PROCEEDINGS B

royalsocietypublishing.org/journal/rspb

Evidence synthesis

Cite this article: de Wit MM, Dimas Martins A, Delecroix C, Heesterbeek H, ten Bosch QA. 2024 Mechanistic models for West Nile virus transmission: a systematic review of features, aims and parametrization. *Proc. R. Soc. B* **291**: 20232432.
https://doi.org/10.1098/rspb.2023.2432

Received: 30 October 2023
Accepted: 14 February 2024

Subject Category:
Ecology

Subject Areas:
health and disease and epidemiology, computational biology, ecology

Keywords:
vector-borne diseases, West Nile virus, mathematical modelling, Ross–Macdonald, epidemiology

Author for correspondence:
Mariken M. de Wit
e-mail: mariken.dewit@wur.nl

[†]Joint first authors.

Electronic supplementary material is available online at https://doi.org/10.6084/m9.figshare.c.7093224.

THE ROYAL SOCIETY
PUBLISHING

Mechanistic models for West Nile virus transmission: a systematic review of features, aims and parametrization

Mariken M. de Wit[1,†], Afonso Dimas Martins[3,†], Clara Delecroix[1,2,†], Hans Heesterbeek[3] and Quirine A. ten Bosch[1]

[1]Quantitative Veterinary Epidemiology, and [2]Department of Environmental Sciences, Wageningen University and Research, Wageningen, The Netherlands
[3]Department of Population Health Sciences, Faculty of Veterinary Medicine, University of Utrecht, Utrecht, The Netherlands

MMdW, 0000-0002-5758-636X; ADM, 0000-0003-2220-5851; CD, 0000-0001-5583-6903; HH, 0000-0001-8537-6418; QAtB, 0000-0002-5227-1406

Mathematical models within the Ross–Macdonald framework increasingly play a role in our understanding of vector-borne disease dynamics and as tools for assessing scenarios to respond to emerging threats. These threats are typically characterized by a high degree of heterogeneity, introducing a range of possible complexities in models and challenges to maintain the link with empirical evidence. We systematically identified and analysed a total of 77 published papers presenting compartmental West Nile virus (WNV) models that use parameter values derived from empirical studies. Using a set of 15 criteria, we measured the dissimilarity compared with the Ross–Macdonald framework. We also retrieved the purpose and type of models and traced the empirical sources of their parameters. Our review highlights the increasing refinements in WNV models. Models for prediction included the highest number of refinements. We found uneven distributions of refinements and of evidence for parameter values. We identified several challenges in parametrizing such increasingly complex models. For parameters common to most models, we also synthesize the empirical evidence for their values and ranges. The study highlights the potential to improve the quality of WNV models and their applicability for policy by establishing closer collaboration between mathematical modelling and empirical work.

1. Introduction

West Nile virus (WNV) is a mosquito-borne pathogen that has caused outbreaks worldwide. While the virus originated from the African continent, human cases have occurred all across the globe. In the past few decades, large outbreaks have mostly been detected in Europe and North America [1]. The virus is transmitted between mosquitoes and birds, but the pathogen can spill over to humans and horses. Although most human cases are asymptomatic, it can cause a variety of symptoms ranging from fever to encephalitis in the most severe cases [2]. There is currently no available vaccine or specific treatment against WNV infections in humans [2]. Thus, current prevention measures mostly consist of mosquito control campaigns [3].

WNV can be transmitted to a wide range of species [4], making its dynamics complex. WNV is primarily transmitted through the bites of infected mosquitoes, with birds serving as the main reservoir host [5]. *Culex* species are the main vector that amplifies WNV as it feeds preferably on competent bird species [6]. This maintenance process in mosquito and bird populations is characterized by a high degree of heterogeneity. Within a single *Culex* species, there can be variations in biting behaviour and transmission efficiency based on factors such as age, sex, time of the year and infection status [7,8]. Occasionally, there is spillover to dead-end species like humans and horses, which are incapable of further transmitting it [9].

Mathematical models aim to capture the diversity of species and processes involved in WNV transmission dynamics. These models capture dynamical processes involved in WNV transmission and thereby contribute to our knowledge of WNV and help to predict the course of future outbreaks. Mathematical models can help understand the transmission and establishment of WNV, as well as which factors contribute to this, by estimating metrics such as the basic reproduction number R_0, the force of infection, or human infection risk [10]. Models are also used to inform policy, for example by estimating the current levels of WNV transmission based on surveillance data, by determining the risk of future outbreaks or by estimating the effect of control interventions. This can be done on both short- and long-term scales and coarse and fine spatial scales and could specifically include predictions under change scenarios. Two recent reviews focusing on the United States have studied the use of WNV models for guiding interventions and made recommendations on how to make models more useful for policy [10]. Both reviews recommended the development of models on finer spatial scales as that corresponds better to the scale at which vector control interventions are implemented. Keyel *et al*. [11] also recommended a closer alignment of model outputs with required information for decision-making.

The foundation for many modelling efforts to understand WNV dynamics was laid by Ronald Ross' work on malaria, extended by George Macdonald [12,13]. Although several Ross–Macdonald-type models have been developed, they typically centre around the concept of a basic reproduction number and include a simplified description of the transmission cycle [14]: (i) an infectious mosquito passes the pathogen to a host upon a bite, (ii) the pathogen infects the host, multiplies and reaches high densities in the host bloodstream, (iii) the pathogen is passed to a mosquito upon a bite on the infectious host, and (iv) the pathogen infects the mosquito and multiplies so that the virus reaches sufficiently high concentrations in the salivary glands to be transmitted upon a bite. Some common assumptions of these models are: bites are evenly distributed among the host population, transmission only happens between vectors and hosts, and the incubation period and biting rate are constant over time, as is the mosquito-to-host ratio. This is implemented in the framework of compartmental models in which the host and vector populations are divided into classes based on their infection status (for example, the so-called SIR models, where individuals are assumed to be in one of three epidemiological states: susceptible, infectious or recovered). For a more thorough explanation of the use of mathematical models in epidemiology, we refer to Heesterbeek *et al*. [15]. Within this context, models have been expanded to answer present-day challenges posed by mosquito-borne diseases. Examples of adaptations include temperature dependence, vertical transmission and multiple host species. The Ross–Macdonald framework is used to represent the full enzootic transmission cycle and is therefore commonly used in mechanistic transmission models. It is important to note that many other modelling approaches exist that do not follow this framework such as statistical or hybrid models.

To study how the Ross–Macdonald approach has changed since its development, Reiner *et al*. [16] compiled an exhaustive list of mathematical models for mosquito-transmitted diseases, spanning over 388 models published between 1970 and 2010. Of these, 31 had WNV as the pathogen of study. The authors argue that many models developed over the years still bear a strong resemblance to the foundational Ross–Macdonald ideas and suggest that new theory could benefit from including concepts like heterogeneous mosquito biting, poorly mixed mosquito–host encounters, spatial heterogeneity and temporal variation in the transmission process.

More than a decade after the Reiner *et al*. review [16], both the geographical range of WNV has expanded, as has the number and complexity of models aimed specifically at the WNV system. These developments raise important questions about the availability and suitability of empirical studies necessary to inform these more complex models and their application in new locations. Adapting previously developed models to new environmental and ecological contexts is essential, and adding heterogeneity in models can substantially improve their quality and applicability [17,18]. This can lead to models becoming more parameter rich, while at the same time, the values for parameters common to the most basic mechanistic descriptions of the system can differ between regions and contexts where they were previously determined and the new situations where the model will be applied. With an increasing use of models focused on understanding and predicting outbreaks to direct policy, these developments can have important consequences for decision-making in new areas or populations.

Building on the study by Reiner *et al*. [16], we first describe trends in assumptions of compartmental models, with a specific focus on WNV, extending the analysis with models published between 2010 and 2022. Additionally, we explore the purpose and type of WNV models, also in relation to their similarity to the basic Ross–Macdonald framework, to identify the conceptual developments that have been implemented in the last decade. We then provide insight into the evidence base of empirical studies that are cited to have informed the values of the parameters common to most of these models. Finally, we synthesize the challenges emerging from this.

2. Methods

(a) Literature search strategy

We searched the peer-reviewed literature for mechanistic models of WNV transmission on a population level (figure 1) that used parameter values based on data. Specifically, we were interested in compartmental models that modelled population dynamics over time with differential equations. The database search query combined terms related to 'WNV', 'mathematical' and 'model' (see electronic supplementary material, S1 for full details). This search was conducted in three databases: PubMed, Scopus and Web of Science.

