Tropical Medicine and Infectious Disease

MDPI

Brief Report

The Effect of Increased Temperature on Dengue Virus in the Vector *Aedes aegypti* from New Caledonia

Méryl Delrieu [1,*], Olivia O'Connor [1], Nicolas Pocquet [2], Kenny Teraiharoa [2], Anne-Fleur Griffon [1], Christophe Menkes [3], Morgan Mangeas [3], Elvina Viennet [4], Valérie Burtet-Sarramegna [5], Myrielle Dupont-Rouzeyrol [1,†] and Francesca D. Frentiu [4,†]

[1] Institut Pasteur of New Caledonia, Institut Pasteur International Network, URE Dengue and Arboviruses, Noumea Cedex BP 61 98845, New Caledonia; ooconnor@pasteur.nc (O.O.); gannefle@pasteur.nc (A.-F.G.); mdupont@pasteur.nc (M.D.-R.)
[2] Institut Pasteur of New Caledonia, Institut Pasteur International Network, URE Medical Entomology, Noumea Cedex BP 61 98845, New Caledonia; npocquet@pasteur.nc (N.P.); keteraih@pasteur.nc (K.T.)
[3] Institut de Recherche Pour le Développement, UMR ENTROPIE, University of New Caledonia, University of La Réunion, CNRS, Ifremer, Noumea Cedex BP A5 98845, New Caledonia; christophe.menkes@ird.fr (C.M.); morgan.mangeas@ird.fr (M.M.)
[4] School of Biomedical Sciences, Centre for Immunology and Infection Control, Queensland University of Technology, Brisbane, QLD 4000, Australia; eviennet@redcrossblood.org.au (E.V.); francesca.frentiu@qut.edu.au (F.D.F.)
[5] Institute of Exact and Applied Sciences (ISEA), University of New Caledonia, Noumea Cedex BP R4 98 851, New Caledonia; valerie.sarramegna@unc.nc
* Correspondence: mdelrieu@pasteur.nc
† These authors contributed equally to this work.

Abstract

Dengue virus (DENV) is a major public health concern in tropical and subtropical regions, including the Pacific. Temperature is recognised as a major driver of transmission under climate change. Understanding how higher temperatures may alter DENV transmission is essential to anticipate future dengue risk. Therefore, we assessed the effect of temperature on DENV-1 in *Aedes aegypti* from New Caledonia. Mosquitoes were orally infected and maintained for 14 days at 26.6 °C (average temperatures during recent outbreaks) or 31.1 °C (SSP5-8.5 scenario projected temperatures). Mosquito bodies, heads, and saliva were analysed separately to determine infection, dissemination, and transmission rates as well as transmission efficiencies. Infectious virus was detected by using a fluorescent focus assay, and viral titres were quantified via $TCID_{50}$ assays. No significant differences were observed in infection, dissemination, and transmission rates or transmission efficiencies between the two temperatures. However, DENV titres in mosquito bodies and heads were significantly higher at 31.1 °C than 26.6 °C. Our results indicate that elevated temperature increases viral loads within the insect but not the proportion of infectious mosquitoes, highlighting the importance of considering temperature as a key parameter in assessing dengue risk under climate change. Further studies are needed to investigate the effects of temperature on virus–mosquito interactions.

Keywords: temperature; climate change; dengue virus; *Aedes aegypti*; viral replication

Academic Editors: Harriet Lawford and Adam Craig

Received: 15 January 2026
Revised: 7 February 2026
Accepted: 12 February 2026
Published: 14 February 2026

1. Introduction

Rising temperatures are one of the most noticeable effects of climate change and contribute to the resurgence of vector-borne diseases, including dengue fever, chikungunya, West Nile fever, and Zika, by influencing both vector biology and viral dynamics [1–4].

Dengue virus (DENV) is one of the most important arboviruses worldwide, with 390 million estimated cases annually [5], and is primarily transmitted by the mosquito *Aedes aegypti*. Modelling studies predict a geographical expansion of vector ranges and longer seasons of vector-borne pathogen circulation, leading to an overall increased risk of transmission [6–9]. Virus infection is favoured under higher temperatures, as in vivo experiments show that exposure to warmer temperatures often leads to elevated mosquito infection rates and a shortened extrinsic incubation period [10]. Island territories are particularly vulnerable to the effects of climate change, yet they remain underrepresented in experimental studies exploring its impact on infectious diseases. New Caledonia, a tropical island in the Pacific Ocean, has a long history of DENV circulation [11,12]. However, over the past 15 years, a shift in epidemiological patterns has been observed with the persistence of DENV-1 circulation, the co-circulation of multiple DENV serotypes and other arboviruses, and an increase in the severity of dengue disease [12–15]. In 2018, a *Wolbachia* biocontrol strategy was implemented through the World Mosquito Program in four cities of New Caledonia. This intervention led to a decline in DENV cases, with 22 reported for 2025 in New Caledonia, including 9 local cases (https://dass.gouv.nc/votre-sante-maladies/la-dengue-le-chikungunya-et-le-zika; accessed on 7 February 2026) compared to 572 cases between January and April 2018 (https://dass.gouv.nc/sites/default/files/atoms/files/graphedenguedu01092017au06042018.pdf; accessed on 7 February 2026). A recent modelling study predicts an increased risk of DENV epidemics and longer transmission periods in New Caledonia due to rising temperatures, with annual outbreaks anticipated under the SSP5-8.5 scenario, assuming no *Wolbachia*-based vector control strategies [8]. Among the five climate scenarios proposed by the Intergovernmental Panel on Climate Change (IPCC), SSP5-8.5 represents a worst-case trajectory with increases reaching +4.5 °C (between +3.3 °C and +5.7 °C) by 2100 [16]. Here, we investigated the impact of this scenario on DENV infection in *Ae. aegypti* mosquitoes from New Caledonia by contrasting present-day mean temperatures with projected end-of-century conditions under SSP5-8.5. Two temperature conditions were assessed: 26.6 °C, which is the current average temperature during recent DENV outbreaks [17], and 31.1 °C, which represents the SSP5-8.5 scenario, with a 4.5 °C increase over the current temperature.

