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TEMPORAL AND SPATIAL IMPACTS OF HURRICANE DAMAGE ON WEST NILE VIRUS TRANSMISSION AND HUMAN RISK

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ABSTRACT. Hurricanes have profound impacts on zoonotic pathogen ecosystems that exhibit spatial and temporal waves in both distance from and time since the event. Wind, rain, and storm surge directly affect mosquito vectors and animal hosts of these pathogens. In this analysis, we apply a West Nile virus transmission model parameterized for the Northern coast of the Gulf of Mexico to explore the effect of event timing of hurricane landfall, time since the event, and damage extent on human West Nile virus neuro-invasive disease (WNV-NID) risk. Early-season hurricanes, which make landfall prior to the peak of baseline WNV transmission activity, increase the average total WNV-infectious mosquitoes for the year by 7.8% and human WNV-NID incidence by 94.3% across all areas with hurricane damage. The indirect effects on human exposure to mosquito bites in the immediate aftermath and long-term recovery from the event have strong impacts on the risk of infection. The resultant interactive direct and indirect storm effects on the pathogen system are spatially and temporally heterogenous among the generalized time and space categories modeled.

KEY WORDS Gulf Coast, hurricanes, mathematical model, mosquito, West Nile virus

INTRODUCTION

Tropical cyclones or hurricanes structure coupled wildlife (Cely 1991) and human communities in ways that have profound short- and long-term effects on the zoonotic pathogens that are transmitted between the groups. Few occurrences of increased human transmission of zoonotic arboviruses following a hurricane in the United States have been documented (Nasci and Moore 1998). Caillouët et al. (2008b) demonstrated significant geographic-specific increases in human West Nile virus neuro-invasive disease (WNV-NID) following Hurricane Katrina in 2005 and up to 1 year later.

Although storm-related impacts on mosquito populations are well documented for floodwater nuisance species (Brown 1997, Simpson 2006, Breidenbaugh et al. 2008, Morrow et al. 2010, Lucas et al. 2019), the impact on West Nile virus (WNV) vectors is less well known. Hurricane impacts on vector populations appear to be variable with respect to time since landfall and damage dependent. It is widely hypothesized, but not documented, that adult mosquitoes suffer significant direct mortality from hurricane strength winds (Ahmed and Memish 2017, CDC 2019). Hurricane winds can also expand aquatic larval habitats with the creation of water-holding root ball voids from knocked down trees (Caillouët, personal observation 2006). Flooding caused by storm surge or rainfall may directly impact mosquito larval habitats by salinization in coastal areas, direct destruction, or by expanding available habitats. Barrera et al. (2019) found that populations of the container-inhabiting mosquito, *Aedes aegypti* (L.), doubled following Hurricane Maria due to an increase in water-holding containers. Morrow et al. (2010) documented an increase in *Culex* spp. mosquitoes immediately following tropical storms in Belize. Caillouët et al. (2008b) found storm-surge associated flooding in Hurricane Katrina left thousands of abandoned swimming pools available for habitat expansion of the WNV vector, *Culex quinquefasciatus* (Say). Huang (2019), in a non–peer reviewed thesis, demonstrated population decreases of *Cx. quinquefasciatus*, in flooded areas immediately following Hurricane Maria, but widespread long-term increases in vector populations. Finally, human recovery activities, including trash piles, may promote larval habitat expansion by inhibiting drainage and creating a proliferation of water-holding containers (Caillouët, personal observation 2006).

West Nile virus avian host communities also suffer variable hurricane-related damage and time-dependent effects (Rittenhouse et al. 2010). Hurricane-interrupted nest attempts may cause direct destruction of nests or a dearth of available food for nestlings and hatch-year birds. Near local extinction of resident urban birds following Hurricane Katrina was documented in flooded areas of New Orleans (Yaukey 2008). The locally important WNV avian host, the Northern Cardinal, was half as abundant as prior to Hurricane Katrina for up to 3.4 years following the event (Yaukey 2012). Reductions in WNV avian hosts could increase mosquito bites on the few remaining birds, potentially exacerbating short-term arbovirus amplification (Foppa et al. 2011, Caillouët et al. 2013a, Krebs et al. 2014). Given the sensitivity of WNV to avian host community assemblages (Ezenwa et al. 2006, Hamer et al. 2011), hurricane-related environmental impacts that affect certain bird species may dramatically alter

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local WNV transmission. Wind driven changes to the forested canopy cover led to changes in avian species composition in the Pearl River basin following Hurricane Katrina (Brown et al. 2011). Though unique given the prolonged flooding of New Orleans, studies of other hurricanes have documented significant impacts on a variety of bird species in diverse habitats. Coastal bird species appear to be particularly vulnerable to hurricanes (Cely 1991, Marsh and Wilkinson 1991, Raynor et al. 2013). Perdomo-Velázquez et al. (2017) found significant reductions in understory forest birds in Yucatan, Mexico, for up to 3 years following consecutive hurricanes Wilma and Emily.

In their review documenting the rarity of natural disasters to cause or exacerbate arboviral epidemics, Nasci and Moore (1998) outline the potential circumstances necessary for hurricanes and floods to increase human transmission risk. The authors highlighted the role of increased vector abundance "coupled with expanded human exposure to mosquitoes after a disaster" (Nasci and Moore 1998). Increased human exposure to mosquito bites was hypothesized as a mechanism partly responsible for the observed increase in WNV neuro-invasive disease following Hurricane Katrina, but it was not documented (Caillouët et al. 2008b). Increases in human exposure to mosquitoes following hurricanes is largely inferred from reports of nuisance mosquito landings and housing (CDC 1993, CDC 2019). One recent study by Seger et al. (2019) reported that 87% of households noted an increase in mosquito biting during the response period from Hurricanes Irma and Maria in the US Virgin Islands.

There is little doubt that the ecological dynamics between animal hosts, mosquito vectors, and humans are significantly altered by hurricane-associated winds, rainfall, flooding, and preparatory or repair activities. We hypothesize that hurricane-related effects on arbovirus transmission can be expected to vary based on the damage extent (distance is a proxy) and the time since the hurricane event. In addition, we hypothesize that the timing of the hurricane event within the arboviral season has profound impacts on whether human transmission risk increases or decreases. We modified a previously described WNV mathematical transmission model (Robertson and Caillouët 2016) to simulate the effects of a major hurricane making landfall along the Northern Coast of the Gulf of Mexico of the United States on WNV human incidence. Specifically, we compared storm effects across regions differentially impacted by the hurricane at multiple time periods in relation to landfall. Finally, we analyzed the differential human WNV case outcomes for hurricanes making landfall at different times throughout the WNV transmission season.