This study focused on compartmental models of WNV; other pathogens and types of models were therefore excluded. Compartmental models were of particular interest, because these can use information from empirical studies through the use of biologically relevant

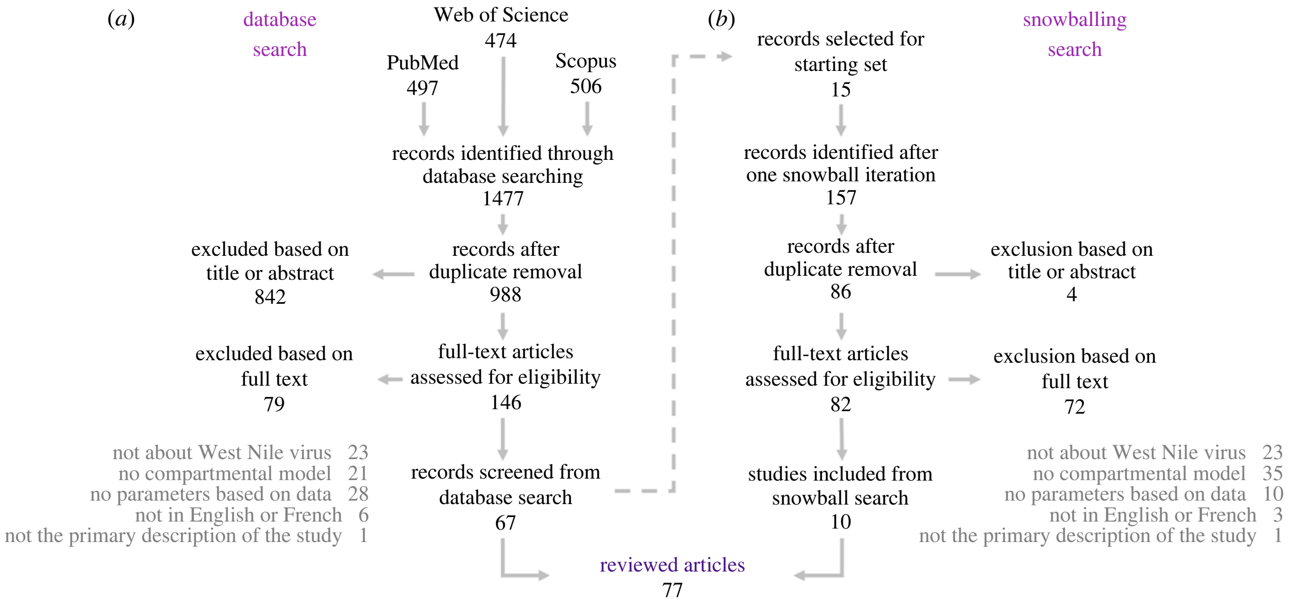

Figure 1. Complementing the systematic literature search with one iteration of snowballing search. (*a*) PRISMA diagram depicting inclusion and exclusion steps of the database searching. (*b*) Decision tree of the snowball searching.

parameter values, contrary to for example statistical models. Agent-based models of WNV were also excluded as they are still small in number and have been discussed elsewhere [19]. We aimed at characterizing empirical evidence used to parametrize WNV models. We therefore only considered studies citing other references to parametrize their models, or estimating their parameters based on data. Additionally, we only considered primary, peer-reviewed publications in English or French. The selection was performed in two rounds: first based on title/abstract and then based on full text. Both rounds of inclusion/exclusion were performed by two reviewers independently, and disagreements were discussed until a consensus was reached. A third author was involved in making the final decision when necessary.

To minimize the risk of missing relevant papers, we then used a forward snowballing approach [20], where we used the 15 most cited studies identified through the database search to add the papers that cited these. The same inclusion and exclusion process was applied in the systematic database search and in the snowball round. The final search was completed on 14 December 2023. No date restrictions were used. The full list of identified papers, selection steps and final list of included papers is presented in electronic supplementary material, S2.

(b) Model classification

For each publication, we retrieved the purpose and type of the model and classified the refinements compared with the basic Ross–Macdonald assumptions. We defined three categories for the purpose of models: understand, predict and control, as defined in Cecilia *et al*. [21]. Models were classified as ‘understand’ if they aimed at exploring the impact of various mechanisms on the transmission dynamics, as ‘predict’ if they were forecasting the evolution of WNV transmission in time and as ‘control’ if they were exploring the impact of intervention strategies. Additionally, we used the categories defined in Cecilia *et al*. [21] to classify the types of models: applied, theoretical and grey. Applied models describe a specific area and use data to calibrate or validate the model, theoretical models are generic models and do not use any data, and grey refers to models that do not fit into these categories.

We classified the refinements by calculating a dissimilarity index as proposed in Reiner *et al*. [16] to quantify the divergence of the models from the basic Ross–Macdonald (RM) assumptions. Even though this RM index is not specific to WNV, it has been investigated in other studies and thus provides a comparable measure of the number of refinements used in a model. For each publication, the model was classified based on 15 criteria (table 1). For each criterion, the model scored one if it is refined compared with the assumption of the basic Ross–Macdonald model and zero otherwise. The resulting sum of the scores for all criteria is a dissimilarity index, between 0 and 15, for each publication. When several models were described in a publication, we used the one having the most refinements. Each publication was read and classified by two authors. Disagreements were discussed until a consensus was reached, and a third author was involved in making the final decision when necessary.

(c) Empirical evidence for parameter values

To study the use of available evidence from empirical studies in model parametrization, we extracted references cited for parameter values in all our included studies and traced the empirical sources of these values. We focused on values for the six most common virus-specific parameters: extrinsic incubation period (EIP; the time the virus takes to incubate within the vector), intrinsic incubation period (the time the virus takes to incubate within the host), recovery rate (i.e. duration of infectious period), disease-induced mortality rate, transmission probability from vector to host and from host to vector. We extracted references provided for these parameter values from all studies and traced back to empirical studies underlying these values (e.g. in the case where the cited reference was not an empirical study but a modelling paper or a review). For each of the empirical studies, we counted the number of times it was used as underlying source for parameter values by studies included in this review. We distinguished two ways in which empirical studies were used as a source: direct citation (i.e. model paper cited the empirical source) and indirect citation (i.e. model paper cited another paper which cited the empirical source). Parameter extraction and identification of underlying empirical evidence was done by two authors independently for each study.

Table 1. Details on the dissimilarity index criteria.

index	criterion	Ross–Macdonald assumption	deviation from Ross–Macdonald assumption
1	aquatic stages of mosquito populations	not modelled	explicitly modelled, with at least one state variable
2	number of spatial locations	one without immigration	one with immigration, or more than one
3	number of mosquito taxa, genotypes or phenotypes	one	more than one
4	number of pathogen taxa, genotypes or phenotypes	one	more than one
5	number of vertebrate taxa, genotypes or phenotypes	one	more than one (including non-competent hosts)
6	mosquito mortality in the absence of control	constant *per capita* mortality rate	any refinement (temperature-dependent, humidity-dependent, etc.)
7	mosquito blood-feeding rate in the absence of control	constant *per capita* blood-feeding rate	any refinement (temperature dependent, dependent on host availability, etc.)
8	proportion of blood meals on hosts	feeding on other hosts was not included	any refinement (feeding on non-competent hosts, etc.)
9	pathogen latency in mosquitoes	not modelled	explicitly modelled as a state variable
10	waning immunity	not modelled	explicitly modelled
11	superinfections and co-infections	not modelled	explicitly modelled
12	distribution of blood meals among hosts	homogeneous distribution of blood meals among vertebrate hosts	heterogeneous distribution of blood meals among vertebrate hosts
13	mixing	well-mixed	not well-mixed, modelled using a contact network or individual-based model
14	transmission from mosquito to host	constant	any refinement (temperature-dependent, time-dependent, etc.)
15	transmission from host to mosquito	constant	any refinement (temperature-dependent, time-dependent, etc.)

3. Results

We identified a total of 77 papers that published compartmental WNV models using parameter values based on data [18,22–97]. The oldest paper included was published in 2001, and since then, the number of modelling papers published per year has increased over time ($R = 0.68$, p-value $= 9.4 \times 10^{-4}$, figure 2). The majority of studies included in our review were published in the past decade (66%), with 44% being published in the past 5 years.

(a) Classification

The dissimilarity index, calculated using 15 criteria (table 1), increased slightly over time ($R = 0.27$, p-value $= 0.016$), indicating that WNV compartmental models tended to increasingly deviate from the approaches of Ross and Macdonald (figure 3*a*). The mean number of refinements included was 3.1, and varied between 0 [37,52,59,72,77,78] and 9 [46,79,91]. We identified 64% of models ($n = 49$) aiming at understanding transmission, 27% ($n = 20$) aiming at evaluating control strategies and 9% ($n = 7$) aiming at predicting case numbers. Models aiming at predicting had a higher dissimilarity index on average (mean = 5.9, p-value $= 2.7 \times 10^{-7}$). Additionally, 66% of the models ($n = 51$) were classified as theoretical, 22% ($n = 17$) as applied and 12% ($n = 9$) as grey. Applied models had a higher dissimilarity index on average (mean = 4.6, p-value $= 2.2 \times 10^{-7}$). A variety of data types were used in applied and grey models, including demographic and environmental data such as mosquito abundance and temperature, as well as epidemiological data such as incidence in humans, serological data and prevalence in trapped mosquitoes.