2. Materials and Methods

Seven- to nine-day-old female *Ae. aegypti* mosquitoes originating from field collections during 2022 in Païta, New Caledonia (F3-F5 generation), were used in experimental infections and confirmed to be free of *Wolbachia* using established qPCR methods previously described [18,19]. Mosquitoes were deprived of sugar overnight before being fed for 30 min on an infectious blood meal composed of DENV-1 diluted in bovine blood, with a final concentration of 1×10^7 FFU/mL. The DENV-1 genotype I strain used in this study (GenBank: MW315195.1) was isolated from human serum during the 2017 outbreak in New Caledonia [20], and viral stock was produced on Vero E6 cells (ATCC, Manassas, VA, USA). After blood feeding using a Hemotek system (Hemotek Ltd., Blackburn, UK), fully engorged females were incubated for 14 days at 26.6 °C or 31.1 °C, representing present-day and projected future temperatures, respectively, with 80% relative humidity. At 14 days post infection (dpi), 28 and 30 mosquitoes were sampled from the 26.6 °C and 31.1 °C regimes, respectively, to determine infection, dissemination, and transmission (see below for methods). Mosquitoes were anesthetized with 7% CO_2 and induced to salivate for 30 min into 5 µL of FBS (Foetal Bovine Serum, Gibco, ThermoFisher Scientific, Waltham, MA, USA). Each saliva sample was then added to 45 µL of DMEM (Gibco, ThermoFisher Scientific, Waltham, MA, USA). Mosquitoes were dissected, with heads and bodies stored separately in 350 µL of DMEM (Dulbecco's Modified Eagle Medium; Gibco, Thermo Fisher

Scientific, Waltham, MA, USA) supplemented with 2% FBS and 1% Antibiotic–Antimycotic (Gibco, ThermoFisher Scientific, Waltham, MA, USA). Saliva, heads and bodies were stored at −80 °C, with heads and bodies mechanically ground up with glass beads in a BeadBeater (BioSpec Products Inc., Bartlesville, OK, USA) at the time of analysis [15].

The presence of infectious virus in mosquito bodies, heads, and saliva (the last as a proxy for transmission) was assessed by using a fluorescent focus assay (FFA) as described below. Diluted homogenates (20-fold dilutions of ground-up body and head samples) or undiluted saliva were inoculated on Vero E6 cell monolayers and overlaid after two hours in DMEM supplemented with 5% FBS and 3.2% carboxymethylcellulose (Sigma-Aldrich, Merck, St. Louis, MO, USA). Cells were then maintained for 5 days at 37 °C under 5% CO_2. Cells were fixed, permeabilized [21], and stained using an anti-dengue complex monoclonal antibody (clone D3-2H2-9-21, Chemicon® Merck Millipore, Burlington, MA, USA) followed by Alexa Fluor 488 goat anti-mouse IgG (Invitrogen, ThermoFisher Scientific, Waltham, MA, USA). The presence of infectious virus was determined by visualising DENV foci under a fluorescent microscope (ZEISS Axiovert 5 TL FL SCB, Carl Zeiss Microscopy GmbH, Jena, Germany) at 100× magnification [15]. The infection rate was calculated as the proportion of mosquitoes with infectious virus detected in the body. The dissemination rate was the proportion of infected mosquitoes showing virus in the head, and the transmission rate was the proportion of mosquitoes with disseminated infection, confirmed with virus detected in the saliva. The transmission efficiency was calculated as the proportion of total tested mosquitoes with virus in the saliva. Viral titres in the bodies and heads of infected mosquitoes were quantified by using $TCID_{50}$ assays with serial 10-fold dilutions inoculated on Vero E6 cell monolayers, incubated for 5 days, and stained and visualised as for FFAs. The presence/absence of infectious virus was scored for each well, and $TCID_{50}$/mL values were calculated using the Spearman–Kärber method and converted to FFU/mL [22].

Pairwise comparisons of infection rate, dissemination rate, transmission rate, and transmission efficiencies between temperatures were performed using Fisher's exact tests. Viral titres were tested for normality using the Shapiro–Wilk test. Given the small sample sizes, datasets were analysed using non-parametric methods. Overall differences between groups were assessed with Wilcoxon rank sum tests. Data were visualized as boxplots showing medians and interquartile ranges, with analyses performed in R software version 4.5.1 [23].