MATERIALS AND METHODS

Model development

We use a mathematical model based on those described in Robertson and Caillouët (2016) and Beebe and Robertson (2017) to simulate the dynamics of WNV over a single year in each type of damage-affected region. The primary vector of interest is *Cx. quinquefasciatus*, which is an opportunistic species that feeds on avian hosts, humans, and other nonhuman mammals. The percentage of bites upon each host type is highly variable between studies (Takken and Verhulst 2013) and likely to depend upon the relative densities and exposures of available hosts. Birds are the primary definitive host for WNV, with species varying greatly in competence or their ability to transmit the pathogen (Komar et al. 2003, Kilpatrick et al. 2007). Here we consider 2 representative species of avian hosts that vary in competence, assuming one species is twice as likely to transmit WNV to susceptible mosquitoes when bitten as the other. For each host species, we model 2 age classes, juvenile (hatch-year birds, not able to reproduce) and reproductive adults (birds returning from the previous season), assuming no age differences in transmission-related parameters for simplicity. Adults produce juveniles according to a Gaussian recruitment curve, with parameters chosen so newly hatched birds, or nestlings, are concentrated during the period between March 15 (day 74) and August 10 (day 222) to align with observed nesting curves in St. Tammany Parish (Caillouët, personal observation). We note that Robertson and Caillouët (2016) and Beebe and Robertson (2017) considered a nestling/juvenile period with a mean duration of 2 weeks, so maturation to an older stage was modeled. Since our juvenile stage includes all hatch-year birds, which will not become reproductive adults until the year after they are born, the maturation process is not included in our model. Susceptible juveniles and adults of either avian species (J_S, $\hat{J}_S$, A_S, $\hat{A}_S$) become infected with probability β (species 1) or $\hat{\beta}$ (species 2) upon being bitten by an infectious mosquito (M_I) and move into the respective infectious class (J_I, $\hat{J}_I$, A_I, $\hat{A}_I$). Most competent avian species will develop high enough levels of viremia to become infectious within a day of being bitten by an infectious mosquito (Komar et al. 2003); therefore, we do not include a latent class. Infectious birds recover at rate γ (species 1) or $\hat{\gamma}$ (species 2) into the respective recovered class (J_R, $\hat{J}_R$, A_R, $\hat{A}_R$). All classes of birds are subject to natural mortality at rate μ (species 1) or $\hat{\mu}$ (species 2). While humans are noncompetent, or dead-end hosts, susceptible humans (H_S) develop WNV neuro-invasive disease with probability β_H if bitten by an infectious mosquito, moving to the infected (but not infectious) class H_I. Since we are only interested in the total number of human cases in a year, rather than when they occur, we do not

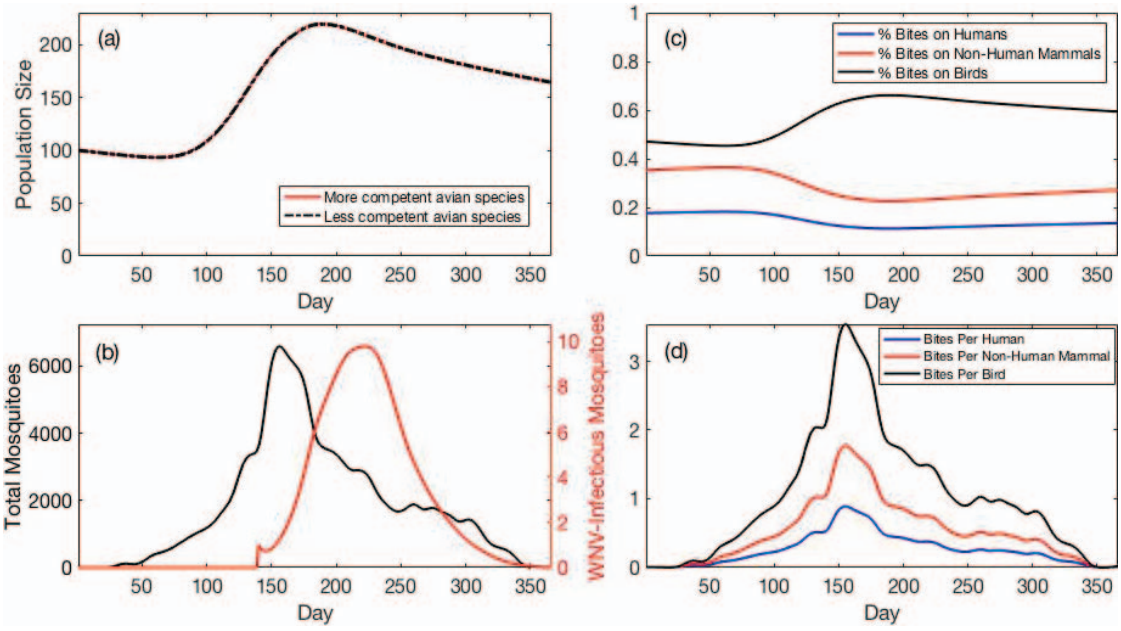

Fig. 1. Modeled host populations and West Nile virus transmission outcomes for a baseline year without a hurricane. Plots (a) and (b) show the total avian and vector population sizes over the year, along with the number of WNV-infectious mosquitoes. Plot (c) shows the percentage of all mosquito bites on each type of host, while (d) shows the corresponding per capita biting rate for each host type.

model a latent period or recovery; the number of infected humans at the end of the year represents the total number of human cases. We account for the presence of other alternative hosts with a parameter representing total density of other nonhuman mammals (O_T) whose value may be affected by the hurricane.

While the models in Robertson and Caillouët (2016) and Beebe and Robertson (2017) assume logistic growth of the vector population, here total vector density $V(t)$ is determined by a smoothed spline (in MATLAB) of the weekly average *Cx. quinquefasciatus* abundance per trap night collected in St. Tammany Parish from 2009 to 2018, scaled by a factor of 50 (Caillouët, unpublished data; shown in Fig. 1b). Negative values resulting from the smoothing were replaced with zero. We assume vectors bite at a constant rate (a) not limited by host availability and that the distribution of bites among hosts varies according to the relative densities of each host type and their exposure coefficients according to the following functions:

$$\alpha_B(t) = \frac{a_B B_T(t)}{a_B B_T(t) + a_H H_T(t) + a_O O_T(t)}$$

$$\alpha_H(t) = \frac{a_H H_T(t)}{a_B B_T(t) + a_H H_T(t) + a_O O_T(t)}$$

where α_B is the fraction of total bites that are on birds, and α_H is the fraction of total bites that are on humans. These fractions are between 0 and 1 and determined both by host densities B_T (total birds), H_T (total humans), and O_T (total other nonhuman mammals), as well as the exposure coefficient of each type of host, where a_B, a_H, and a_O denote the exposure coefficients of birds, humans, and other nonhuman mammals, respectively. Exposure coefficients are assumed constant in the absence of a hurricane. As a baseline, we take $a_B = 4$, $a_H = 1$, and $a_O = 2$, so the per capita biting rate on birds is twice that on other nonhuman mammals, and 4 times as great as on humans. Fluctuations in mosquito bites on birds mirror changes in the avian host total population size, which increases as juvenile birds are produced and decreases due to natural and disease mortality. The per capita biting rate on each host type changes throughout the year with the total number of mosquitoes, but birds always receive twice as many bites as nonhuman mammals and 4 times as many bites as humans. While vector feeding preferences may exist for certain avian species and/or ages and impact transmission (Robertson and Caillouët 2016, Beebe and Robertson 2017), we do not incorporate them here. Susceptible vectors (M_S) become infected with probability δ or $\hat{\delta}$ after biting an infected avian host of species 1 or 2, respectively, and move into the exposed or latent class (M_L). Latent vectors become infectious vectors (M_I) at rate k, determined by the average length of the extrinsic incubation period of the disease. Both latent and infectious vectors have natural mortality rate μ_M. Susceptible vectors are