The most common refinements of the Ross–Macdonald framework were including multiple spatial locations (or taking migration into account) (48%), including pathogen latency in mosquitoes (51%) and including multiple host species (44%) (figure 3*b*). Half of the models with multiple host species ($n = 17$, 50% of those with more than one host species) included non-competent hosts (humans $n = 17$, equids $n = 1$). Additional competent hosts were studied in 20 studies (59% of those with more than one host species studies), including up to eight different bird species. Contrarily, only 8% of models included more than one mosquito species, of which 83% included multiple *Culex* species, and one included *Aedes albopictus* in addition to the most commonly modelled *Culex pipiens*. The least common refinements were waning of immunity (3%), number of pathogen taxa

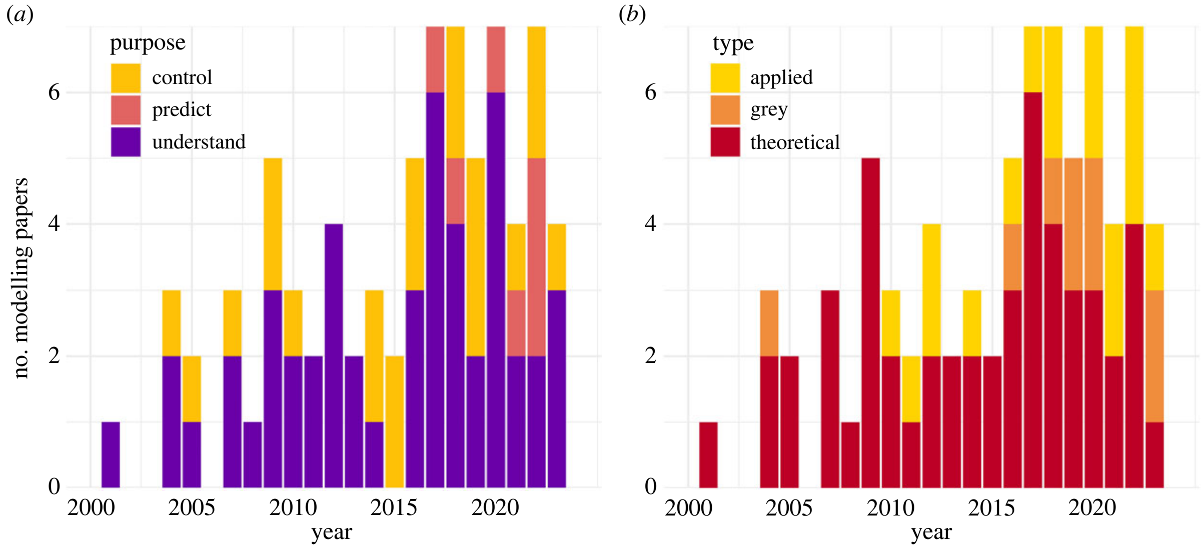

Figure 2. Frequency of modelling studies included in the review classified according to (*a*) their research purpose and (*b*) study type.

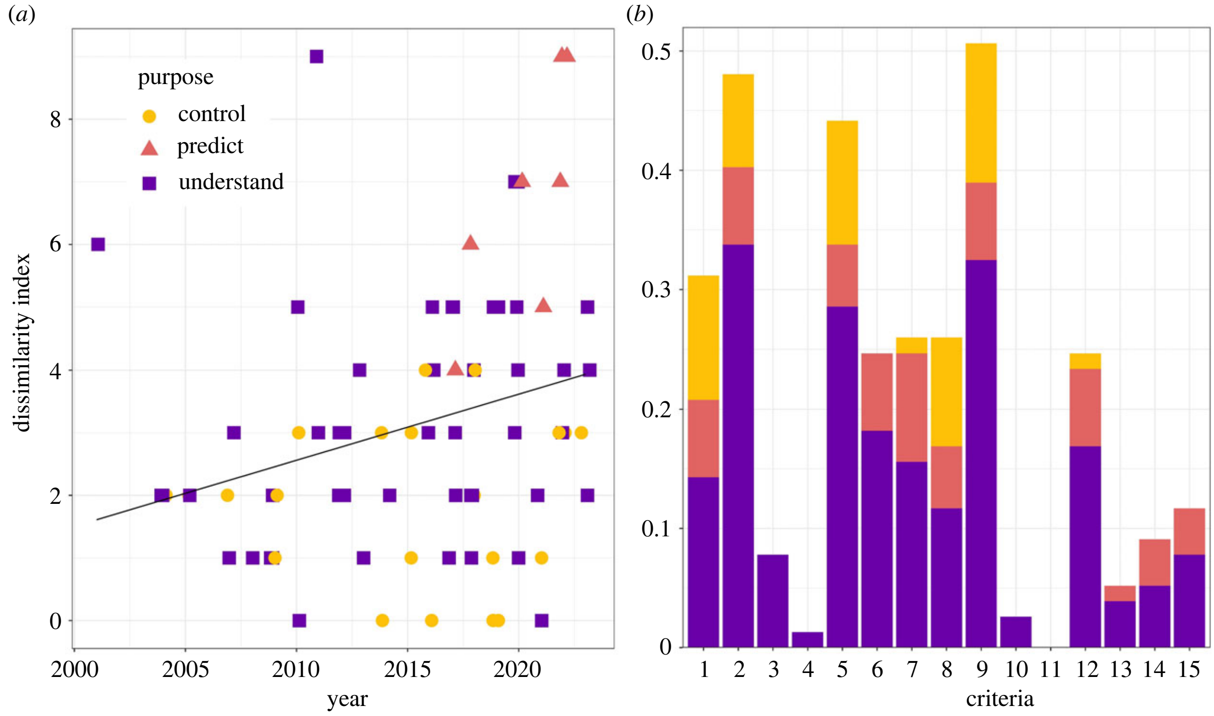

Figure 3. (*a*) Dissimilarity index of publications per year of publication. (*b*) Proportion of models deviating from criteria of the dissimilarity index.

(1%) and superinfection (0%). We found no study that explicitly modelled more than one WNV circulating strain. Likewise, the only study considering more than one pathogen included avian malaria in addition to WNV but did not explicitly model co- or super-infection [38]. All classification results per paper are presented in electronic supplementary material, S3.

(b) Empirical underpinnings of key model parameters

The reviewed body of literature resulted in a collection of infection and transmission parameters. The complete datasets on the parameters and their details can be found in the electronic supplementary material, S4. Here, we describe the body of evidence used by the authors of the studies to estimate key virus-specific parameters: the disease-induced death rate, the recovery rate, the intrinsic incubation period, the latency or extrinsic incubation period, the transmission probability from host to vector and the transmission probability from vector to host.

The number of unique empirical studies used as underlying source for parameters varied from 10 for the intrinsic incubation rate to 28 for the transmission probability from mosquito to host. Some papers cited these empirical studies directly, but most (62%) used other models or reviews as a reference (i.e. indirect citation, figure 4). A small number of papers were clearly used more frequently, while 51% of studies were only used once. This trend was especially strong for the recovery rate parameter, with one paper [98] used as a source in 73% of all citations to empirical studies. A comparison of the parameter values used in the models for the most cited empirical sources is provided in electronic supplementary material, S6.

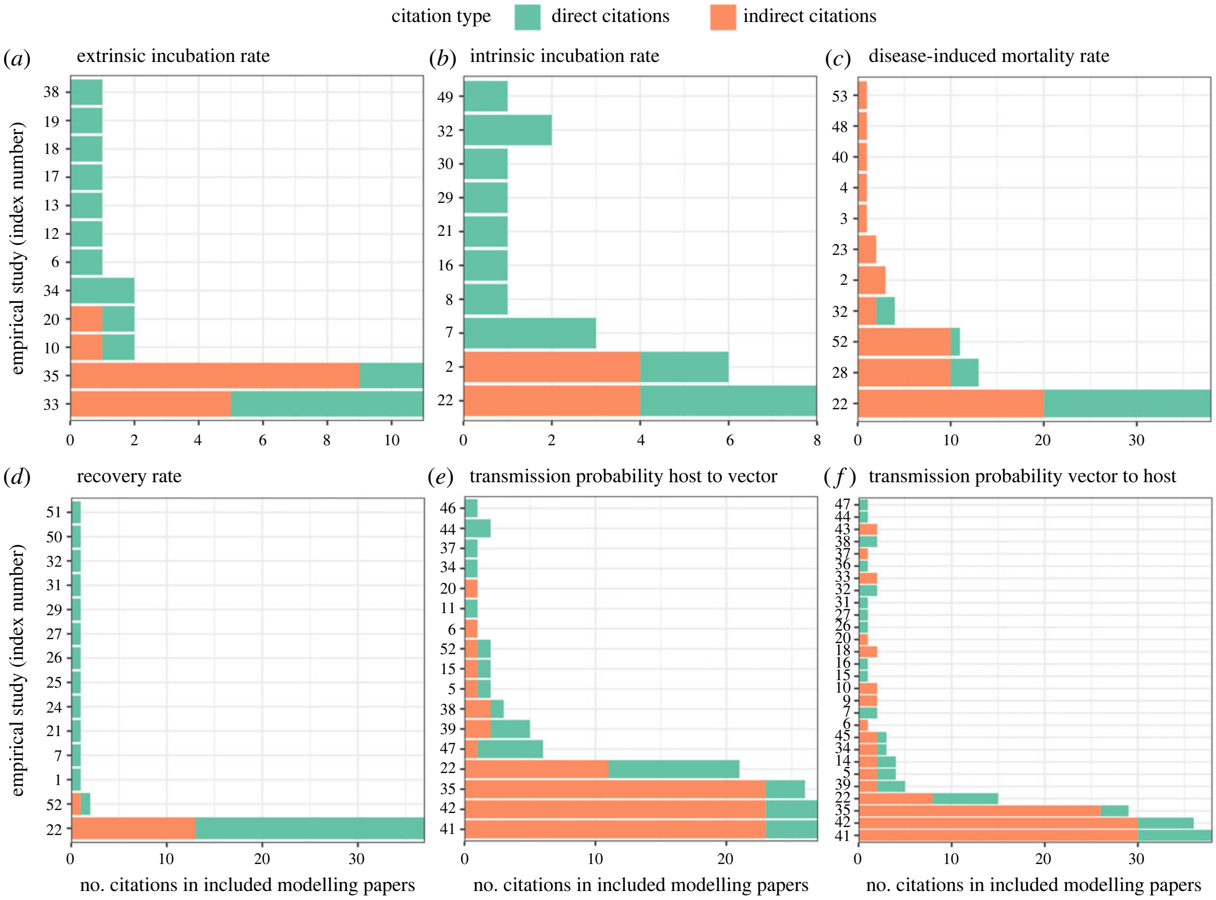

Figure 4. (*a*–*f*) Number of direct (green) and indirect (orange) citations per experimental study used as source for each key model parameter. Full list of original sources (index numbers on *y*-axis) and model studies that cited them can be found in the electronic supplementary material, S5.

