

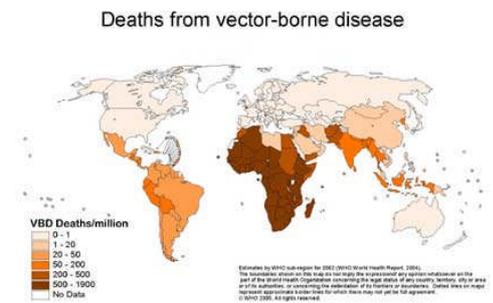
RWBAHC 2014 April Newsletter

01Apr2014

Editor: CPT Lauren Baldwin, DVM



Beginning this week, March is a distant memory as we move on to discuss April 2014's national health topics. That's right—we're diving into World Health Day as we discuss the global concern of vector-borne diseases pertinent to both you and your pet! Read below for more.



I hope you enjoy this week's newsletter! And, as always, you can check out our previous issues by visiting the RWBAHC web site. Have a great week!

CPT Baldwin

Get Excited for World Health Day!

Overview



World Health Day (WHD) is celebrated annually on 07 April to mark the founding of the World Health Organization (WHO) and draw attention to important health issues facing the globe.

More specifically, WHO, first founded in 1948, functions under the United Nations (UN) to address issues/emergencies pertaining to health on a global scale.

In fact, the organization has played a major role in the control/eradication of historical diseases such as polio, smallpox, chickenpox etc. which were earlier seen in epidemic proportions in several developing countries. Apart from global health concerns, the organization is also responsible for producing the World Health report, which gives an account of overall health of people in UN member nations.

World Health Day (WHD) was first instituted during the 1950 UN Geneva gathering. Since then, a specific, global health theme is chosen annually and awareness as it pertains to this topic is stressed by the organization throughout the remaining year. For instance, one of its most effective World Health Day themes has been Global Polio Eradication first adopted in 1995. Ever since, the awareness level of people towards polio has risen considerably and most parts of the world have been freed from the dreaded disease. Other important themes adopted by WHO for the day have been Safe Motherhood in 1998; Emerging Infectious Diseases in 1997; Move for Health in 2002; International Health Security in 2007, etc. This year, WHO has chosen to focus on vector-borne diseases with the logo, "Small Bite, Big Threat."

World Health Day 2014: "Small Bite, Big Threat"

Vector borne diseases represent a major threat to the human population both on a domestic as well as a global scale. Due to their epidemiology and means of transmission, these diseases often spread quickly across major geographical areas with movement of their hosts.

Read below as World Health Day 2014 will spotlight the growing risk of vector-borne diseases as well as means of protection via a multifactorial approach.

What are vectors and vector-borne diseases?



Vector-borne disease is the term commonly used to describe an illness caused by an infectious microbe that is transmitted to people most commonly by blood-sucking arthropods. The arthropods (insects or arachnids) that most commonly serve as vectors include: 1.) insects such as mosquitoes, fleas, lice, biting flies and bugs, and 2.) arachnids such as mites and ticks. The term “vector” refers to any organism that transmits a disease through feeding activity.

Vectors typically become infected by a disease agent while feeding on infected vertebrates (e.g., birds, rodents, other larger animals, or humans), then pass on the microbe to a susceptible person or other animal. In almost all cases, an infectious microbe must infect and multiply inside the arthropod/vector before the arthropod is able to transmit the disease through its salivary glands.

Vector-borne diseases are most commonly found in tropical areas and/or regions where access to safe drinking-water and sanitation systems is problematic and account for 17% of the estimated global burden of all infectious diseases, affecting over 100 countries worldwide. Historically, the most deadly vector-borne disease, malaria, has caused an estimated 660,000 deaths in 2010, predominantly within African youth. As a second example, the world’s fastest growing vector-borne disease, dengue, has shown a 30-fold increase in disease incidence over the last 50 years. Globalization of trade and travel as well as environmental challenges such as climate change and urbanization are having a substantial impact on transmission of vector-borne diseases, resulting in their appearance in countries where they were previously unknown.