determined by subtracting the latent and infectious vector population sizes from $V(t)$. The model equations are given by

$$\frac{dJ_s}{dt} = b(t)A_T - a\alpha_B\beta M_I\frac{J_s}{B_T} - \mu J_s$$

$$\frac{dJ_I}{dt} = a\alpha_B\beta M_I\frac{J_s}{B_T} - \gamma J_I - (\upsilon+\mu)J_I$$

$$\frac{dJ_R}{dt} = \gamma J_I - \mu J_R$$

$$\frac{dA_s}{dt} = -a\alpha_B\beta M_I\frac{A_s}{B_T} - \mu A_s$$

$$\frac{dA_I}{dt} = a\alpha_B\beta M_I\frac{A_s}{B_T} - \gamma A_I - (\upsilon+\mu)A_I$$

$$\frac{dA_R}{dt} = \gamma A_I - \mu A_R$$

$$\frac{d\hat{J}_s}{dt} = b(t)\hat{A}_T - a\alpha_B\hat{\beta}M_I\frac{\hat{J}_s}{B_T} - \hat{\mu}\hat{J}_s$$

$$\frac{d\hat{J}_I}{dt} = a\alpha_B\hat{\beta}M_I\frac{\hat{J}_s}{B_T} - \hat{\gamma}\hat{J}_I - (\hat{\upsilon}+\hat{\mu})\hat{J}_I$$

$$\frac{d\hat{J}_R}{dt} = \hat{\gamma}\hat{J}_I - \hat{\mu}\hat{J}_R$$

$$\frac{d\hat{A}_s}{dt} = -a\alpha_B\hat{\beta}M_I\frac{\hat{A}_s}{B_T} - \hat{\mu}\hat{A}_s$$

$$\frac{d\hat{A}_I}{dt} = a\alpha_B\hat{\beta}M_I\frac{\hat{A}_s}{B_T} - \hat{\gamma}\hat{A}_I - (\hat{\upsilon}+\hat{\mu})\hat{A}_I$$

$$\frac{d\hat{A}_R}{dt} = \hat{\gamma}\hat{A}_I - \hat{\mu}\hat{A}_R$$

$$\frac{dM_L}{dt} = a\alpha_B\left(\delta\left(\frac{J_s+A_s}{B_T}\right) + \hat{\delta}\left(\frac{\hat{J}_s+\hat{A}_s}{B_T}\right)\right)M_S - kM_L - \mu_M M_s$$

$$\frac{dM_I}{dt} = kM_L - \mu_M M_I$$

$$\frac{dH_s}{dt} = -a\alpha_H\beta_H M_I\frac{H_S}{H_T}$$

$$\frac{dH_I}{dt} = a\alpha_H\beta_H M_I\frac{H_S}{H_T}$$

$$M_s = V(t) - M_L - M_I$$

where B_T denotes the total bird population, H_T denotes the total human population, and

$$b(t) = \frac{f}{\sigma\sqrt{2\pi}}e^{-\frac{(q-t)^2}{2\sigma^2}}$$

Baseline parameters are given in Table 1.

All simulations are run in Matlab using ode45, starting on Julian day 1 (January 1) with 100 adult birds of each species, 10% recovered and 90% susceptible, as well as 300 susceptible humans and 300 other nonhuman mammals. Total human density and other nonhuman mammal densities remain constant unless otherwise noted. One infectious vector is introduced into the population on day 140 to align with the timeframe when WNV-infected mosquito pools are typically first observed in St. Tammany Parish (Caillouët, unpublished data). The model is run through day 365.

Study design

We separately consider WNV transmission models for the hurricane-impacted geographic areas including: significantly damaged areas 1) with prolonged flooding and 2) without prolonged flooding, 3) an area experiencing moderate damage, and 4) areas that experience no hurricane-related damage but experienced an influx of evacuees. Each area has associated changes that occur at the time of impact and last for a specific period following the storm (Table 2). These changes are implemented as an instantaneous modification to parameters or state variables. For Gulf Coast hurricanes, historic landfall dates vary between Julian day 167 (~June 16) and 277 (~October 4), where day 1 is January 1. Landsea (1993) reported the median date of landfall of hurricanes for the Gulf Coast of the United States as day 248 (September 5). We consider 4 potential dates of landfall spread over the season: 190 (~July 9), 219 (~August 7), 248 (~September 5), and 277 (~October 4), as well as a null of no hurricane for comparison. The number of WNV-infectious mosquitoes over the year as well as the human per capita biting rate that results from a storm on each date are shown in Fig. 2 for each geographic area. In Fig. 3 we show the total number of WNV-infectious mosquitoes during the season (counting a mosquito each day it is alive and infectious), and total number of WNV-NID human cases per 100,000 person-years for each geographic area and date of landfall. The latter measurement accounts for differential changes in human density that occur in a given area during the year due to hurricanes and serves as a standardized measure of human risk. It is computed as 100,000 × total human cases/total susceptible person-years.

Table 1. Baseline (no-hurricane) West Nile virus transmission model parameters.

Parameter	Description	Baseline value
a	Vector biting rate	1/3 (Wonham et al. 2004)
a_B	Exposure coefficient: birds	4
a_H	Exposure coefficient: humans	1
a_O	Exposure coefficient: other mammals	2
β	Avian susceptibility (Species 1)	1
$\hat{\beta}$	Avian susceptibility (Species 2)	1
δ	Avian infectivity (Species 1)	0.4
$\hat{\delta}$	Avian infectivity (Species 2)	0.2
γ	Avian recovery rate (Species 1)	1/3 day^{-1}
$\hat{\gamma}$	Avian recovery rate (Species 2)	1/3 day^{-1}
υ	Avian disease mortality (Species 1)	0.1 day^{-1}
$\hat{\upsilon}$	Avian disease mortality (Species 2)	0.1 day^{-1}
μ	Avian natural mortality (Species 1)	0.0014 day^{-1} (Simpson et al. 2012)
$\hat{\mu}$	Avian natural mortality (Species 2)	0.0014 day^{-1} (Simpson et al. 2012)
f	Scaling parameter for juvenile recruitment curve	1.9 (Caillouët et al. 2013b)
q	Mean of juvenile recruitment curve	134
σ	Standard deviation of juvenile recruitment curve	30
k	Virus extrinsic incubation period	0.106 day^{-1} (Wonham et al. 2004)
μ_M	Mosquito natural mortality	0.096 (Jones et al. 2012)
β_H	Human WNV-NID susceptibility	1/150 (CDC 2019)

To explore the impact of a hurricane on WNV transmission in the following year for each geographic area, we also simulate the WNV transmission model with the *Year after storm* assumptions from Table 2 for the median landfall date of September 5 (day 248). The initial conditions are modified so the initial percentage of recovered avian hosts reflects the composition of the population at the end of the previous year, with eliminated species assumed to return at the baseline recovered percentage of 10%. Total WNV-infectious mosquitoes and WNV-NID human incidence are recorded for each geographic area in Table 3, along with the percentage change from the outcomes expected the year following a season with no hurricane.