3. Results

Infection, dissemination, and transmission rates and efficiencies are summarized in Table 1.

Table 1. DENV infection, dissemination, and transmission in *Aedes aegypti* from New Caledonia at different temperatures. Infection, dissemination, and transmission rates, as well as transmission efficiencies, were measured at 14 days post infection for each tested temperature (26.6 °C and 31.1 °C).

Temperature (°C)	Total of Mosquitoes	Infection Rate % (n/N; 95% CI)	Dissemination Rate % (n/N; 95% CI)	Transmission Rate % (n/N; 95% CI)	Transmission Efficiency % (n/N; 95% CI)
26.6	28	28.6 (8/28; 14–48.9)	100 (8/8; 59.8–100)	12.5 (1/8; 0.7–53.3)	3.6 (1/28; 0.2–20.2)
31.1	30	30 (9/30; 15.4–49.6)	100 (9/9; 62.9–100)	66.7 (6/9; 30.9–91)	20 (6/30; 8.4–39.1)

Table numbers in parentheses correspond to the number of positive mosquitoes (n) out of the total tested (N), followed by the 95% confidence interval.

Infection rates ranged from 28.6% to 30% for the mosquitoes placed at 26.6 °C and 31.1 °C, respectively. At these temperatures, viral dissemination was observed in all

infected mosquitoes (100%). The transmission rates of mosquitoes from 26.6 °C and 31.1 °C were 12.5% and 66.7%, respectively. At 26.6 °C, the transmission efficiency was 3.6%, and at 31.1 °C, the transmission efficiency was 20%. Fisher's exact tests showed no statistically significant differences in infection rate, dissemination rate, transmission rate, and transmission efficiencies between temperatures.

The median viral titres of infected mosquitoes' bodies from the 26.6 °C and 31.1 °C regimes were 8.89 × 10^3 FFU/mL and 3.75 × 10^5 FFU/mL, respectively (Figure 1A), and the difference was statistically significant (p = 0.004). The median viral titres of infected mosquito heads placed at 26.6 °C and 31.1 °C were 3.28 × 10^2 FFU/mL and 3.75 × 10^4 FFU/mL, respectively (Figure 1B). Similarly to bodies, head titres from mosquitoes at 31.1 °C were significantly higher than those at 26.6 °C (p = 0.006).

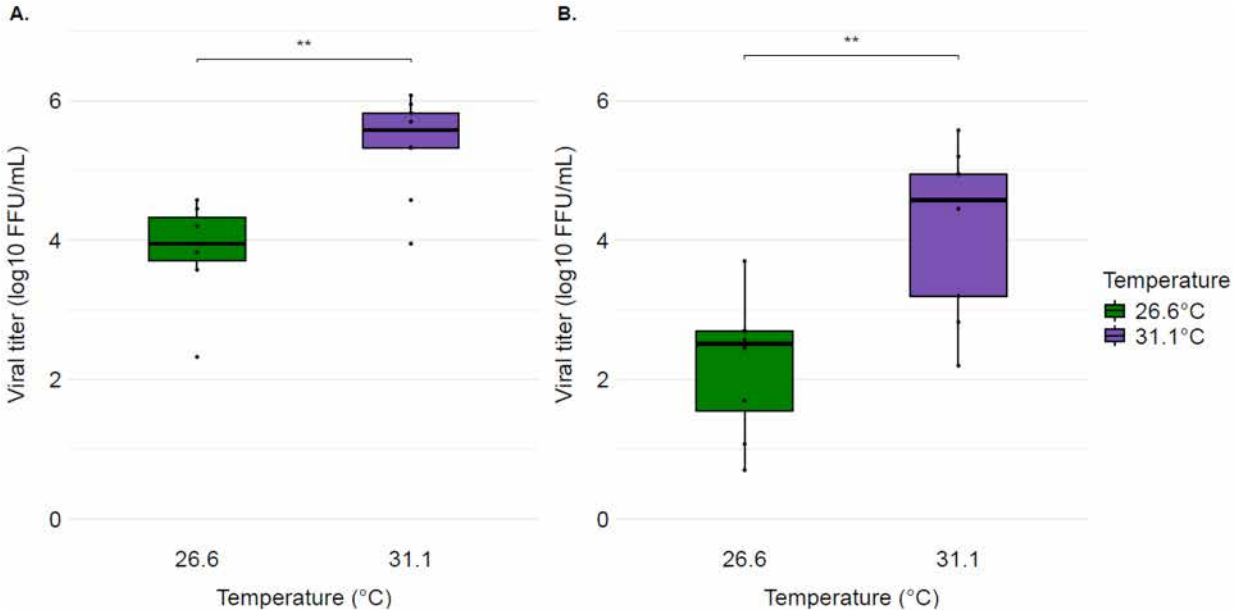

Figure 1. DENV-1 titres in mosquito tissues under different temperature conditions (26.6 °C and 31.1 °C) at 14 days post infection, as determined via TCID50 assay. (**A**) Viral titres in mosquito bodies. (**B**) Viral titres in mosquito heads. Significance levels: ns = not significant. ** $p < 0.01$.