4. Discussion

We identified 77 papers presenting compartmental WNV models that make use of empirical studies to inform their parameter values. The number of refinements to the Ross–Macdonald framework increased over time. Models aiming at predicting transmission and those including data from a specific area included the highest number of refinements. Compared with Reiner *et al.* [16], models included here addressed more refinements on average. We found an uneven distribution in which model refinements are addressed, with spatial structure and/or migration and pathogen latency in mosquitoes being addressed in about half the models, while others, such as superinfection, were never addressed. Most papers referred to other modelling papers and/or reviews rather than the underlying empirical studies for their parameter values. A small number of empirical studies were highly influential through indirect citations and frequently used as a source of parameter values, whereas half of the empirical studies used were only referred to once.

Not all refinements of the RM index were addressed to the same extent in the included studies. One of the main recommendations put forward in Reiner *et al.* [16] related to the incorporation of host heterogeneity, which we encountered in several WNV models. This was achieved through the inclusion of multiple host species with host-specific infection parameters in 34 models (44%) (e.g. [25,74]) and/or species-specific biting preferences in 19 models (25%) (e.g. [18,52]) sometimes extended with changes in biting preferences over time in three models (4%) [33,34,49]. Like Reiner *et al.*, we observed that heterogeneity through imperfect mixing of hosts and mosquitoes and any spatial heterogeneity was still rarely included. This may be due to lack of available data or a desire to keep inferences at a higher level of aggregation. For some refinements, it may also matter what the time scale is that is being considered for the application of the model. For example, if a study is focused on emergence and the very onset of an outbreak, refinements such as waning immunity or strain replacement may not be major influences on that time scale. Additionally, the RM-index is not specific to WNV and some of the refinements are less relevant for WNV. For instance, waning immunity (addressed in two studies) may be considered irrelevant given that immunity loss takes a long time relative to the average lifespan of hosts [99]. Similarly, co-infection with WNV (not addressed in our included studies) has only recently been observed and studied [100]. The inclusion of specific refinements could also be associated with the chosen modelling approach. The current work focused exclusively on compartmental models using parameters based on data. Other types of models, such as statistical, theoretical or agent-based models, might be more suited to address specific research questions and therefore include refinements that were often not used in compartmental models. For example, agent-based models are particularly useful to study the impact of host heterogeneity and imperfect mixing.

Another major factor in the inclusion of refinements is the availability of empirical data to underpin parametrization. This is illustrated by the development of models including multiple host species. The experimental work by Komar *et al.* [98] enabled researchers to study the role of multiple host species and the differences between these on WNV dynamics. All papers including multiple host species were published after this experimental study. Especially models classified as 'predictive' and 'applied' would benefit from high-quality empirical studies, as these types of models included most refinements and relied on data for their modelling efforts. It is important to highlight that our search strategy used published modelling papers as a starting point for identifying empirical studies. It is therefore possible that relevant empirical studies were missed if they were never used in modelling studies. A systematic review aimed at identifying all empirical studies for a specific parameter could explore whether such 'missed' studies exist, but this was outside the scope of the current work.

(a) Challenges in translating parameter values from empirical sources

Based on our results, we observed two trends regarding the use of empirical evidence in modelling efforts. We found that the body of evidence from empirical studies is not used equally, with many studies rarely being used and a small number of studies being used frequently mostly because they were cited in other model papers. This could be a consequence of researchers building on previous work without considering alternatives but could also reflect that some studies are being viewed as more applicable to the modelled system or methodologically stronger. In papers that cited empirical studies as the source of a parameter value, it was often not described how the data were translated to a relevant parameter value. Secondly, we noticed that models using parameters based on data (the focus of this review) are limited in what refinements can be studied by the availability of reliable and relevant data. The inclusion of model refinements often leads to an increase in the number of model parameters, giving rise to challenges in model parametrization. Based on how empirical studies were used in the model papers, we highlight three challenges that researchers encounter when parametrizing their models with empirical data: (i) experimental conditions may not reflect conditions in the modelled system, (ii) not all model parameters can be (easily) estimated in an experimental setting, and (iii) it is not straightforward how to combine results from multiple studies, especially when different species and pathogen strains are involved.

One of the major assumptions that must be made is that **experimental conditions reflect conditions in the modelled system**. This includes environmental conditions such as temperature and humidity as well as inoculation route and dose. Several steps of the transmission cycle have been shown to be sensitive to temperature conditions [101]. Only 20 of the 77 models included used temperature-dependent parameters. However, instead of parameter values changing as a function of temperature, models can also use temperature-explicit parameters with constant values, which represent a specific temperature scenario, such as in Vogels *et al.* [65]. The extrinsic incubation period strongly depends on temperature [102], so when interpreting mosquito infection experiments, it is important to compare the temperature in the experiment with the temperature of the modelled system to determine if the results from this experiment are useful. This is, however, complicated by the fact that temperature can be controlled and kept constant in laboratory settings, whereas it is difficult to know what temperatures mosquitoes are exposed to outside an experimental setting. Although coarse-scale air temperature is often used as a proxy, these estimates can differ significantly from the small-scale microclimates mosquitoes are exposed to, which can have a substantial impact on transmission potential [103]. Only one of the cited empirical studies for EIP looked at the impact of varying temperatures on EIP (by using an outside cage) [104]. Also, the inoculation route and dose have a strong impact on experimental results. In the two most cited papers as a source for the host-to-mosquito transmission probability, mosquitoes fed on viraemic chickens with two different viraemia levels, showing that the exposure dose has a large impact on resulting transmission rates [105,106]. After selecting a relevant route that matches the modelled system, choosing which dose to use for the parameter value can be difficult as it is not always known which best reflects natural infection, especially as viraemia levels change over the course of an infection. Ideally, transmission rates are determined for animals that were exposed through a natural route. For WNV, this was done through a host–mosquito–host system [107], and this has also been done using a host–mosquito–host system as well as mosquito–host–mosquito system for Rift Valley fever virus [108,109]. Another consideration a modeller needs to take into account is to make sure that the parameters used in a model corresponds to the appropriate WNV strain. Most papers cited Komar [110] and since this study was based on a North American strain, the inference of studies focusing on transmission in Europe could be affected (e.g. [111,112] for a discussion on WNV strains and strain replacement).

It is not always possible to directly estimate model parameters in experimental and field studies. Additionally, the quantities estimated in experimental and field studies do not always match the interpretation of model parameters. Experimental infection studies investigating the extrinsic incubation rate, included in 49% of the included studies, often show viral titres over time after infection, but interpreting which titre corresponds to a dose sufficiently high to represent infectiousness is not straightforward. Infectiousness of both host and mosquitoes is a function of viral load, with a higher viral load corresponding to a higher transmission probability [111]. Additionally, individuals with low viral titres can also contribute significantly to the transmission dynamics, depending on their numerical prominence [113,114]. Defining a cut-off value for the extrinsic incubation period is therefore a somewhat arbitrary choice. Secondly, heterogeneous biting of the mosquito towards different hosts was accounted for in 24% of the models using feeding preference coefficients. These are typically quantified in field experiments, where captured blood-fed mosquitoes are analysed to determine what hosts they fed on [18,115]. However, these estimates are limited to the context of host availability in the field settings [116]. Relative host availability is known to influence mosquito feeding behaviour [117], but mosquito host-seeking behaviour operates on small spatial scales for which host availability is often unknown. Thus, results of feeding preference experiments cannot be easily translated into a feeding preference coefficient in a model and context with different host availability. This challenge may limit the integration of heterogeneous biting in models. This limitation hampers, among others, the understanding of the impact of the ecosystem composition or the change of biodiversity on disease risk using models.

It is sometimes required to combine multiple empirical studies to parametrize a model. When multiple estimates of a parameter are available, combining studies can increase the accuracy of parameter values or provide a better notion of the variability of the parameter. Similarly, when no information is available for a species of interest, multiple studies can be combined to average parameter values of related species [49]. However, studies can differ in their designs and outcome measures, and each have their own limitations, such as measurement errors, biases or missing values, which can introduce uncertainty into the parameter estimation process. Additionally, different data sources may provide conflicting or inconsistent information, requiring careful consideration and potential reconciliation of the discrepancies. For example, the empirical studies used for parametrizing the disease-induced death rate in included models show considerable variation in mortality rates in crows. Laboratory experiments reported a 100% mortality rate [110], while field observations indicated an overall mortality of 43% [118], with further variations being observed in different locations [46,119,120]. While for some species multiple studies are available, authors including less well-studied species may not be able to find empirical evidence for their species of interest. For example, in Lord & Day [49], the authors had no available information for their species of interest and so decided to consider a large interval for those parameters based on sources for similar species.