Hurricane transmission model forcings: year of storm

Significant damage area with flooding: For this area, we assume that 75% of existing susceptible, latent, and infectious vector populations are killed at the time of the hurricane, and the total vector population $V(t)$ is reduced to 25% of normal for 4 wk. The more competent avian species is reduced by 50% at the time of the storm, while the less competent avian species is eliminated. Juvenile and adult birds are affected equally. If the storm is prior to the end of the avian nesting season (determined by the function $b(t)$), additional juvenile birds can be produced by the surviving adults. Other nonmammal hosts are reduced by 50%. The susceptible human population is reduced by 75% at the time of the storm due to evacuation and mortality, and the exposure coefficient a_H is increased from 1 to 3. After 4 wk, the vector population $V(t)$ is increased to 125% of normal for the rest of the season. At this time, half the human evacuees return as susceptible and the exposure coefficient a_H is reduced from 3 to 2.

Significant damage area without flooding: For this area, we assume that 50% of existing susceptible, latent, and infectious vector populations are killed at the time of the hurricane, and the total vector population $V(t)$ is reduced to 50% of normal for 2 wk. The more WNV competent avian species is reduced by 25% at the time of the storm, while the less competent avian species is reduced by 75%. Other nonmammal hosts are reduced by 25%. The susceptible human population is reduced by 50% at the time of the storm, and the exposure coefficient a_H is increased from 1 to 2. After 2 wk, the vector population $V(t)$ is increased to 125% of normal for the rest of the season. At this time, half the human evacuees return as susceptible, and the exposure coefficient a_H is reduced from 2 to 1.5.

Moderate damage area: For this area, we assume that 25% of existing susceptible, latent, and infectious vector populations are killed at the time of the hurricane, and the total vector population $V(t)$ is reduced to 25% of normal for 1 wk. The more competent avian species is reduced by 10% at the time of the storm, while the less competent avian species is reduced by 50%. Other nonmammal hosts are reduced by 10%. The susceptible human population is increased by 25% at the time of the storm, and the exposure coefficient a_H is increased from 1 to 1.5. After 1 wk, the vector population $V(t)$ is increased to 125% of normal for the rest of the season. Half the human evacuees leave, and the exposure coefficient a_H is reduced from 1.5 to the baseline value of 1.

No-damage area: For this area, vectors and other nonmammal hosts are not affected by the storm. The susceptible human population is increased by 25% at the time of the storm due to evacuees from the significant and moderately damaged areas. The

Table 2. Hurricane-impacted damage and year of event specific parameter forcing for vectors, definitive hosts, other mammal hosts, and humans.

Time period	Vectors	Avian hosts	Nonhuman mammals	Humans
Significant damage area with flooding				
Year of storm	Immediately reduced to 0.25× normal; 1.25× normal after 4 weeks	More WNV competent species 0.5× normal; less WNV competent species eliminated	0.5× normal	0.25× normal; exposure 3× normal; after 4 weeks half evacuees return, exposure 2× normal
Year after storm	2× normal	More WNV competent species 0.75× normal; less WNV competent species 0.25× normal	0.75× normal	0.75× normal; exposure 2 × normal
Significant damage area no flooding				
Year of storm	Immediately reduced to 0.5× normal; 1.25× after 2 weeks	More WNV competent species 0.75× normal; less WNV competent species 0.25× normal	0.75× normal	0.5× normal; exposure 2× normal; after 2 weeks half evacuees return, exposure 1.5× normal
Year after storm	1.5× normal	More WNV competent species 0.9× normal; less WNV competent species 0.5× normal	0.9× normal	0.9× normal; exposure 1.5×
Moderate damage area				
Year of storm	Immediately reduced to 0.75× normal; 1.25× normal for season after 1 week	More WNV competent species 0.9× normal; less WNV competent species 0.5× normal	0.9× normal	1.25× normal; exposure 1.5× normal; after 1 week half evacuees leave, exposure normal
Year after storm	1.25× normal	More WNV competent species no change; less WNV competent species 0.75× normal	No change	1.1× normal; exposure normal
No-damage area				
Year of storm	No change	No change	No change	1.25× normal; exposure normal
Year after storm	No change	No change	No change	1.1× normal; exposure normal

population remains elevated for the rest of the season, but there is no change in human exposure.

Hurricane transmission model forcings: year after storm

Significant damage area with flooding: The year after the storm the vector population $V(t)$ is 2 times greater than baseline in this area. More competent avian adults return at 75% of their baseline levels, while less competent avian adults return at 25% of baseline. Nonhuman mammals and humans are assumed to be at 75% of their baseline levels, and the human exposure coefficient a_H is increased from 1 to 2.

Significant damage area without flooding: The year after the storm the vector population $V(t)$ is 1.5 times greater than baseline in this area. More competent avian adults return at 90% of their baseline levels, while less competent avian adults return at 50% of baseline. Nonhuman mammals and humans are assumed to be at 90% of their baseline levels, and the human exposure coefficient a_H is increased from 1 to 1.5.

Moderate damage area: The year after the storm the vector population $V(t)$ is 1.25 times greater than baseline in this area. More competent avian adults return at their baseline levels, while less competent avian adults return at 50% of baseline. Nonhuman mammals are assumed to be at their baseline levels, and human populations are increased to 110% of baseline with no increase in human exposure.

No-damage area: Vector, avian, and nonhuman mammal populations are all at baseline levels. Human populations are increased to 110% of baseline with no increase in human exposure.