4. Discussion

In our study, we observed that DENV-1 infection rates were around 30%. Although few mosquitoes were infected, they all disseminated the virus. These observations are in concordance with previous studies from New Caledonia using the standard temperature (28 °C) for mosquito vector competence experiments. Indeed, *Ae. aegypti* populations orally infected with DENV-1 displayed low-to-moderate infection rates but high dissemination rates reaching 100% [15,24]. These observations suggest that, once midgut infection is established in our local *Ae. aegypti*, there appears to be no restriction to viral dissemination.

In our experiments, infection, dissemination, transmission rates and dissemination and transmission efficiencies did not significantly differ between 26.6 °C and 31.1 °C. This was somewhat unexpected, as the majority of studies reported an increase in infection, dissemination, and transmission of the tested virus at higher temperatures, as reviewed here [10]. However, some studies have found no clear differences even when using temperature ranges similar to those reported in the studies mentioned above. Limited variation in DENV infection and transmission was observed in Florida populations of *Ae. aegypti* and *Aedes albopictus* [25], while others have observed that transmission potential by *Ae. albopictus* did not systematically increase across all tested temperature conditions [26]. Our

findings provide additional useful data, but larger sample sizes will be needed in future studies to confirm these trends.

In our study, infection rates were observed to be low to moderate. While such values are not uncommon, they complicate the interpretation of our results. However, similar patterns have been observed in New Caledonia at 28 °C for DENV-1 and depending on the mosquito population [15,24]. Studies from other regions of the globe have reported highly variable outcomes, ranging from very low values in Kenya and Senegal [27,28] to much higher values in Cuba, Brazil, Ghana, and Japan [29–32]. Such variability highlights the influence of mosquito population, viral strain, and geographic context. Vector competence may also be shaped by virus–vector compatibilities [33–35] and further modulated by environmental factors such as temperature [36,37] or biological factors like the mosquito gut microbiome [38–40].

Temperature appears to enhance viral loads in mosquitoes, as significantly higher DENV titres were observed at 31.1 °C than at 26.6 °C. Another study reported that viral genome levels in the bodies and salivary gland tissues of *Ae. aegypti* were generally higher at 28–34 °C, though significant differences appeared only at certain dpi and did not affect infection rates [41]. More recently, *Ae. aegypti* mosquitoes infected by DENV-2 or co-infected by DENV-2 and Mayaro virus and maintained at 32 °C displayed significantly higher viral titres than mosquitoes held at 27 °C [42]. In parallel, in vitro experiments using mosquito and mammalian cell lines also demonstrated that DENV replication was enhanced at 32 °C, with faster viral kinetics and higher titres than at 27 °C [41]. These studies reinforce the idea that temperature influences not only viral loads and transmission but also modulates the molecular and physiological response of mosquitoes to infection [43,44]. At the molecular level, high temperatures enhance the activity of viral RNA-dependent enzymes and may destabilize viral RNA structures, while simultaneously affecting host physiology by accelerating metabolism and potentially weakening innate immune defences [45,46]. In addition, temperature has been shown to modulate tissue barriers such as the midgut escape barrier. Importantly, the impact of temperature on this barrier is highly context-dependent, varying according to mosquito population, viral strains, and the thermal conditions experienced during larval and adult stages [47].

Beyond these experimental insights, the impact of temperature on DENV transmission has also been demonstrated at the population level through climate-based models. In New Caledonia, a climate-based statistical model using dengue case data has shown that when daily maximum temperatures exceed 32 °C for 10 consecutive days or more during January to March, the risk of dengue outbreaks increases [48]. Global warming is expected to increase average temperatures worldwide, along with heatwaves that are projected to become more frequent, stronger, and longer. Such conditions may increase the risk of transmission, especially in regions already impacted by dengue. In our study, although infection and dissemination rates did not differ, viral titres were significantly higher at the warmer temperature. These observations support the idea that elevated temperatures can amplify viral replication in mosquitoes and could potentially contribute to increased DENV transmission risk, although a mechanistic link remains unclear. Understanding how increased temperature and heat waves affect mosquito–virus interactions will be important to anticipate future transmission risks and improve mechanistic models for outbreak prediction under climate change.

A major limitation of this study was the very low number of positive saliva samples (n = 1 at 26.6 °C and n = 6 at 31.1 °C), which prevented reliable statistical analysis of transmission potential and limited the interpretation of transmission dynamics. The higher number of positive saliva samples at 31.1 °C suggests that higher viral loads in bodies and heads increase the potential for DENV to reach the salivary glands and, in

turn, result in more mosquitoes being infectious. However, this hypothesis needs further testing. A further limitation is that transmission was assessed at a single time point (14 dpi), which provides only a partial view of this complex process. Additionally, at 26.6 °C, viral titres were obtained for seven individuals, as one body sample could not be titrated due to insufficient material, resulting in a slightly reduced number of data points for viral load analysis at this temperature. Nevertheless, analysis of viral titres shows that higher temperatures promoted higher viral replication of DENV, underscoring the importance of further investigating how temperatures influence viral kinetics of DENV and its transmission. While our study was unable to explore the specific viral or host factors contributing to elevated DENV titres, this remains a valuable direction for future research. It should also be noted that our study did not include *Wolbachia* mosquitoes, despite the use of *Wolbachia*-based strategies for dengue control in New Caledonia. This choice was deliberate, as we aimed to establish baseline data on temperature effects in wild-type *Ae. aegypti* from New Caledonia. In addition, our study was conducted at constant temperatures, which does not reflect field conditions where mosquitoes experience daily temperature variation. Realistic field scenarios need to be considered in future studies by applying fluctuating temperature regimes.