Modellers are often confronted with multiple of the described challenges when developing realistically parametrized models. In such cases, several approaches exist to ensure that model outcomes reflect the uncertainty in the choice of model parameters. In the reviewed literature, some authors accounted for parameter uncertainty by using ranges rather than single values for parameters, such as in [49]. When running the model several times, taking samples from this range, the outcome measures can be presented as an interval rather than a single value. This helps capture the inherent uncertainty and provides a range of possible model outcomes. Another approach to this is to use probabilistic methods, like Monte Carlo simulations, to sample parameter values from probability distributions. Alternatively, explicit sensitivity or elasticity analyses can be performed to assess how variations in parameter values affect model outcomes like the basic reproduction number R_0. Examples of this in the reviewed literature include [25,41]. By systematically varying these parameters within plausible ranges, researchers can gain insights into the model's robustness and identify which life-history effects have the most significant influence on WNV transmission. Ultimately, combining these approaches helps produce more reliable predictions, reflective of the uncertainty in parameter values.

There is a growing interest in comparing models and combining insights across different modelling approaches. Several projects have been undertaken in which public health questions were addressed by combining model-based outcomes in an ensemble forecast [121–123]. For WNV, a recent ensemble effort is the study of Holcomb *et al*. [124], which, based on the 2020 Centers for Disease Control and Prevention (CDC) Challenge data, found that simpler models (i.e. with fewer variables) based on historical cases often outperformed more complex ones in predicting cases of WNV neuroinvasive disease, suggesting minimal gains from additional factors. While guidelines for combining empirical studies exist (e.g. Consolidating Standards of Reporting Trials (CONSORT) and Strengthening the Reporting of Observational Studies in Epidemiology (STROBE)) and are widely used, such methods for systematically reviewing, comparing and combining model studies are still being developed [125–127]. As Pollet *et al*. [125] highlighted, such guidelines have the potential to improve the quality and usability of model-based prediction for public health. Approaches to evaluate and synthesize results across model studies need to address high-level questions such as how to bring together insights from theoretical models with detailed data-driven simulation models and what determines the quality of a model, as well as more practical questions such as how to synthesize results across different spatial and temporal scales. Additionally, this could help understand how including specific refinements in a model affects the outcome. Such an endeavour for WNV holds potential, not only in improving modelling predictions but also in assisting the establishment of policy guidelines for more efficient control of the disease.

5. Conclusion

In conclusion, transmission models of WNV have recently increasingly deviated from the basic Ross–Macdonald framework, which allowed for more complex questions to be answered. However, some extensions have received more attention than others, due to theoretical and biological reasons as well as availability of parameter estimates. Especially applied, predictive models included a large number of refinements. This implies that these types of models, the ones often used to answer policy-related questions, are particularly sensitive to the availability and use of empirical studies to inform parametrization. Bridging the gap between empirical data and mathematical modelling presents its own set of challenges. Translating parameter values from empirical sources into models can be a complex task, as it requires careful consideration of various factors. It is important that experimental conditions reflect the conditions in the modelled system as well as possible to ensure meaningful and reliable results. However, estimating model parameters directly from experimental and field studies is not always feasible due to practical limitations and constraints. Furthermore, the integration of multiple empirical studies to parametrize a model poses additional challenges, such as inconsistencies and variations in data sources and methodologies. Overcoming these obstacles requires a collaborative effort between mathematical modellers and experimentalists, where both are involved in the design of empirical studies and in the design and parametrization of models. This will create opportunities to advance our understanding of WNV transmission and enhance its practical applications in decision-making for policy.

Ethics. This work did not require ethical approval from a human subject or animal welfare committee.

Data accessibility. The data are provided in electronic supplementary material [128].

Declaration of AI use. We have not used AI-assisted technologies in creating this article.

Authors' contributions. M.M.d.W.: conceptualization, data curation, formal analysis, methodology, writing—original draft, writing—review and editing; A.D.M.: conceptualization, data curation, formal analysis, methodology, writing—original draft, writing—review and editing; C.D.: conceptualization, data curation, formal analysis, methodology, writing—original draft, writing—review and editing; H.H.: conceptualization, funding acquisition, methodology, supervision, writing—review and editing; Q.A.t.B.: conceptualization, funding acquisition, methodology, supervision, writing—review and editing.

All authors gave final approval for publication and agreed to be held accountable for the work performed therein.

Conflict of interest declaration. We declare we have no competing interests.

Funding. All authors are part of the project 'Preparing for Vector-Borne Virus Outbreaks in a Changing World: a One Health Approach' (NWA.1160.1S.210), which is (partly) financed by the Dutch Research Council (NWO)).

References

1. Chancey C, Grinev A, Volkova E, Rios M. 2015 The global ecology and epidemiology of West Nile virus. *BioMed Res. Int.* **2015**, 1–20. (doi:10.1155/2015/376230)
2. Petersen LR, Brault AC, Nasci RS. 2013 West Nile virus: review of the literature. *JAMA* **310**, 308. (doi:10.1001/jama.2013.8042)
3. Bellini R, Zeller H, Van Bortel W. 2014 A review of the vector management methods to prevent and control outbreaks of West Nile virus infection and the challenge for Europe. *Parasit. Vectors* **7**, 323. (doi:10.1186/1756-3305-7-323)
4. Nikolay B. 2015 A review of West Nile and Usutu virus co-circulation in Europe: how much do transmission cycles overlap? *Trans. R. Soc. Trop. Med. Hyg.* **109**, 609–618. (doi:10.1093/trstmh/trv066)
5. Taieb L, Ludwig A, Ogden NH, Lindsay RL, Iranpour M, Gagnon CA, Bicout DJ. 2020 Bird species involved in West Nile virus epidemiological cycle in southern Québec. *Int. J. Environ. Res. Public Health* **17**, 4517. (doi:10.3390/ijerph17124517)
6. Andreadis TG. 2012 The contribution of *Culex pipiens* complex mosquitoes to transmission and persistence of West Nile virus in North America. *J. Am. Mosq. Control Assoc.* **28**, 137–151. (doi:10.2987/8756-971X-28.4s.137)
7. Kim KS, Tsuda Y. 2010 Seasonal changes in the feeding pattern of *Culex pipiens pallens* govern the transmission dynamics of multiple lineages of avian malaria parasites in Japanese wild bird community. *Mol. Ecol.* **19**, 5545–5554. (doi:10.1111/j.1365-294X.2010.04897.x)
8. Haba Y, McBride L. 2022 Origin and status of *Culex pipiens* mosquito ecotypes. *Curr. Biol.* **32**, R237–R246. (doi:10.1016/j.cub.2022.01.062)
9. Londono-Renteria B, Colpitts TM. 2016 A brief review of West Nile virus biology. *Methods Mol. Biol.* **1435**, 1–13. (doi:10.1007/978-1-4939-3670-0_1)
10. Barker CM. 2019 Models and surveillance systems to detect and predict West Nile virus outbreaks. *J. Med. Entomol.* **56**, 1508–1515. (doi:10.1093/jme/tjz150)
11. Keyel AC *et al.* 2021 A proposed framework for the development and qualitative evaluation of West Nile virus models and their application to local public health decision-making. *PLoS Negl. Trop. Dis.* **15**, e0009653. (doi:10.1371/journal.pntd.0009653)
12. Ross R. 1911 *The prevention of malaria*. London, UK: Murray.
13. Macdonald G. 1956 Epidemiological basis of malaria control. *Bull. World Health Organ.* **15**, 613–626.
14. Smith DL, Battle KE, Hay SI, Barker CM, Scott TW, Mckenzie FE. 2012 Ross, Macdonald, and a theory for the dynamics and control of mosquito-transmitted pathogens. *PLoS Pathog.* **8**, e1002588. (doi:10.1371/journal.ppat.1002588)
15. Heesterbeek H *et al.* 2015 Modeling infectious disease dynamics in the complex landscape of global health. *Science* **347**, aaa4339. (doi:10.1126/science.aaa4339)
16. Reiner RC *et al.* 2013 A systematic review of mathematical models of mosquito-borne pathogen transmission: 1970–2010. *J. R. Soc. Interface* **10**, 20120921. (doi:10.1098/rsif.2012.0921)
17. Kilpatrick AM, Daszak P, Jones MJ, Marra PP, Kramer LD. 2006 Host heterogeneity dominates West Nile virus transmission. *Proc. R. Soc. B* **273**, 2327–2333. (doi:10.1098/rspb.2006.3575)
18. Simpson JE, Hurtado PJ, Medlock J, Molaei G, Andreadis TG, Galvani AP, Diuk-Wasser MA. 2012 Vector host-feeding preferences drive transmission of multi-host pathogens: West Nile virus as a model system. *Proc. R. Soc. B* **279**, 925–933. (doi:10.1098/rspb.2011.1282)
19. Nasrinpour HR, Friesen MR, McLeod RD. 2019 Agent based modelling and West Nile virus: a survey. *J. Med. Biol. Eng.* **39**, 178–183. (doi:10.1007/s40846-018-0396-8)
20. Wohlin C. 2014 Guidelines for snowballing in systematic literature studies and a replication in software engineering. In *Proc. of the 18th Int. Conf. on Evaluation and Assessment in Software Engineering, London, UK, 13–14 May*. New York, NY: ACM.
21. Cecilia H, Drouin A, Métras R, Balenghien T, Durand B, Chevalier V, Ezanno P. 2022 Mechanistic models of Rift Valley fever virus transmission: a systematic review. *PLoS Negl. Trop. Dis.* **16**, e0010339. (doi:10.1371/journal.pntd.0010339)
22. Abdelrazec A, Lenhart S, Zhu H. 2014 Transmission dynamics of West Nile virus in mosquitoes and corvids and non-corvids. *J. Math. Biol.* **68**, 1553–1582. (doi:10.1007/s00285-013-0677-3)
23. Allen LJS, van den Driessche P. 2013 Relations between deterministic and stochastic thresholds for disease extinction in continuous- and discrete-time infectious disease models. *Math. Biosci.* **243**, 99–108. (doi:10.1016/j.mbs.2013.02.006)
24. Beebe TA, Robertson SL. 2017 A two-species stage-structured model for West Nile virus transmission. *Lett. Biomath.* **4**, 112–132. (doi:10.30707/LiB4.1Beebe)
25. Bergsman LD, Hyman JM, Manore CA. 2016 A mathematical model for the spread of West Nile virus in migratory and resident birds. *Math. Biosci. Eng.* **13**, 401–424. (doi:10.3934/mbe.2015009)
26. Bhowmick S, Gethmann J, Conraths FJ, Sokolov IM, Lentz HHK. 2020 Locally temperature - driven mathematical model of West Nile virus spread in Germany. *J. Theor. Biol.* **488**, 110117. (doi:10.1016/j.jtbi.2019.110117)
27. Blayneh KW, Gumel AB, Lenhart S, Clayton T. 2010 Backward bifurcation and optimal control in transmission dynamics of West Nile virus. *Bull. Math. Biol.* **72**, 1006–1028. (doi:10.1007/s11538-009-9480-0)
28. Bowman C, Gumel AB, Van Den Driessche P, Wu J, Zhu H. 2005 A mathematical model for assessing control strategies against West Nile virus. *Bull. Math. Biol.* **67**, 1107–1133. (doi:10.1016/j.bulm.2005.01.002)
29. Caillouet KA, Robertson SL. 2020 Temporal and spatial impacts of hurricane damage on West Nile virus transmission and human risk. *J. Am. Mosq. Control Assoc.* **36**, 106–119. (doi:10.2987/19-6887.1)
30. Chen J, Huang J, Beier JC, Cantrell RS, Cosner C, Fuller DO, Zhang G, Ruan S. 2016 Modeling and control of local outbreaks of West Nile virus in the United States. *Discr. Contin. Dyn. Syst. Ser. B* **21**, 2423–2449. (doi:10.3934/dcdsb.2016054)
31. Cruz-Pacheco G, Esteva L, Vargas C. 2009 Seasonality and outbreaks in West Nile virus infection. *Bull. Math. Biol.* **71**, 1378–1393. (doi:10.1007/s11538-009-9406-x)
32. Cruz-Pacheco G, Esteva L, Montaño-Hirose JA, Vargas C. 2005 Modelling the dynamics of West Nile virus. *Bull. Math. Biol.* **67**, 1157–1172. (doi:10.1016/j.bulm.2004.11.008)
33. Defelice NB, Little E, Campbell SR, Shaman J. 2017 Ensemble forecast of human West Nile virus cases and mosquito infection rates. *Nat. Commun.* **8**, 14592. (doi:10.1038/NCOMMS14592)
34. DeFelice NB *et al.* 2018 Use of temperature to improve West Nile virus forecasts. *PLoS Comput. Biol.* **14**, e1006047. (doi:10.1371/JOURNAL.PCBI.1006047)