RESULTS

Simulations of the WNV transmission model with parameters in Table 1 result in a no-hurricane baseline of 870 WNV-infectious mosquitoes and 77.1 human cases of WNV-NID per 100,000 susceptible human-years (Fig. 1). The percentage of mosquito bites on

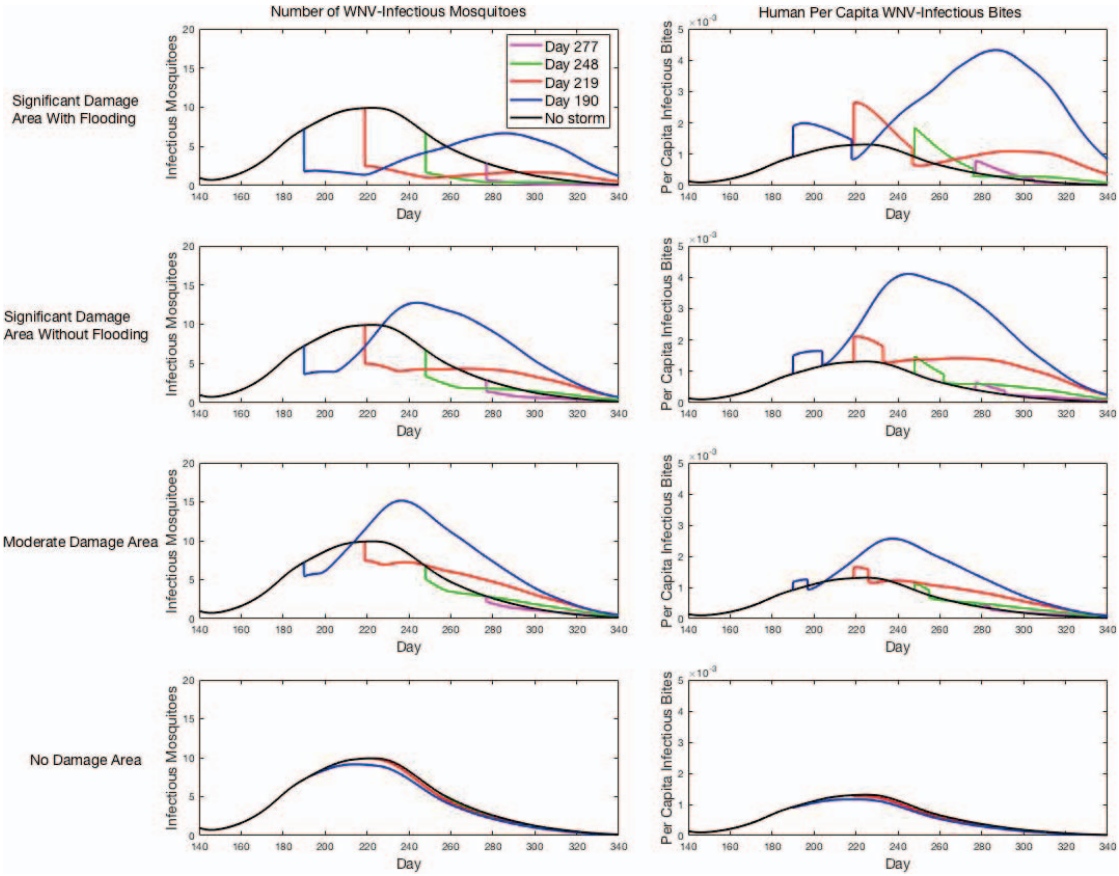

Fig. 2. The number of WNV-infectious mosquitoes over the year (left column) and human per capita biting rate (right column) resulting from a storm on Julian day 190 (blue lines), 219 (red lines), 248 (green lines), or 277 (pink lines) for each category of damage. Similar increases in human WNV risk (per capita WNV-infectious bites) are noted in all hurricane damaged areas for early-season storms.

avian hosts ranges from 45% to 67%, similar to reports from the Northern Gulf Coast region (Mackay et al. 2010). Results for transmission-related model outcomes are given in Figs. 2 and 3.

WNV transmission across hurricane-impacted geography

For the significant damage area with flooding, all 4 landfall dates considered result in fewer WNV-infectious mosquitoes over the season compared with simulations of no storm. In the most severely damaged area with flooding, the peak number of WNV-infectious mosquitoes never rises above baseline levels. However, per capita annual infectious bites on humans are elevated following a hurricane (Fig. 2), with the highest biting rates occurring for early landfall (day 190). Human WNV-NID incidence correspondingly increases relative to a storm-free year for all dates of landfall and is greatest for

Table 3. Significant increases in human West Nile virus risk in the year after the hurricane landfall are observed in areas experiencing the most infrastructure damage compared with a second-year baseline with no storm event the year prior. Year 2 effects were observed with a hurricane landfall during the year prior at the peak of the Gulf Coast hurricane season: September 5 (Julian day 248).

Hurricane-impacted geography	Total WNV-infectious mosquitoes (% change from no hurricane)	WNV-NID human incidence[1] (% change from no hurricane)
Significant damage area with flooding	5,845.6 (935.0)	1,597.9 (3,091.7)
Significant damage area no flooding	3003.9 (431.9)	498.3 (906.7)
Moderate damage area	1,399.3 (147.8)	152.8 (208.7)
No-damage area	519.1 (−8.1)	44.9 (−9.3)

[1] Number of human *West Nile virus* neuro-invasive disease cases/100,000 person-years.