5. Conclusions

In conclusion, we explored the effect of temperature on DENV-1 infection in *Ae. aegypti* from New Caledonia, the primary vector on this island. Despite low-to-moderate infection rates, all infected mosquitoes showed viral dissemination, with significantly higher viral titres at 31.1 °C than at 26.6 °C, indicating that higher temperature can enhance viral amplification in mosquitoes. Further studies are needed to investigate how elevated temperatures influence viral replication over time and the mosquito's potential for virus transmission and assess the possibility of DENV variant evolution under heat stress. Given that several countries in the Pacific region are currently experiencing DENV outbreaks, these insights will be critical for anticipating the broader consequences of climate change on DENV evolution and epidemiology.

Author Contributions: Conceptualization, M.D., O.O., N.P., C.M., M.M., E.V., V.B.-S., M.D.-R. and F.D.F.; methodology, M.D., O.O., N.P., V.B.-S., M.D.-R. and F.D.F.; validation, M.D.-R. and F.D.F.; formal analysis, M.D.; investigation, M.D., K.T. and A.-F.G.; data curation, M.D., O.O. and N.P.; writing—original draft preparation, M.D.; writing—review and editing, O.O., N.P., K.T., A.-F.G., C.M., M.M., E.V., V.B.-S., M.D.-R. and F.D.F.; visualization, M.D.; supervision, M.D.-R. and F.D.F.; project administration, F.D.F.; funding acquisition, C.M., M.M., E.V., V.B.-S., M.D.-R. and F.D.F. All authors have read and agreed to the published version of the manuscript.

Funding: This research was funded by PACIFIC FUND, under agreement number 2129 (CLIMATIC project). MD is supported by "Prix d'Encouragement à la Recherche", Southern Province, New Caledonia.

Institutional Review Board Statement: The dengue virus strain used in this study was isolated from a human serum sample collected under ethical approval in 2015 and 2018 and informed consent as part of a previous project (ethics approval by Comité de Protection des Personnes Ile-de-France I, n° ID-RCB 2015-A00205-44, CPP n°2015-march-13851 and 2018-january-14793 and by the Consultative Ethics Committee of New Caledonia).

Informed Consent Statement: Not applicable.

Data Availability Statement: The data supporting the conclusions of this article are included within the article.

Acknowledgments: We want to thank Catherine Inizan for providing the DENV-1 virus strain. We would like to thank Morgane Pol, Clarisse Delaunay, and Mathew Aznar for their invaluable technical support.

Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

DENV	dengue virus
DMEM	Dulbecco's Modified Eagle Medium
dpi	day post infection
FBS	Foetal Bovine Serum
FFA	fluorescent focus assay
FFU	Focus Forming Unit
qPCR	quantitative Polymerase Chain Reaction
$TCID_{50}$	Tissue Culture Infectious Dose 50%

