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Editorial

Resurgence of West Nile Virus in 2012: Lack of Evidence for Viral Genetic Determinants

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Since the introduction into North America in 1999 of West Nile virus (WNV) and its subsequent dramatic spread across the United States and Canada, human cases of WNV infection declined from their 2003 peak of 9,862 to only 712 cases and 43 fatalities confirmed in 2011 (http://www.cdc.gov/ncidod/dvbid/westnile/index.htm). The reasons for this decline remain unclear because avian amplification host and human herd immunity remains low in most regions and WNV continues to circulate in most areas of the United States. However, last year an unexpected resurgence in West Nile fever and encephalitis cases occurred; a total of 5,674 human cases was detected in the United States, with 286 deaths, making 2012 the deadliest year yet. The epidemic was focused in the central part of the United States, with Texas reporting 1,739 human infections and 89 deaths, or about 33% of the national total and more than three times the number of any other state. Within Texas, Dallas County was the epicenter, with 398 human infections and 20 deaths.

The reasons for this dramatic resurgence in WNV infections are not clear; however, several factors could be involved ranging from increased enzootic bird-mosquito-bird circulation, resulting in more human spillover infections, to increased human virulence. In this issue, Duggal and others report complete genomic sequences of 17 WNV isolates from several regions of Texas and phylogenetic studies to determine if viral genetic changes might have been responsible for the resurgence in human cases. Two distinct WNV genotypes were identified, and both circulated in the Dallas and Houston regions where disease incidence differed markedly (about four times higher in Dallas). Importantly, no signs of adaptive evolution that could have resulted in more efficient infection of mosquitoes or birds were identified in any of the recent WNV strains. These results, along with epidemiological analyses, suggest that the 2012 resurgence in the Dallas area resulted from an increase in transmission by mosquitoes from birds to humans (spillover infections), rather than an increase in WNV neuroinvasiveness. Unusually high summer temperatures in the Dallas area, perhaps combined with ecological changes affecting mosquito or avian populations, probably played important roles in driving the 2012 outbreak.

These critical findings presented by Duggal and others[1] and the magnitude of the 2012 West Nile encephalitis epidemic underscore the importance of a better understanding of how WNV, other arboviruses, and vector-borne diseases in general will respond to climate and anthropogenic ecological changes that now appear inevitable. Rising temperatures exert a wide variety of complex effects on mosquito survival, reproduction, and behavior. Because temperature also influences vector infection, dissemination, and transmission of arboviruses, often in unpredictable ways, we remain unable to anticipate the effects of global warming on arboviral disease. Droughts, which are predicted to increase in some regions of the United States and elsewhere, can also have dramatic impacts on mosquito populations, especially on West Nile mosquito vectors because of their affinity for standing, highly polluted water and to irrigated agricultural lands. Drought conditions can also concentrate avian amplifying hosts in wetter locations amenable to mosquito transmission of WNV. Thus, rigorous experimental and modeling studies are needed to improve the ability of our public health systems to anticipate and respond to the effects of climate change and its influences on vector-borne pathogens such as WNV.

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