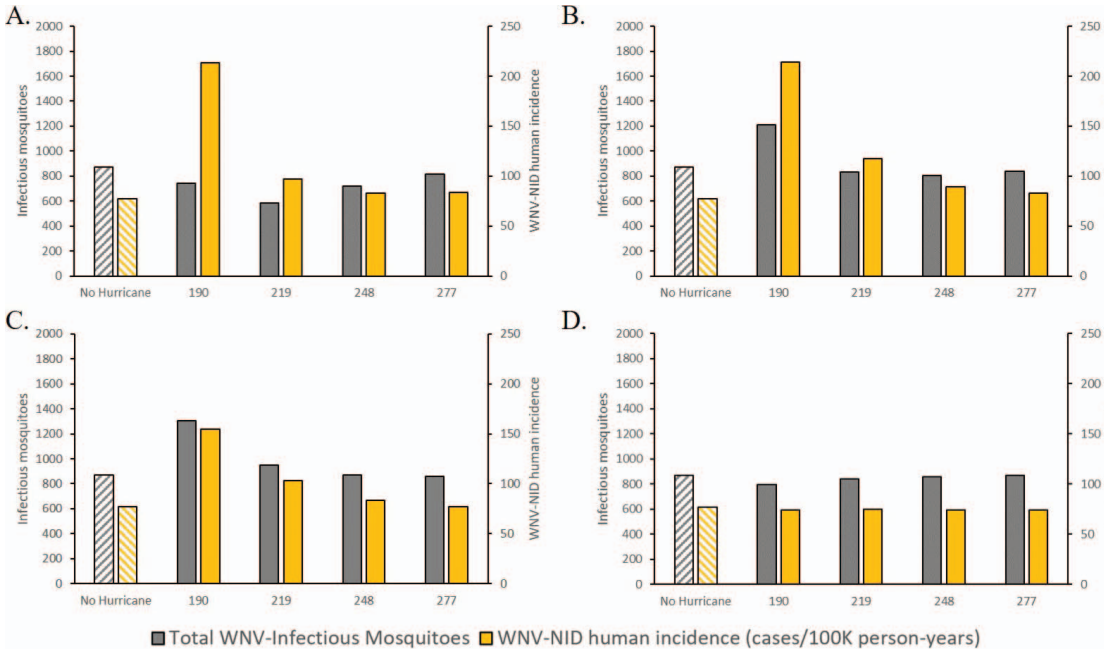

Fig. 3. Early hurricane landfall dates exacerbate West Nile virus neuro-invasive disease (WNV-NID) incidence, but not necessarily WNV-infectious mosquitoes in areas experiencing infrastructure damage during the year of the storm event. Sum of WNV-infectious mosquitoes (gray) and WNV-NID incidence (yellow) for the year of the storm event by landfall Julian day for hurricane-impacted geographic regions: (A) significant damage area with flooding, (B) significant damage area no flooding, (C) moderate damage area, (D) no-damage area.

early-season landfall (day 190). Median (Julian day 248; ~September 5) or later landfall dates result in less than 7 cases per 100,000 person-years above the no-storm baseline.

For the significant damage area without flooding, there are increased numbers of vectors and competent hosts poststorm compared with the significant damage area with flooding, along with a less severe reduction in human hosts and less of an exposure increase. Consequently, a hurricane making landfall on day 190 results in 471 more infectious mosquitoes over the season than when there is prolonged flooding yet increases WNV-NID human incidence by less than 1 case (0.4%). Levels of WNV-infectious mosquitoes for an early landfall storm (day 190) are highest in the moderate damage area (90 over the significant damage without flooding area and 434 over baseline level). Human WNV-NID incidence for an early landfall storm (day 190) is elevated here relative to baseline but is less than both the significant damage areas.

For the median hurricane landfall date of September 5 (day 248), the total number of infectious mosquitoes increases as damage becomes less severe (for the 3 areas incurring damage). All 3 areas have lower than baseline levels of total infectious mosquitoes and above baseline human incidence. The no-damage area has more infectious vectors than the significant damage areas, but they are still under baseline, along with incidence levels.

Among the 3 damage-affected areas, early landfall storms (day 190) lead to the highest human WNV-NID incidence in the significant damage area without flooding (though the significant damage with flooding is only slightly lower), and lowest incidence is in the moderate damage area. For midseason landfall (day 219 and 248), highest incidence occurs in the significant damage area without flooding, and lowest occurs in the significant damage area with flooding. For storms making landfall late in the season, the highest incidence is in the significant damage area with flooding, with lowest risk in the moderate damage area.

In the no-damage area, vectors are not affected, but the human population is increased after a hurricane. Here both total infectious mosquitoes and human incidence are lowered compared with baseline for all dates of landfall. Though raw numbers of human cases increase, per capita risk is lowered given the influx of hurricane evacuees. Increasing human density results in more bites on humans as a population but also lowers the number of bites on WNV competent hosts.

Effect of hurricane landfall date on WNV transmission

Hurricanes making landfall prior to the peak of WNV activity in mosquitoes significantly alter transmission activity in mosquitoes and WNV-NID

human incidence. Early-season hurricanes making landfall on Julian day 190 (~July 9) and day 219 (~August 7) increase the average total WNV-infectious mosquitoes for the year by 7.8% across all areas with hurricane damage. Meanwhile, early-season hurricanes across all damage areas increase WNV-NID human incidence by 94.3%. Later season landfall dates (Julian days 248 and 277; ~September 5 and October 4, respectively) result in an average reduction in WNV-infectious mosquitoes of 6.1% and average reduction in WNV-NID human incidence of 7.8% across areas with damage relative to baseline no-hurricane models (see Fig. 3).

Long-term hurricane impacts on WNV transmission

Lingering effects into the year after the hurricane, including increased vectors, reduced avian hosts, and increased human exposure, result in significantly higher WNV transmission measures and human incidence in a positive association with the level of damage (Table 3). Modeled raw WNV-NID human cases are 24.0-fold higher in the most damaged area with flooding compared with the second-year baseline and 9.0-fold higher in the next most damaged area. Moderately damaged areas experience a 3.4-fold increase, while no-damage areas have slightly lower WNV-NID case counts. Despite high relative increases in human case counts across damaged areas, raw case counts are still in the low single digits given the rarity of WNV-NID. Substantially larger increases of 32.3 and 10.1-fold are noted when measuring WNV-NID human incidence in significant damage areas considering the change in underlying population denominators given hurricane-induced shifts in demography.

Effect of WNV competent avian species elimination

Presented hurricane effects on the WNV ecosystem across damage areas and for different landfall dates assume a differential negative impact on the less WNV competent avian species. For the significant damage area with prolonged flooding, the more competent species is reduced by 50%, while the less competent species is eliminated, for an overall avian population reduction of 75%. To further assess the effect of differential avian host reduction at the time of the hurricane on total WNV-infectious mosquitoes and WNV-NID human incidence, we compare the results from the assumptions in Table 2 to the cases where: 1) the less WNV competent species is reduced by 50% and the more competent species eliminated and 2) both species are reduced by 75% (Fig. 4). Both measures of risk are greatest when the less competent species is eliminated and lowest when the more competent species is eliminated, with the difference between them decreasing as the date of hurricane landfall moves later in the year. The total number of WNV-infectious vectors is reduced compared with the no-hurricane baseline in all cases except when the less competent species is eliminated and there is very early-season landfall. When the less WNV competent species is eliminated, human risk (as measured by WNV-NID human incidence) is always elevated compared with the no-hurricane baseline, with greatest risk for early-season landfall. When both species are reduced equally, human risk is elevated for early or late season landfalls but reduced for midseason landfall dates. When the more competent species is eliminated, risk is lowest for early-season landfall and increases as the landfall date moves later in the year.

Effect of vector abundance

The vector population in our baseline analysis is fit to mosquito trap data from St. Tammany Parish, LA. The related WNV transmission model in Robertson and Caillouët (2016) assumes vectors have logistic growth, and the peak number of infectious vectors was found to be highly sensitive to the vector carrying capacity. Here we have scaled the trap counts by a factor of 50, and our baseline outcomes are 870 total WNV-infectious mosquitos and 77 WNV-NID human cases per 100,000 susceptible person-years. These outcomes increase with scaling factor, which determines overall vector abundance (Fig. 5). A scaling factor of 25 changes the baseline outcomes to 66 WNV-infectious mosquitoes and human WNV-NID incidence of 6, while a scaling factor of 75 results in 3,556 WNV-infectious mosquitoes and human WNV-NID incidence of 330 in a year with no storm.