References

1. Anikeeva, O.; Hansen, A.; Varghese, B.; Borg, M.; Zhang, Y.; Xiang, J.; Bi, P. The impact of increasing temperatures due to climate change on infectious diseases. *BMJ* **2024**, *387*, e079343. [CrossRef] [PubMed]
2. Dennington, N.L.; Grossman, M.K.; Ware-Gilmore, F.; Teeple, J.L.; Johnson, L.R.; Shocket, M.S.; McGraw, E.A.; Thomas, M.B. Phenotypic adaptation to temperature in the mosquito vector, *Aedes aegypti*. *Glob. Change Biol.* **2024**, *30*, e17041. [CrossRef] [PubMed]
3. Reinhold, J.M.; Lazzari, C.R.; Lahondère, C. Effects of the environmental temperature on *Aedes aegypti* and *Aedes albopictus* mosquitoes: A review. *Insects* **2018**, *9*, 158. [CrossRef]
4. Semenza, J.C.; Rocklöv, J.; Ebi, K.L. Climate change and cascading risks from infectious disease. *Infect. Dis. Ther.* **2022**, *11*, 1371–1390. [CrossRef]
5. Bhatt, S.; Gething, P.W.; Brady, O.J.; Messina, J.P.; Farlow, A.W.; Moyes, C.L.; Drake, J.M.; Brownstein, J.S.; Hoen, A.G.; Sankoh, O.; et al. The global distribution and burden of dengue. *Nature* **2013**, *496*, 504–507. [CrossRef]
6. Mordecai, E.A.; Cohen, J.M.; Evans, M.V.; Gudapati, P.; Johnson, L.R.; Lippi, C.A.; Miazgowicz, K.; Murdock, C.C.; Rohr, J.R.; Ryan, S.J.; et al. Detecting the impact of temperature on transmission of Zika, dengue, and chikungunya using mechanistic models. *PLoS Neglected Trop. Dis.* **2017**, *11*, e0005568. [CrossRef]
7. Ngonghala, C.N.; Ryan, S.J.; Tesla, B.; Demakovsky, L.R.; Mordecai, E.A.; Murdock, C.C.; Bonds, M.H. Effects of changes in temperature on Zika dynamics and control. *J. R. Soc. Interface* **2021**, *18*, 20210165. [CrossRef]
8. Ochida, N.; Mangeas, M.; Dupont-Rouzeyrol, M.; Dutheil, C.; Forfait, C.; Peltier, A.; Descloux, E.; Menkes, C. Modeling present and future climate risk of dengue outbreak, a case study in New Caledonia. *Environ. Health* **2022**, *21*, 20. [CrossRef]
9. Ryan, S.J.; Carlson, C.J.; Tesla, B.; Bonds, M.H.; Ngonghala, C.N.; Mordecai, E.A.; Johnson, L.R.; Murdock, C.C. Warming temperatures could expose more than 1.3 billion new people to Zika virus risk by 2050. *Glob. Change Biol.* **2021**, *27*, 84–93. [CrossRef] [PubMed]
10. Delrieu, M.; Martinet, J.-P.; O'Connor, O.; Viennet, E.; Menkes, C.; Burtet-Sarramegna, V.; Frentiu, F.D.; Dupont-Rouzeyrol, M. Temperature and transmission of chikungunya, dengue, and Zika viruses: A systematic review of experimental studies on *Aedes aegypti* and *Aedes albopictus*. *Curr. Res. Parasitol. Vector Borne Dis.* **2023**, *4*, 100139. [CrossRef]
11. Dupont-Rouzeyrol, M.; Aubry, M.; O'Connor, O.; Roche, C.; Gourinat, A.-C.; Guigon, A.; Pyke, A.; Grangeon, J.-P.; Nilles, E.; Chanteau, S.; et al. Epidemiological and molecular features of dengue virus type-1 in New Caledonia, South Pacific, 2001–2013. *Virol. J.* **2014**, *11*, 61. [CrossRef]
12. Inizan, C.; Tarantola, A.; O'Connor, O.; Mangeas, M.; Pocquet, N.; Forfait, C.; Descloux, E.; Gourinat, A.-C.; Pfannstiel, A.; Klement-Frutos, E.; et al. Dengue in New Caledonia: Knowledge and Gaps. *Trop. Med. Infect. Dis.* **2019**, *4*, 95. [CrossRef]
13. Dupont-Rouzeyrol, M.; O'Connor, O.; Calvez, E.; Daurès, M.; John, M.; Grangeon, J.P.; Gourinat, A.-C. Co-infection with Zika and dengue viruses in 2 patients, New Caledonia, 2014. *Emerg. Infect. Dis.* **2015**, *21*, 381–382. [CrossRef]
14. Marois, I.; Forfait, C.; Inizan, C.; Klement-Frutos, E.; Valiame, A.; Aubert, D.; Gourinat, A.-C.; Laumond, S.; Barsac, E.; Grangeon, J.-P.; et al. Development of a bedside score to predict dengue severity. *BMC Infect. Dis.* **2021**, *21*, 470. [CrossRef]
15. O'Connor, O.; Calvez, E.; Inizan, C.; Pocquet, N.; Richard, V.; Dupont-Rouzeyrol, M. Vector competence of *Aedes aegypti* from New Caledonia for the four recent circulating dengue virus serotypes. *PLoS Neglected Trop. Dis.* **2020**, *14*, e0008303. [CrossRef] [PubMed]
16. Intergovernmental Panel on Climate Change (IPCC). *Technical Summary. The Physical Science Basis: Working Group I Contribution to the Sixth Assessment Report of the Intergovernmental Panel on Climate Change*; Cambridge University Press: Cambridge, UK, 2023; pp. 35–144.