35. Durand B, Balança G, Baldet T, Chevalier V. 2010 A metapopulation model to simulate West Nile virus circulation in Western Africa, Southern Europe and the Mediterranean basin. *Vet. Res.* **41**, 32. (doi:10.1051/vetres/2010004)

36. Ewing DA, Purse BV, Cobbold CA, White SM. 2021 A novel approach for predicting risk of vector-borne disease establishment in marginal temperate environments under climate change: West Nile virus in the UK. *J. R. Soc. Interface* **18**, 20210049. (doi:10.1098/rsif.2021.0049)

37. Fan G, Liu J, van den Driessche P, Wu J, Zhu H. 2010 The impact of maturation delay of mosquitoes on the transmission of West Nile virus. *Math. Biosci.* **228**, 119–126. (doi:10.1016/j.mbs.2010.08.010)

38. Ferraguti M *et al.* 2021 The role of different *Culex* mosquito species in the transmission of West Nile virus and avian malaria parasites in Mediterranean areas. *Transbound. Emerg. Dis.* **68**, 920–930. (doi:10.1111/tbed.13760)

39. Gourley SA, Liu R, Wu J. 2006 Some vector borne diseases with structured host populations: extinction and spatial spread. *SIAM J. Appl. Math.* **67**, 408–433. (doi:10.1137/050648717)

40. Gourley SA, Liu R, Wu J. 2007 Eradicating vector-borne diseases via age-structured culling. *J. Math. Biol.* **54**, 309–335. (doi:10.1007/s00285-006-0050-x)

41. Hartemink NA, Davis SA, Reiter P, Hubálek Z, Heesterbeek JAP. 2007 Importance of bird-to-bird transmission for the establishment of West Nile virus. *Vector-Borne Zoonotic Dis.* **7**, 575–584. (doi:10.1089/vbz.2006.0613)

42. Hartley DM, Barker CM, Le Menach A, Niu T, Gaff HD, Reisen WK. 2012 Effects of temperature on emergence and seasonality of West Nile virus in California. *Am. J. Trop. Med. Hyg.* **86**, 884–894. (doi:10.4269/ajtmh.2012.11-0342)

43. Hu X, Liu Y, Jlanhong WU. 2009 Culling structured hosts to eradicate vector-borne diseases. *Math. Biosci. Eng.* **6**, 301–319. (doi:10.3934/mbe.2009.6.301)

44. Jiang J, Qiu Z, Wu J, Zhu H. 2009 Threshold conditions for West Nile virus outbreaks. *Bull. Math. Biol.* **71**, 627–647. (doi:10.1007/s11538-008-9374-6)

45. Kioutsioukis I, Stilianakis NI. 2019 Assessment of West Nile virus transmission risk from a weather-dependent epidemiological model and a global sensitivity analysis framework. *Acta Trop.* **193**, 129–141. (doi:10.1016/j.actatropica.2019.03.003)

46. Laperriere V, Brugger K, Rubel F. 2011 Simulation of the seasonal cycles of bird, equine and human West Nile virus cases. *Prev. Vet. Med.* **98**, 99–110. (doi:10.1016/j.prevetmed.2010.10.013)

47. Li F, Liu J, Zhao XQ. 2020 A West Nile virus model with vertical transmission and periodic time delays. *J. Nonlinear Sci.* **30**, 449–486. (doi:10.1007/s00332-019-09579-8)

48. Liu J, Zhang T, Chen Q. 2020 A periodic West Nile virus transmission model with stage-structured host population. *Complexity* **2020**, 1–4. (doi:10.1155/2020/2050587)

49. Lord CC, Day JF. 2001 Simulation studies of St. Louis encephalitis and West Nile viruses: the impact of bird mortality. *Vector-Borne Zoonot. Dis.* **1**, 317–329. (doi:10.1089/15303660160025930)

50. Maidana NA, Yang HM. 2009 Spatial spreading of West Nile virus described by traveling waves. *J. Theor. Biol.* **258**, 403–417. (doi:10.1016/j.jtbi.2008.12.032)

51. Maidana NA, Yang HM. 2008 Assessing the spatial propagation of West Nile virus. *Biophys. Rev. Lett.* **3**, 227–239. (doi:10.1142/S179304800800071X)

52. Maidana NA, Yang HM. 2011 Dynamic of West Nile virus transmission considering several coexisting avian populations. *Math. Comput. Model.* **53**, 1247–1260. (doi:10.1016/j.mcm.2010.12.008)

53. Maliyoni M. 2021 Probability of disease extinction or outbreak in a stochastic epidemic model for West Nile virus dynamics in birds. *Acta Biotheor.* **69**, 91–116. (doi:10.1007/s10441-020-09391-y)

54. Marini G, Rosá R, Pugliese A, Heesterbeek H. 2017 Exploring vector-borne infection ecology in multi-host communities: a case study of West Nile virus. *J. Theor. Biol.* **415**, 58–69. (doi:10.1016/j.jtbi.2016.12.009)

55. Marini G, Rosà R, Pugliese A, Rizzoli A, Rizzo C, Russo F, Montarsi F, Capelli G. 2018 West Nile virus transmission and human infection risk in Veneto (Italy): a modelling analysis. *Sci. Rep.* **8**, 1–10.

