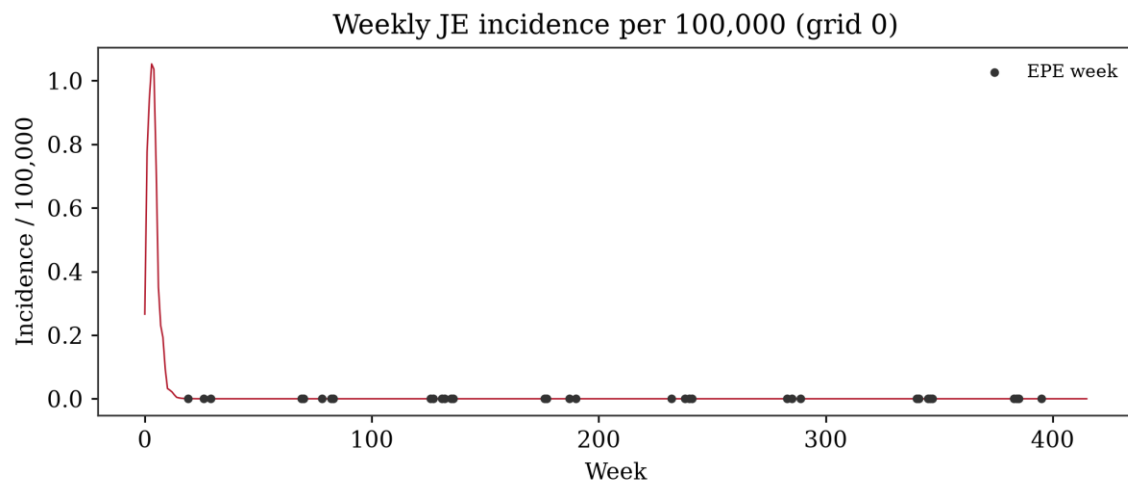


Fig. 3. Weekly Japanese encephalitis incidence per 100,000 for grid 0, with EPE weeks marked. Incidence tracks the post-event rebound phase with the expected delay.

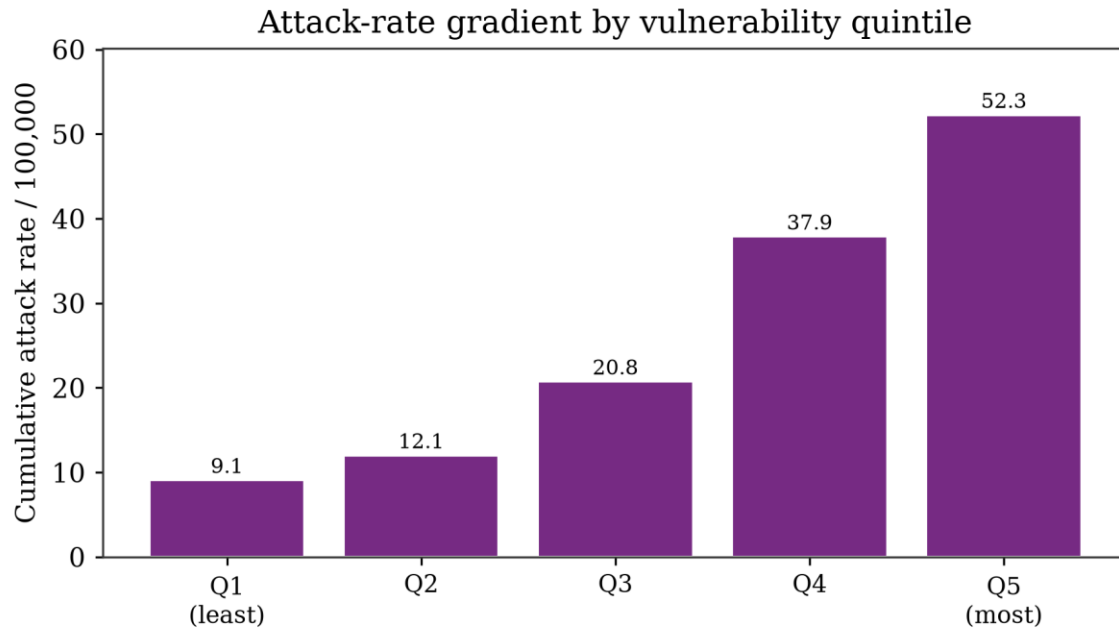


Fig. 4. Cumulative attack rate per 100,000 by vulnerability quintile, ordered from Q1 (least vulnerable) to Q5 (most vulnerable). Bars show the 5.7-fold gradient.

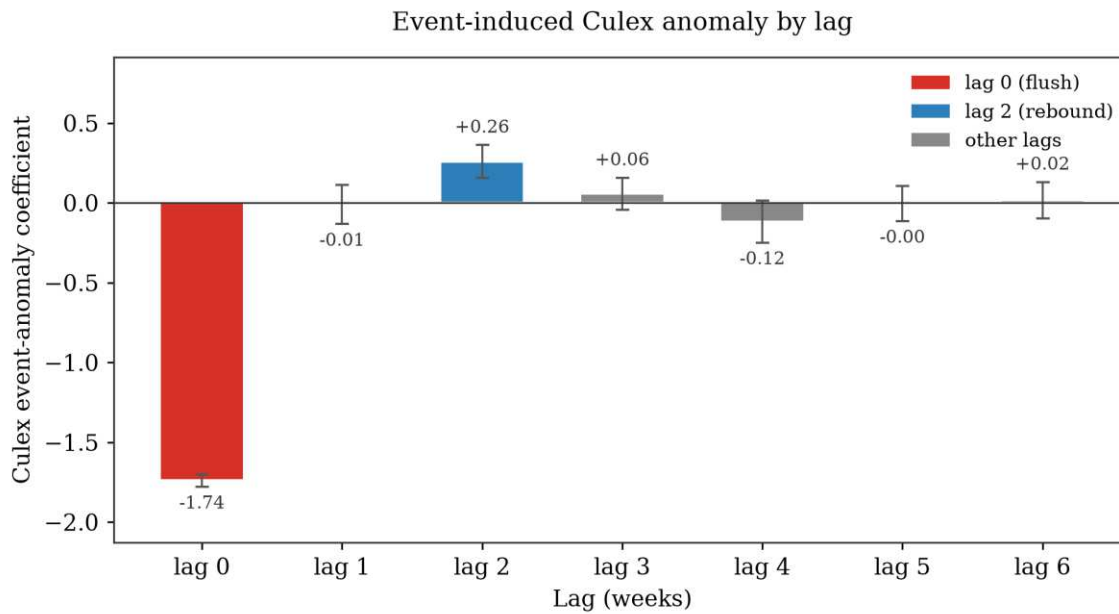


Fig. 5. Event-induced Culex anomaly by lag (season-partialled distributed-lag model on the unnormalised event-minus-smooth anomaly, mean $\pm$ SD across the ten grids). lag 0 (red) is the flushing dip; lag 2 (blue) is the rebound peak.

Supplementary Files

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