17. Teurlai, M.; Menkès, C.E.; Cavarero, V.; Degallier, N.; Descloux, E.; Grangeon, J.-P.; Guillaumot, L.; Libourel, T.; Lucio, P.S.; Mathieu-Daudé, F.; et al. Socio-economic and climate factors associated with dengue fever spatial heterogeneity: A worked example in New Caledonia. *PLoS Neglected Trop. Dis.* **2015**, *9*, e0004211. [CrossRef] [PubMed]
18. Pocquet, N.; O'Connor, O.; Flores, H.A.; Tutagata, J.; Pol, M.; Hooker, D.J.; Inizan, C.; Russet, S.; Duyvestyn, J.M.; Pacidônio, E.C.; et al. Assessment of fitness and vector competence of a New Caledonia wMel *Aedes aegypti* strain before field-release. *PLoS Neglected Trop. Dis.* **2021**, *15*, e0009752.
19. Rakotonirina, A.; Caruzzo, C.; Ballan, V.; Kainiu, M.; Marin, M.; Colot, J.; Richard, V.; Dupont-Rouzeyrol, M.; Selmaoui-Folcher, N.; Pocquet, N. Wolbachia detection in *Aedes aegypti* using MALDI-TOF MS coupled to artificial intelligence. *Sci. Rep.* **2021**, *11*, 21355. [CrossRef] [PubMed]
20. Inizan, C.; Minier, M.; Prot, M.; O'Connor, O.; Forfait, C.; Laumond, S.; Marois, I.; Biron, A.; Gourinat, A.-C.; Goujart, M.-A.; et al. Viral evolution sustains a dengue outbreak of enhanced severity. *Emerg. Microbes Infect.* **2021**, *10*, 536–544. [CrossRef]
21. Carneiro, P.H.; Mendes-Monteiro, L.; Mohana-Borges, R. Virus propagation and plaque assays for dengue virus (DENV). In *Dengue Virus: Methods and Protocols*; Mohana-Borges, R., Ed.; Springer Nature: Berlin/Heidelberg, Germany, 2022; Volume 2409, pp. 3–9.
22. Hierholzer, J.C.; Killington, R.A. Virus isolation and quantitation. In *Virology Methods Manual*; Brian, W.J.M., Hillar, O.K., Eds.; Academic Press: London, UK, 1996; pp. 25–46.
23. R Core Team. *R: A Language and Environment for Statistical Computing*, 4.5.1 ed.; R Foundation for Statistical Computing: Vienna, Austria, 2025.
24. Calvez, E.; Guillaumot, L.; Girault, D.; Richard, V.; O'Connor, O.; Paoaafaite, T.; Teurlai, M.; Pocquet, N.; Cao-Lormeau, V.-M.; Dupont-Rouzeyrol, M. Dengue-1 virus and vector competence of *Aedes aegypti* (Diptera: Culicidae) populations from New Caledonia. *Parasites Vectors* **2017**, *10*, 381. [CrossRef]
25. Richards, S.L.; Anderson, S.L.; Alto, B.W. Vector competence of *Aedes aegypti* and *Aedes albopictus* (Diptera: Culicidae) for dengue virus in the Florida Keys. *J. Med. Entomol.* **2012**, *49*, 942–946. [CrossRef]
26. Mercier, A.; Obadia, T.; Carraretto, D.; Velo, E.; Gabiane, G.; Bino, S.; Vazeille, M.; Gasperi, G.; Dauga, C.; Malacrida, A.R.; et al. Impact of temperature on dengue and chikungunya transmission by the mosquito *Aedes albopictus*. *Sci. Rep.* **2022**, *12*, 6973. [CrossRef]
27. Chepkorir, E.; Lutomiah, J.; Mutisya, J.; Mulwa, F.; Limbaso, K.; Orindi, B.; Ng'ang'a, Z.; Sang, R. Vector competence of *Aedes aegypti* populations from Kilifi and Nairobi for dengue 2 virus and the influence of temperature. *Parasites Vectors* **2014**, *7*, 435. [CrossRef]
28. Diallo, M.; Ba, Y.; Faye, O.; Soumare, M.L.; Dia, I.; Sall, A.A. Vector competence of *Aedes aegypti* populations from Senegal for sylvatic and epidemic dengue 2 virus isolated in West Africa. *Trans. R. Soc. Trop. Med. Hyg.* **2008**, *102*, 493–498. [CrossRef]
29. Amoa-Bosompem, M.; Kobayashi, D.; Itokawa, K.; Murota, K.; Faizah, A.N.; Azerigyik, F.A.; Hayashi, T.; Ohashi, M.; Bonney, J.H.K.; Dadzie, S.; et al. Determining vector competence of *Aedes aegypti* from Ghana in transmitting dengue virus serotypes 1 and 2. *Parasites Vectors* **2021**, *14*, 228. [CrossRef]
30. Chaves, B.A.; Godoy, R.S.M.; Campolina, T.B.; Júnior, A.B.V.; Paz, A.d.C.; Vaz, E.B.d.C.; Silva, B.M.; Nascimento, R.M.; Guerra, M.d.G.V.B.; Lacerda, M.V.G.; et al. Dengue infection susceptibility of five *Aedes aegypti* populations from Manaus (Brazil) after challenge with virus serotypes 1–4. *Viruses* **2022**, *14*, 20. [CrossRef]
31. Gutiérrez-Bugallo, G.; Boullis, A.; Martinez, Y.; Hery, L.; Rodríguez, M.; Bisset, J.A.; Vega-Rúa, A. Vector competence of *Aedes aegypti* from Havana, Cuba, for dengue virus type 1, chikungunya, and Zika viruses. *PLoS Neglected Trop. Dis.* **2020**, *14*, e0008941. [CrossRef] [PubMed]
32. Kobayashi, D.; Kai, I.; Faizah, A.N.; Moi, M.L.; Tajima, S.; Takasaki, T.; Sasaki, T.; Isawa, H. Comparative analysis of the susceptibility of *Aedes aegypti* and Japanese *Aedes albopictus* to all dengue virus serotypes. *Trop. Med. Health* **2023**, *51*, 61. [CrossRef] [PubMed]
33. Dickson, L.B.; Sanchez-Vargas, I.; Sylla, M.; Fleming, K.; Black, W.C.I.V. Vector competence in West African *Aedes aegypti* is Flavivirus species and genotype dependent. *PLoS Neglected Trop. Dis.* **2014**, *8*, e3153. [CrossRef] [PubMed]
34. Fansiri, T.; Fontaine, A.; Diancourt, L.; Caro, V.; Thaisomboonsuk, B.; Richardson, J.H.; Jarman, R.G.; Ponlawat, A.; Lambrechts, L. Genetic mapping of specific interactions between *Aedes aegypti* mosquitoes and dengue viruses. *PLoS Genet.* **2013**, *9*, e1003621. [CrossRef]
35. Lambrechts, L.; Chevillon, C.; Albright, R.G.; Thaisomboonsuk, B.; Richardson, J.H.; Jarman, R.G.; Scott, T.W. Genetic specificity and potential for local adaptation between dengue viruses and mosquito vectors. *BMC Evol. Biol.* **2009**, *9*, 160. [CrossRef]
36. Gloria-Soria, A.; Armstrong, P.M.; Powell, J.R.; Turner, P.E. Infection rate of *Aedes aegypti* mosquitoes with dengue virus depends on the interaction between temperature and mosquito genotype. *Proc. Biol. Sci.* **2017**, *284*, 20171506. [PubMed]
37. Lambrechts, L.; Paaijmans, K.P.; Fansiri, T.; Carrington, L.B.; Kramer, L.D.; Thomas, M.B.; Scott, T.W. Impact of daily temperature fluctuations on dengue virus transmission by *Aedes aegypti*. *Proc. Natl. Acad. Sci. USA* **2011**, *108*, 7460–7465. [CrossRef]