56. Marini G *et al.* 2020 A quantitative comparison of West Nile virus incidence from 2013 to 2018 in Emilia-Romagna, Italy. *PLoS Negl. Trop. Dis.* **14**, e0007953. (doi:10.1371/journal.pntd.0007953)

57. Montecino-Latorre D, Barker CM. 2018 Overwintering of West Nile virus in a bird community with a communal crow roost. *Sci. Rep.* **8**, 6088. (doi:10.1038/S41598-018-24133-4)

58. Moschini P, Bisanzio D, Pugliese A. 2017 A seasonal model for West Nile virus. *Math. Model. Nat. Phenom.* **12**, 58–83. (https://www.mmnp-journal.org/articles/mmnp/abs/2017/02/mmnp2017122p58/mmnp2017122p58.html)

59. Nie LF, Shen JY. 2019 A state-dependent control against transmission of West Nile virus from mosquitoes to birds. *Nonlinear Dyn.* **96**, 751–763. (doi:10.1007/s11071-019-04819-8)

60. Robertson SL, Caillouët KA. 2016 A host stage-structured model of enzootic West Nile virus transmission to explore the effect of avian stage-dependent exposure to vectors. *J. Theor. Biol.* **399**, 33–42. (doi:10.1016/j.jtbi.2016.03.031)

61. Schaefer E, Caillouët KA, Robertson SL. 2018 Methods for prophylactic management of West Nile virus using a stage-structured avian host-vector model with vaccination, larvicide, and adulticide. *Nat. Resour. Model.* **31**, e12165. (doi:10.1111/nrm.12165)

62. Shan C, Fan G, Zhu H. 2020 Periodic phenomena and driving mechanisms in transmission of West Nile virus with maturation time. *J. Dyn. Differ. Equ.* **32**, 1003–1026. (doi:10.1007/s10884-019-09758-x)

63. Tarboush AK, Lin ZG, Zhang MY. 2017 Spreading and vanishing in a West Nile virus model with expanding fronts. *Sci. China Math.* **60**, 841–860. (doi:10.1007/s11425-016-0367-4)

64. Thomas D, Weedermann M, Billings L, Hoffacker J, Washington-Allen RA. 2009 When to spray: a time-scale calculus approach to controlling the impact of West Nile virus. *Ecol. Soc.* **14**, 21. (doi:10.5751/ES-03006-140221)

65. Vogels CBF, Hartemink N, Koenraadt CJM. 2017 Modelling West Nile virus transmission risk in Europe: effect of temperature and mosquito biotypes on the basic reproduction number. *Sci. Rep.* **7**, 1–11. (doi:10.1038/s41598-016-0028-x)

66. Wang FB, Wu R, Zhao XQ. 2019 A West Nile virus transmission model with periodic incubation periods. *SIAM J. Appl. Dyn. Syst.* **18**, 1498–1535. (doi:10.1137/18M1236162)

67. Wang Y, Pons W, Fang J, Zhu H. 2017 The impact of weather and storm water management ponds on the transmission of West Nile virus. *R. Soc. Open Sci.* **4**, 170017. (doi:10.1098/RSOS.170017)

68. Wonham MJ, De-Camino-Beck T, Lewis MA. 2004 An epidemiological model for West Nile virus: invasion analysis and control applications. *Proc. R. Soc. B* **271**, 501. (doi:10.1098/rspb.2003.2608)

69. Xu X, Xiao Y, Cheke RA. 2015 Models of impulsive culling of mosquitoes to interrupt transmission of West Nile virus to birds. *Appl. Math. Model*. **39**, 3549–3568. (doi:10.1016/j.apm.2014.10.072)

70. Zhang J, Jin Z, Zhu H, Zhang J, Jin Z, Zhu H. 2018 Birds movement impact on the transmission of West Nile virus between patches. *J. Appl. Anal. Comput.* **8**, 443–456. (doi:10.11948/2018.443)

71. Zhang J, Cosner C, Zhu H. 2018 Two-patch model for the spread of West Nile virus. *Bull. Math. Biol.* **80**, 840–863. (doi:10.1007/s11538-018-0404-8)

72. Zhou W, Xiao Y, Heffernan JM. 2019 A two-thresholds policy to interrupt transmission of West Nile virus to birds. *J. Theor. Biol.* **463**, 22–46. (doi:10.1016/j.jtbi.2018.12.013)
73. Calistri P, Savini L, Candeloro L, Di Sabatino D, Cito F, Bruno R, Danzetta ML. 2016 A transitional model for the evaluation of West Nile virus transmission in Italy. *Transbound. Emerg. Dis.* **63**, 485–496. (doi:10.1111/tbed.12290)
74. Cruz-Pacheco G, Esteva L, Vargas C. 2012 Multi-species interactions in West Nile virus infection. *J. Biol. Dyn.* **6**, 281–298. (doi:10.1080/17513758.2011.571721)
75. Pawelek KA, Niehaus P, Salmeron C, Hager EJ, Hunt GJ. 2014 Modeling dynamics of *Culex pipiens* complex populations and assessing abatement strategies for West Nile virus. *PLoS ONE* **9**, e108452. (doi:10.1371/journal.pone.0108452)
76. West N, Chellamuthu VK, Chellamuthu V. 2020 Modeling the effects of passive immunity in birds for the disease dynamics of West Nile virus. *Spora* **6**, 16–25. (doi:10.30707/SPORA6.1/PAMG6515)
77. Ferguson JM, Langebrake JB, Cannataro VL, Garcia AJ, Hamman EA, Martcheva M, Osenberg CW. 2014 Optimal sampling strategies for detecting zoonotic disease epidemics. *PLoS Comput. Biol.* **10**, 1003668. (doi:10.1371/journal.pcbi.1003668)
78. Zhou W, Xiao Y, Cheke RA. 2016 A threshold policy to interrupt transmission of West Nile virus to birds. *Appl. Math. Model* **40**, 8794–8809. (doi:10.1016/j.apm.2016.05.040)
79. Kerkow A, Wieland R, Gethmann JM, Hölker F, Lentz HHK. 2022 Linking a compartment model for West Nile virus with a flight simulator for vector mosquitoes. *Ecol. Modell.* **464**, 109840. (doi:10.1016/j.ecolmodel.2021.109840)
80. Bai Z, Zhao XQ. 2022 Threshold dynamics of a nonlocal and time-delayed West Nile virus model with seasonality. *Commun. Nonlinear Sci. Numer. Simul*. **115**, 106758. (doi:10.1016/j.cnsns.2022.106758)
81. Angelou A, Kioutsioukis I, Stilianakis NI. 2021 A climate-dependent spatial epidemiological model for the transmission risk of West Nile virus at local scale. *One Health* **13**, 100330. (doi:10.1016/j.onehlt.2021.100330)
82. Garba SM, Safi MA. 2013 Mathematical analysis of West Nile virus model with discrete delays. *Acta Math. Sci.* **33**, 1439–1462. (doi:10.1016/S0252-9602(13)60095-8)
83. Malik T. 2018 A discrete time West Nile virus transmission model with optimal bird- and vector-specific controls. *Math. Biosci.* **305**, 60–70. (doi:10.1016/j.mbs.2018.08.008)
84. Abdelrazec A, Lenhart S, Zhu H. 2015 Dynamics and optimal control of a West Nile virus model with seasonality. *Can. Appl. Math. Quart.* **23**, 12–33.
85. Chowell-Puente D, Delgado P, Pérez D, Tapia CHS, Sánchez F, Murillo D. 2004 *The impact of mosquito-bird interaction on the spread of west Nile virus to human populations*. Department of Biometrics, Cornell University, Technical Report Series.
86. Malik TU, Salceanu PA, Mubayi AN, Tridane AB, Imran MU. 2012 West Nile dynamics: virus transmission between domestic and wild bird populations through vectors. *Can. Appl. Math. Quart.* **20**, 535–556.
87. Sweilam NH, Saad OM, Mohamed DG. 2019 Numerical treatments of the transmission dynamics of West Nile virus and it's optimal control. *Elect. J. Math. Anal. Appl.* **7**, 9–38.
88. Cruz-Pacheco G, Esteva L, Montano JA, Vargas C. 2004 A mathematical model for the dynamics of West Nile virus. *IFAC Proc. Vol.* **37**, 475–480. (doi:10.1016/S1474-6670(17)30514-1)
89. Liu M, Zhu M, Song X. 2021 Modeling the transmission of West Nile virus with *Wolbachia* in a heterogeneous environment. *J. Nonlinear Model. Anal.* **3**, 301-–3319. (doi:10.12150/jnma.2021.301)
90. Agusto F, Bond D, Cohen A, Ding W, Leander R, Royer A. 2022 Optimal impulse control of West Nile virus. *AIMS Math.* **7**, 19 597–19 628. (doi:10.3934/math.20221075)
91. Fasano A, Riccetti N, Angelou A, Gomez-Ramirez J, Ferraccioli F, Kioutsioukis I, Stilianakis NI. 2022 An epidemiological model for mosquito host selection and temperature-dependent transmission of West Nile virus. *Sci. Rep.* **12**, 1–14. (doi:10.1038/s41598-022-24527-5)
92. Ferraguti M, Dimas Martins A, Artzy-Randrup Y. 2023 Quantifying the invasion risk of West Nile virus: insights from a multi-vector and multi-host SEIR model. *One Health* **17**, 100638. (doi:10.1016/J.ONEHLT.2023.100638)
93. Martins AD, ten Bosch Q, Heesterbeek JAP. 2022 Exploring the influence of competition on arbovirus invasion risk in communities. *PLoS ONE* **17**, e0275687.
94. Fesce E, Marini G, Rosà R, Lelli D, Cerioli MP, Chiari M, Farioli M, Ferrari N. 2023 Understanding West Nile virus transmission: mathematical modelling to quantify the most critical parameters to predict infection dynamics. *PLoS Negl. Trop. Dis.* **17**, e0010252. (doi:10.1371/journal.pntd.0010252)
95. Holcomb KM, Nguyen C, Komar N, Foy BD, Panella NA, Baskett ML, Barker CM. 2023 Predicted reduction in transmission from deployment of ivermectin-treated birdfeeders for local control of West Nile virus. *Epidemics* **44**, 100697. (doi:10.1016/J.EPIDEM.2023.100697)
96. Shao M, Zhang Q, Zhao H. 2023 Necessary and sufficient conditions for near-optimal controls of a stochastic West Nile virus system with spatial diffusion. *Math. Methods Appl. Sci.* **46**, 5029–5059. (doi:10.1002/mma.8817)
97. Bai Z, Zhang Z, Bai Z, Zhang Z. 2022 Dynamics of a periodic West Nile virus model with mosquito demographics. *Commun. Pure Appl. Anal.* **21**, 3755–3775. (doi:10.3934/cpaa.2022121)
98. Komar N, Langevin S, Hinten S, Nemeth N, Edwards E, Hettler D, Davis B, Bowen R, Bunning M. 2003 Experimental infection of North American birds with the New York 1999 strain of West Nile virus. *Emerg. Infect. Dis.* **9**, 311–322. (doi:10.3201/eid0903.020628)
99. McKee EM *et al.* 2015 West Nile virus antibody decay rate in free-ranging birds. *J. Wildl. Dis.* **51**, 601–608. (doi:10.7589/2014-07-175)
100. Wang H, Abbo SR, Visser TM, Westenberg M, Geertsema C, Fros JJ, Koenraadt CJM, Pijlman GP. 2020 Competition between Usutu virus and West Nile virus during simultaneous and sequential infection of *Culex pipiens* mosquitoes. *Emerg. Microbes Infect.* **9**, 2642–2652. (doi:10.1080/22221751.2020.1854623)
101. Shocket MS, Verwillow AB, Numazu MG, Slamani H, Cohen JM, El Moustaid F, Rohr J, Johnson LR, Mordecai EA. 2020 Transmission of West Nile and five other temperate mosquito-borne viruses peaks at temperatures between 23°C and 26°C. *Elife* **9**, 1–67. (doi:10.7554/eLife.58511)
102. Kilpatrick AM, Meola MA, Moudy RM, Kramer LD. 2008 Temperature, viral genetics, and the transmission of West Nile virus by *Culex pipiens* mosquitoes. *PLoS Pathog.* **4**, e1000092. (doi:10.1371/journal.ppat.1000092)
103. Murdock CC, Evans MV, McClanahan TD, Miazgowicz KL, Tesla B. 2017 Fine-scale variation in microclimate across an urban landscape shapes variation in mosquito population dynamics and the potential of *Aedes albopictus* to transmit arboviral disease. *PLoS Negl. Trop. Dis.* **11**, e0005640. (doi:10.1371/journal.pntd.0005640)
104. Cornel AJ, Jupp PG, Blackburn NK. 1993 Environmental temperature on the vector competence of *Culex univittatus* (Diptera: Culicidae) for West Nile virus. *J. Med. Entomol.* **30**, 449–456. (doi:10.1093/jmedent/30.2.449)
105. Sardelis MR, Turell MJ. 2001 *Ochlerotatus j. japonicus* in Frederick County, Maryland: discovery, distribution, and vector competence for West Nile virus. *J. Am. Mosq. Control Assoc.* **17**, 137–141.
106. Turell MJ, Oliver J, O'Guinn M. 2000 Potential for New York mosquitoes to transmit West Nile virus. *Am. J. Trop. Med. Hyg.* **62**, 413–414. (doi:10.4269/ajtmh.2000.62.413)
107. Turell MJ, O'Guinn ML, Dohm DJ, Jones JW. 2001 Vector competence of North American mosquitoes (Diptera: Culicidae) for West Nile virus. *J. Med. Entomol.* **38**, 130–134. (doi:10.1603/0022-2585-38.2.130)
108. Wichgers Schreur PJ, Vloet RPM, Kant J, van Keulen L, Gonzales JL, Visser TM, Koenraadt CJM, Vogels CBF, Kortekaas J. 2021 Reproducing the Rift Valley fever virus mosquito-lamb-mosquito transmission cycle. *Sci. Rep.* **11**, 1–10. (doi:10.1038/s41598-020-79267-1)

