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A host stage-structured model of enzootic *West Nile virus* transmission to explore the effect of avian stage-dependent exposure to vectors

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HIGHLIGHTS

- We present a host stage-structured model for *West Nile virus* transmission.
- Biting rates vary across stages to test effects of differential exposure to vectors.
- Increased exposure of juvenile hosts results in earlier, more intense transmission.
- Exposure vulnerability and pathogen susceptibility increase transmission.
- Timing of vector growth and avian nesting is important for virus amplification.

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ABSTRACT

Though seasonal *West Nile virus* (WNV) outbreaks have been widely observed to be associated with the end of the avian nesting season, specific ecological mechanisms accounting for this synchronicity remain poorly understood. In this paper we develop and evaluate a novel mathematical model of enzootic WNV transmission to gain insight into the mechanisms responsible for structuring WNV dynamics. We incorporate avian (host) stage-structure (nestling, fledgling, and adult) and within-species heterogeneity in the form of stage-specific mosquito (vector) biting rates. We determine the extent to which temporal fluctuations in host stage and vector abundance throughout the season, along with the differential exposure of these stages to mosquito bites, affect the timing and magnitude of WNV outbreaks in the vector population. We find heterogeneity in avian stage exposure, particularly an increase in juvenile exposure, to result in earlier, more intense transmission. The effects of differential exposure are dependent upon vector abundance, both at carrying capacity as well as during initial stages of nestling production.

1. Introduction

Introduced into the United States in 1999, *West Nile virus* (WNV) remains an annual public health concern. Since its introduction, the Centers for Disease Control and Prevention (CDC) has documented over 41,000 human cases in 49 states with over 1700 of these cases resulting in death (Centers for Disease Control and Prevention (CDC), 2015). Though WNV infects a wide spectrum of vertebrate animals, only some species of birds, small mammals, and reptiles are able to amplify the pathogen and infect hematophagous mosquitoes, which subsequently spread the pathogen to new hosts (Kilpatrick et al., 2007; Turell et al., 2001). The host community composition, along with species-specific host feeding preferences of vectors, has been shown to be important for determining the WNV risk of an area (Ezenwa et al., 2006).

Although many studies have demonstrated the importance of heterogeneity in the host community structure and host feeding patterns of mosquitoes on WNV transmission (Kilpatrick et al., 2006a; Ezenwa et al., 2006; Hamer et al., 2011), the effect of within-species variability, such as differences among host age groups, has been less thoroughly explored. Susceptible birds, mostly in their first year, have been identified to be the primary amplifying hosts (Hamer et al., 2008). Newly hatched, nest-bound birds, or nestlings, have little feather coverage, are largely immobile, and exhibit very few defense mechanisms (Edman and Scott, 1987; Scott and Edman, 1991). In experimental settings these

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factors have been shown to increase exposure to mosquito bites relative to older birds (Blackmore and Dow, 1958; Kale et al., 1972). Young birds that have left the nest (hereafter, fledglings) have nearly complete feather coverage and exhibit some anti-mosquito defensive behaviors. They presumably are more exposed to vectors than mature adult birds, but less so than nestlings. Vector-borne pathogens with similar transmission patterns (bird-mosquito-bird) such as *Eastern equine encephalitis virus* (EEEV) and *St. Louis encephalitis virus* (SLEV) are thought to be driven primarily by the young-of-year bird hosts (inclusive of nestlings and fledglings), who are also assumed to have increased competence (Unnasch et al., 2006; Lord and Day, 2001b). Despite their similarities, it is not known if young-of-year hosts exhibit increased WNV transmission competence relative to older hosts.

Though the annual transmission of WNV in most regions of the United States is highly seasonal, with the vast majority of human cases occurring during late summer in August and September (Lindsey et al., 2010), the ecological mechanisms governing this seasonality are not well understood (Kilpatrick et al., 2006b). The timing of enzootic (mosquito to bird) outbreaks, which precede human WNV outbreaks, has been widely observed to coincide with the end of the avian nesting season (Kilpatrick et al., 2006b; Caillouët et al., 2013a). Though increased risk of WNV to humans has been linked to seasonal changes in vectors' host feeding preference (Kilpatrick et al., 2006b), specific mechanisms responsible for amplification of the virus in the mosquitoes remain poorly understood.

Mathematical models of WNV have been useful in conceptually demonstrating several important ecological mechanisms that govern WNV transmission (Wonham et al., 2004; Simpson et al., 2012; Unnasch et al., 2006; Lord and Day, 2001b, 2001a; Abdelrazec et al., 2014). Wonham et al. (2004) found the basic reproduction number R_0 of an ODE model for WNV to depend on the ratio of vectors to susceptible hosts, suggesting that a reduction in the number of hosts exacerbates WNV transmission by increasing the contact between vectors and the few remaining hosts. Simpson et al. (2012) showed the mosquito feeding index to be a very influential parameter on WNV transmission in a model with multiple host species. Lord and Day (2001b) developed a two stage juvenile/adult ODE model for SLEV, later incorporating disease related mortality to model WNV (Lord and Day, 2001a). Unnasch et al. (2006) also considered both young-of-year and adult birds in a Stella model for EEEV. These models did allow for age-specific vector biting rates, but juveniles were also considered to be more competent than adults.