38. Chen, J.; Deng, S.; Peng, H. Insect-specific viruses used in biocontrol of mosquito-borne diseases. *Interdiscip. Med.* **2023**, *1*, e20220001. [CrossRef]
39. Ferreira, Q.R.; Lemos, F.F.; Moura, M.N.; Nascimento, J.O.; Novaes, A.F.; Barcelos, I.S.; Fernandes, L.A.; Amaral, L.S.d.B.; Barreto, F.K.; de Melo, F.F. Role of the microbiome in *Aedes* spp. vector competence: What do we know? *Viruses* **2023**, *15*, 779. [CrossRef]
40. Ramirez, J.L.; Souza-Neto, J.; Torres Cosme, R.; Rovira, J.; Ortiz, A.; Pascale, J.M.; Dimopoulos, G. Reciprocal tripartite interactions between the *Aedes aegypti* midgut microbiota, innate immune system and dengue virus influences vector competence. *PLoS Neglected Trop. Dis.* **2012**, *6*, e1561. [CrossRef] [PubMed]
41. Tsai, C.H.; Chen, T.H.; Lin, C.; Shu, P.Y.; Su, C.L.; Teng, H.J. The impact of temperature and Wolbachia infection on vector competence of potential dengue vectors *Aedes aegypti* and *Aedes albopictus* in the transmission of dengue virus serotype 1 in southern Taiwan. *Parasites Vectors* **2017**, *10*, 551. [CrossRef]
42. Terradas, G.; Manzano-Alvarez, J.; Vanalli, C.; Werling, K.; Cattadori, I.M.; Rasgon, J.L. Temperature affects viral kinetics and vectorial capacity of *Aedes aegypti* mosquitoes co-infected with Mayaro and dengue viruses. *Parasites Vectors* **2024**, *17*, 73. [CrossRef] [PubMed]
43. Bellone, R.; Failloux, A.B. The Role of Temperature in Shaping Mosquito-Borne Viruses Transmission. *Front. Microbiol.* **2020**, *11*, 584846. [CrossRef]
44. Wimalasiri-Yapa, B.; Barrero, R.A.; Stassen, L.; Hafner, L.M.; McGraw, E.A.; Pyke, A.T.; Jansen, C.C.; Suhrbier, A.; Yakob, L.; Hu, W.; et al. Temperature modulates immune gene expression in mosquitoes during arbovirus infection. *Open Biol.* **2021**, *11*, 200246. [CrossRef]
45. Bisht, K.; Te Velthuis, A.J.W. Decoding the role of temperature in RNA virus infections. *mBio* **2022**, *13*, e0202122. [CrossRef] [PubMed]
46. Samuel, G.H.; Adelman, Z.N.; Myles, K.M. Temperature-dependent effects on the replication and transmission of arthropod-borne viruses in their insect hosts. *Curr. Opin. Insect Sci.* **2016**, *16*, 108–113. [CrossRef]
47. Alto, B.W.; Bettinardi, D. Temperature and dengue virus infection in mosquitoes: Independent effects on the immature and adult stages. *Am. J. Trop. Med. Hyg.* **2013**, *88*, 497–505. [CrossRef] [PubMed]
48. Descloux, E.; Mangeas, M.; Menkes, C.E.; Lengaigne, M.; Leroy, A.; Tehei, T.; Guillaumot, L.; Teurlai, M.; Gourinat, A.-C.; Benzler, J.; et al. Climate-based models for understanding and forecasting dengue epidemics. *PLoS Neglected Trop. Dis.* **2012**, *6*, e1470. [CrossRef] [PubMed]