To further explore how vector abundance affects our results, we compare the outcomes of total WNV-infectious mosquitoes and human WNV-NID incidence, for each date of landfall and geographic impact area, for scaling factors of 10% above and below 50 (Fig. 6). While both outcome measures increase with scaling factor for all scenarios, the shapes of the curves are not significantly affected.

DISCUSSION

The impact of a major hurricane on WNV transmission and human risk of infection is dependent on date of landfall in relation to the WNV season and the extent of damage impacts to vectors, animal hosts, and human exposure. In this study, the worst-case scenario of the highest human WNV-NID cases and incidence occurs when a hurricane makes landfall early in the hurricane season in the significantly damaged area without flooding. Fortunately, the peak of hurricane landfalls along the Northern Coast of the Gulf of Mexico occurs after peak WNV transmission season (Landsea 1993).

As hurricanes structure vector, avian, human, and other mammal populations differentially for several

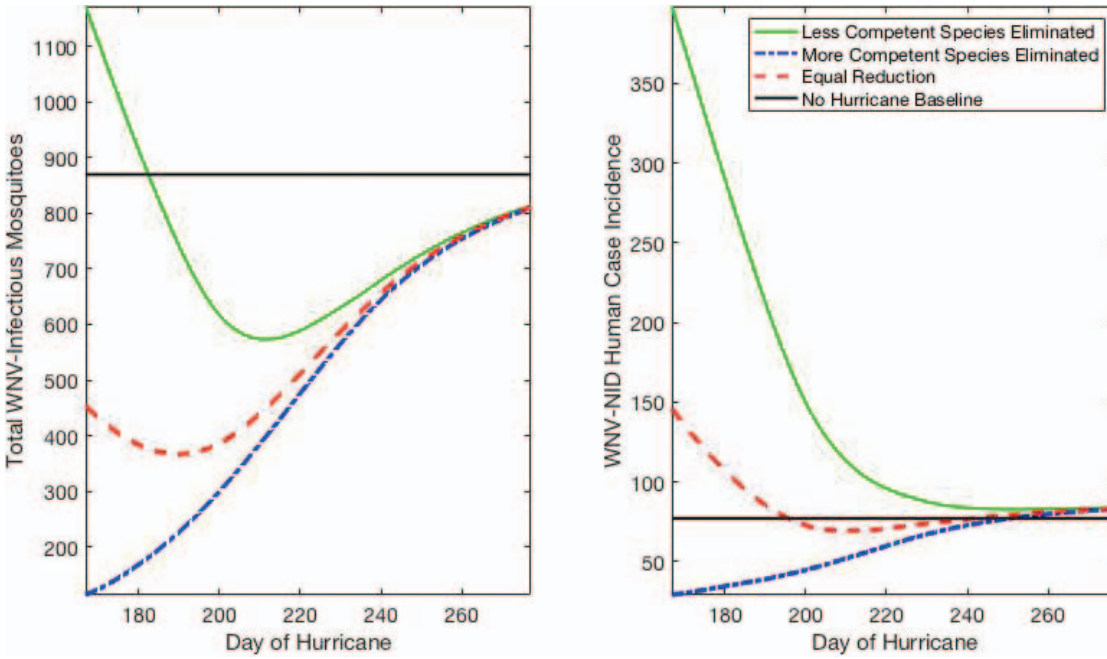

Fig. 4. Effect of reduced overall bird population (dashed red lines) and the concentration of bites onto fewer birds equally, compared with the reduction of a single species (more competent or less competent). If hurricanes negatively impact bird species with higher West Nile virus host competency (dashed blue lines), transmission intensity and human case incidence is lowered due to increased bites on a less competent species. Conversely when the less WNV competent bird species (green line) is limited, WNV risk is heightened.

years following landfall, alterations to WNV transmission can be expected to manifest in both short- and long-term changes. The degree of resiliency in these populations likely determines the temporal extent of alterations to this pathogen's ecosystem. This analysis considered hurricane impacts in the year following a storm event and noted significant increases in latent WNV risk.

Hurricane-related changes to human populations and separately their exposure to mosquito bites play a large role in WNV risk to people. This study observed significant increases in WNV-NID human

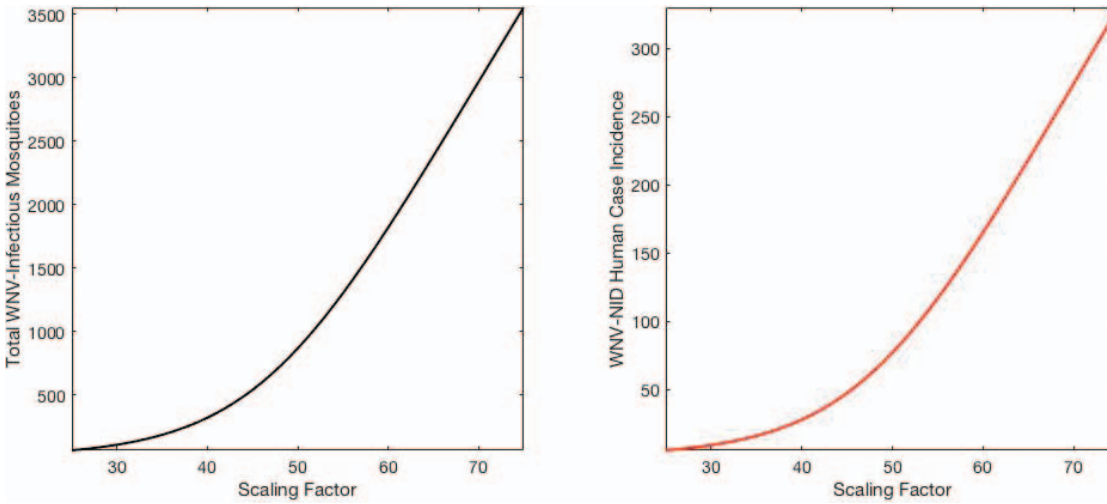

Fig. 5. Effect of vector abundance scaling factor on baseline values for total WNV-infectious mosquitoes and WNV-NID human cases per 100,000 person-years in years with no hurricane. Both measures of transmission increase with scaling factor.