In this paper we develop an avian host stage-structured model of WNV enzootic transmission to investigate the ecological underpinnings of virus amplification. Specifically, we analyze the effect of stage-dependent host differences in mosquito exposure

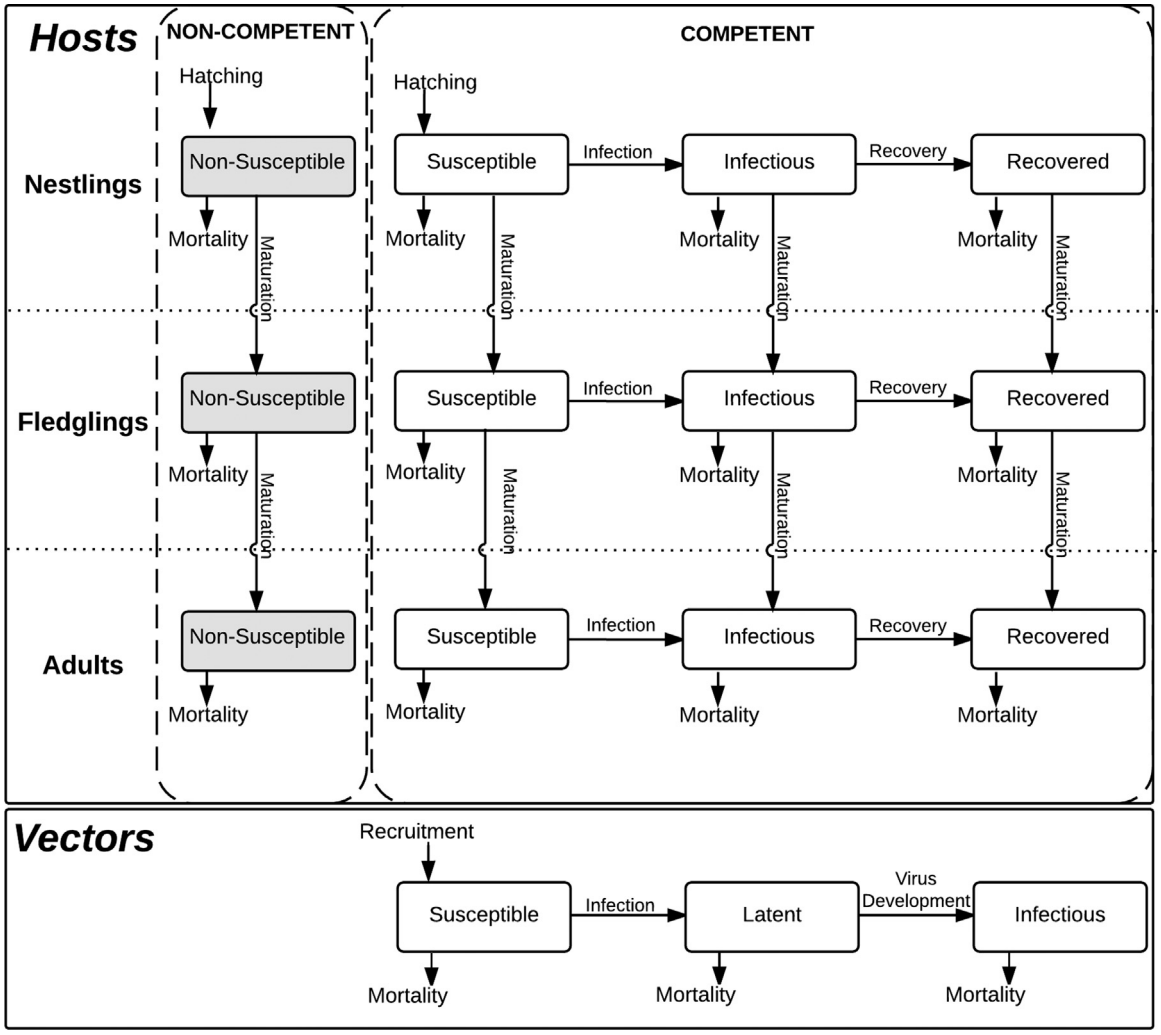

Fig. 1. *West Nile virus* host stage-structured model schematic. All classes of vectors feed on all classes and stages of hosts. Disease transmission can occur when an infectious vector bites a susceptible host or when a susceptible vector bites an infectious host. Shaded compartments are not explicitly modeled, but indicate the presence of non-competent hosts in the system receiving bites that would otherwise go to competent hosts (unshaded compartments).