109. Bron GM, Schreur PJW, de Jong MCM, van Keulen L, Vloet RPM, Koenraadt CJM, Kortekaas J, ten Bosch QA. 2023 Quantifying Rift Valley fever virus transmission efficiency in a lamb-mosquito-lamb model. *bioRxiv* 538218.
110. Komar N. 2003 West Nile virus: epidemiology and ecology in North America. *Adv. Virus Res.* **61**, 185–234. (doi:10.1016/S0065-3527(03)61005-5)
111. Kain MP, Bolker BM. 2017 Can existing data on West Nile virus infection in birds and mosquitos explain strain replacement? *Ecosphere* **8**, e01684.
112. Koch RT, Erazo D, Folly AJ, Johnson N, Dellicour S, Grubaugh ND, Vogels CBF. 2024 Genomic epidemiology of West Nile virus in Europe. *One Health* **18**, 100664. (doi:10.1016/j.onehlt.2023.100664)
113. ten Bosch QA *et al.* 2018 Contributions from the silent majority dominate dengue virus transmission. *PLoS Pathog.* **14**, e1006965. (doi:10.1371/JOURNAL.PPAT.1006965)
114. Cecilia H, Vriens R, Wichgers Schreur PJ, de Wit MM, Métras R, Ezanno P, ten Bosch QA. 2022 Heterogeneity of Rift Valley fever virus transmission potential across livestock hosts, quantified through a model-based analysis of host viral load and vector infection. *PLoS Comput. Biol.* **18**, e1010314. (doi:10.1371/journal.pcbi.1010314)
115. Kilpatrick AM, Kramer LD, Jones MJ, Marra PP, Daszak P. 2006 West Nile virus epidemics in North America are driven by shifts in mosquito feeding behavior. *PLoS Biol.* **4**, 606–610. (doi:10.1371/journal.pbio.0040082)
116. Takken W, Verhulst NO. 2013 Host preferences of blood-feeding mosquitoes. *Annu. Rev. Entomol.* **58**, 433–453. (doi:10.1146/annurev-ento-120811-153618)
117. Lyimo IN, Ferguson HM. 2009 Ecological and evolutionary determinants of host species choice in mosquito vectors. *Trends Parasitol.* **25**, 189–196. (doi:10.1016/j.pt.2009.01.005)
118. McLean RG. 2006 West Nile virus in North American birds. *Ornithol. Monogr.* **60**, 44–64. (doi:10.1642/0078-6594(2006)60[44:WNVINA]2.0.CO;2)
119. Caffrey C, Weston TJ. 2003 High mortality among marked crows subsequent to the arrival of West Nile virus. *Wildl. Soc. Bull. (1973-2006)* **31**, 870–872.
120. Yaremych SA, Warner RE, Mankin PC, Brawn JD, Raim A, Novak R. 2004 West Nile virus and high death rate in American crows. *Emerg. Infect. Dis.* **10**, 709–711. (doi:10.3201/eid1004.030499)
121. Viboud C *et al.* 2018 The RAPIDD ebola forecasting challenge: synthesis and lessons learnt. *Epidemics* **22**, 13–21. (doi:10.1016/j.epidem.2017.08.002)
122. Ezanno P *et al.* 2022 The African swine fever modelling challenge: model comparison and lessons learnt. *Epidemics* **40**, 100615. (doi:10.1016/j.epidem.2022.100615)
123. Borchering RK, Healy JM, Cadwell BL, Johansson MA, Slayton RB, Wallace M, Biggerstaff M. 2023 Public health impact of the U.S. scenario modeling hub. *Epidemics* **44**, 100705. (doi:10.1016/j.epidem.2023.100705)
124. Holcomb KM *et al.* 2023 Evaluation of an open forecasting challenge to assess skill of West Nile virus neuroinvasive disease prediction. *Parasit. Vect.* **16**, 1–13. (doi:10.1186/s13071-022-05630-y)
125. Pollett S *et al.* 2020 Identification and evaluation of epidemic prediction and forecasting reporting guidelines: a systematic review and a call for action. *Epidemics* **33**, 100400. (doi:10.1016/J.EPIDEM.2020.100400)
126. Hollingsworth TD, Medley GF. 2017 Learning from multi-model comparisons: collaboration leads to insights, but limitations remain. *Epidemics* **18**, 1–3. (doi:10.1016/j.epidem.2017.02.014)
127. Pollett S *et al.* 2021 Recommended reporting items for epidemic forecasting and prediction research: the EPIFORGE 2020 guidelines. *PLoS Med.* **18**, e1003793. (doi:10.1371/journal.pmed.1003793)
128. de Wit MM, Dimas Martins A, Delecroix C, Heesterbeek H, ten Bosch QA. 2024 Mechanistic models for West Nile virus transmission: a systematic review of features, aims and parametrization. Figshare. (doi:10.6084/m9.figshare.c.7093224)