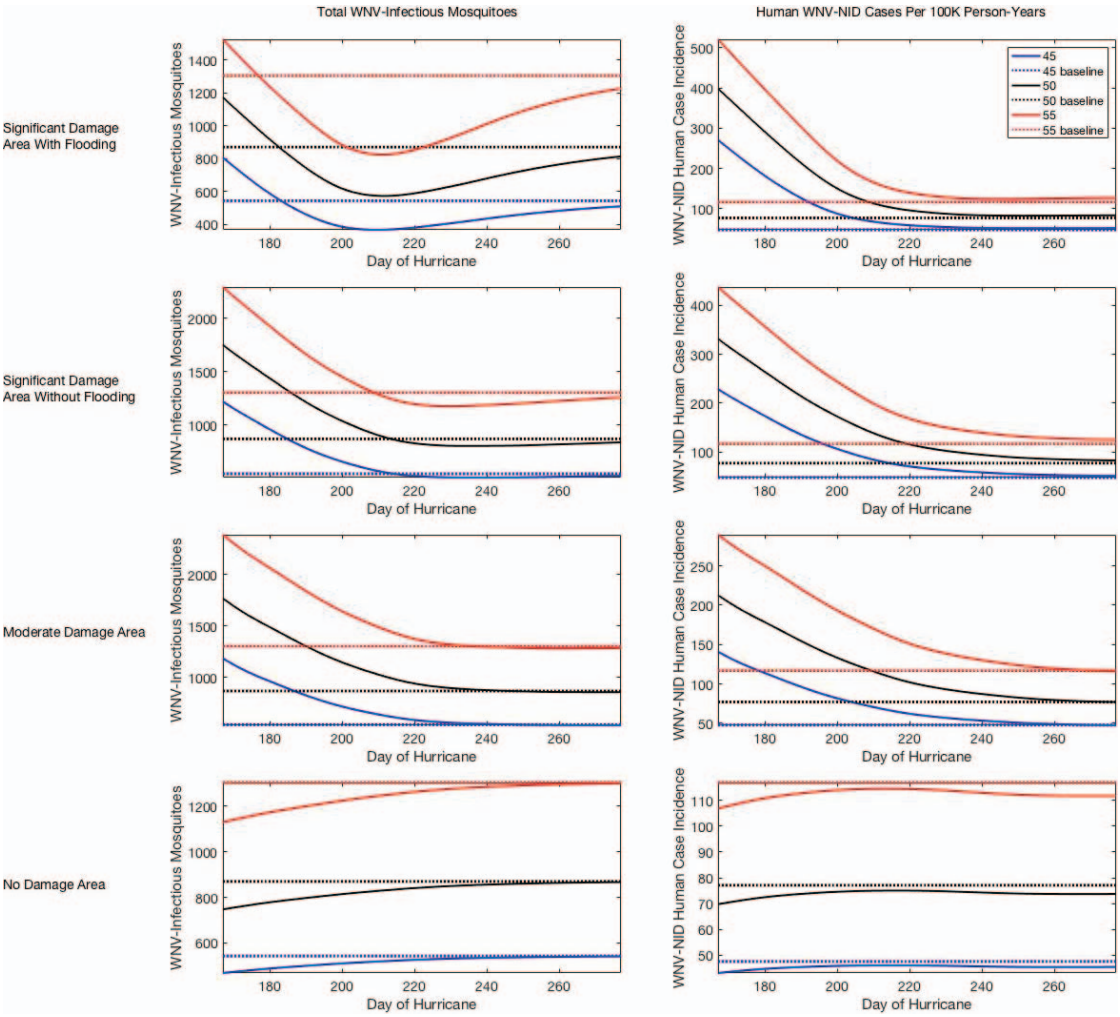

Fig. 6. Effect of vector abundance on total WNV-infectious mosquitoes and human WNV-NID incidence. Outcomes resulting from a storm with landfall dates between day 167 and 177 for each hurricane-impacted geographic area, along with their no-storm baselines, are shown for scaling factors of 45, 50, and 55. Modifying the scaling factor by ±10% results in increased or decreased magnitude of outcome measures, but the shapes of the curves are not significantly altered.

incidence in the early hurricane season scenario in damaged areas with flooding even when the total WNV-infectious mosquito number was lower than the no-storm baseline. This observation is the result of substantial reductions in vector populations due to flooding, as well as parallel reductions in birds and other mammals, which concentrated more bites on the few remaining people who also were more exposed to bites. This outcome is also highly dependent on how avian species are differentially affected by hurricanes. When lower competency species suffer greater storm-related mortality, the host community competency index of the area (Caillouët and Robertson 2016) increases and likely contributes to enzootic WNV amplification. Conversely, increased mortality of highly competent species may reduce enzootic transmission. Vector feeding preferences for host species, while not considered here, may also play an important role in determining how changes in avian community composition affect transmission (Miller and Huppert 2013).

Mathematical models of pathogen transmission are often limited by the underlying parameter assumptions due to inadequately characterized data and due to necessary simplifications of the system. The outcomes of this pathogen model are based on the assumptions of the effects of hurricane damage to vectors, hosts, and on human exposure. Though many of these assumptions are based on literature reported observations following various hurricanes (in particular for birds; Rittenhouse et al. 2010, Brown et al. 2011, Yaukey 2012), many assumptions are inferred due to a lack of available observations. A particularly

important assumption, which is primarily inferred from reports of nuisance species landing rates (Brown 1997, Simpson 2006, Morrow et al. 2010), is the increased human exposure to mosquito bites following hurricane landfall. Seger et al. (2019) reported that most households noticed an increase in biting mosquitoes following Hurricanes Maria and Irma in 2015 in the US Virgin Islands. It is also possible that increased bites from nuisance mosquito species may decrease human exposure to vector species if people are more aware of mosquitoes in general and take measures to avoid bites. Simplifications to the modeled system, including only 2 avian host species and 1 vector species, are necessary for computational efficiency. Dozens of bird, animal, and vector species are inherently involved in complex zoonotic ecosystems. Given the observation of more diverse host ecosystems to dampen WNV transmission (host dilution effect; Ezenwa et al. 2006), we modeled the effects of the elimination and reduction of a single avian host in a simplified 2-host system.

Hurricane damage extent is not so cleanly categorical as simplified in this study. Although a continuum of damage may be expected as a function of distance from the hurricane landfall location, finer geographic scale impacts of hurricanes often exist. As such, geographic alterations in WNV risk could be expected to be somewhat incremental rather than stepwise as presented. In addition, we did not model the potential impacts of mosquito control agencies which operate to mitigate WNV risk to people. Owing to the direct damage hurricanes cause to property, access, and labor resources, storm events have significant impacts on the ability of government agencies to perform their mission—potentially inhibiting preexisting WNV controls.

Despite the theoretical increases in WNV-NID human cases and incidence described in this analysis, observed increases in WNV human incidence after a hurricane remain rare (Nasci and Moore 1998, Caillouët et al. 2008b). There are several challenges to observing hurricane-related WNV transmission effects, including the relative rarity of WNV-NID occurrence, storm-influenced inaccuracy of reporting of human cases, variable effects of storm damage, and a quickly changing human population across space and time. Given these challenges, it is not surprising that many hurricane events have unobservable effects on WNV transmission.

This study modeled hurricane-related impacts on the WNV ecosystem of the Northern Coast of the Gulf of Mexico from the panhandle of Florida to South Texas. Vector abundance was parameterized directly from *Cx. quinquefasciatus* abundance data from Louisiana. Likewise, the avian host nesting data observed in Louisiana were used to parameterize bird population phenology. Despite localized parameterization, concepts such as the effect on WNV transmission of timing of landfall and the spatial damage dependency are likely globally relevant for future storm events.

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