The list of vector-borne diseases is quite extensive and outside the scope of this article. For more information on illnesses not covered below, please visit the World Health Organization web site.

Key Vector-Borne Diseases

1. Dengue:

- Dengue is a mosquito-borne viral infection.
- The infection causes flu-like illness and occasionally develops into a potentially lethal complication called severe dengue.
- The global incidence of dengue has grown dramatically in recent decades.
- About half of the world’s population is now at risk.
- Dengue is found in tropical and sub-tropical climates worldwide, mostly in urban and semi-urban areas.
- Severe dengue is a leading cause of serious illness and death among children in some Asian and Latin American countries.
- There is no specific treatment for dengue/ severe dengue, but early detection and access to proper medical care lowers fatality rates below 1%.
- Dengue prevention and control solely depends on effective vector control measures.



Dengue is a mosquito-borne infection found in tropical and sub-tropical regions around the world. In recent years, transmission has increased predominantly in urban and semi-urban areas and has become a major international public health concern.

Severe dengue (also known as Dengue Haemorrhagic Fever) was first recognized in the 1950s during dengue epidemics in the Philippines and Thailand. Today, severe dengue affects most Asian and Latin American countries and has become a leading cause of hospitalization and death among children in these regions.

There are four distinct, but closely related, serotypes of the virus that cause dengue (DEN-1, DEN-2, DEN-3 and DEN-4). Recovery from infection by one provides lifelong immunity against that particular serotype. However, cross-immunity to the other serotypes after recovery is only partial and temporary. Subsequent infections by other serotypes increase the risk of developing severe dengue.

Global burden of dengue:

The incidence of dengue has grown dramatically around the world in recent decades with over 2.5 billion people – over 40% of the world’s population – now at risk. WHO currently estimates there may be 50–100 million dengue infections worldwide annually.

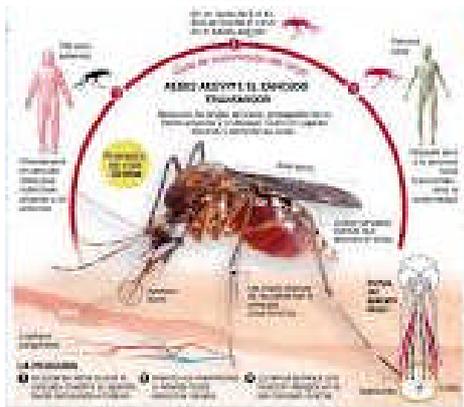
Before 1970, only nine countries had experienced severe dengue epidemics. The disease is now endemic in more than 100 countries in Africa, the Americas, the Eastern Mediterranean, South-east Asia and the Western Pacific with the American, South-east Asian and the Western Pacific regions being the most seriously affected. Cases in these areas have exceeded 1.2 million in 2008 and over 2.3 million in 2010 (based on official data submitted by Member States). Recently the number of reported cases has continued to increase. In 2013, 2.35 million cases of dengue were reported in the Americas alone, of which 37,687 cases were severe dengue!

Not only is the number of cases increasing as the disease spreads to new areas, but explosive outbreaks are occurring. The threat of a possible outbreak of dengue fever now exists in Europe, and local transmission of dengue was reported for the first time in France and Croatia in 2010 while imported cases were detected in three other European countries. **In 2012, an outbreak of dengue on Madeira islands of Portugal resulted in over 2,000 cases and imported cases were detected in 10 other countries in Europe apart from mainland Portugal.**

In 2013, cases have occurred in Florida (US) and Yunnan province of China. Dengue also continues to affect several south American countries notably Honduras, Costa Rica and Mexico. In Asia, Singapore has reported an increase in cases after a lapse of several years and outbreaks have also been diagnosed in Laos. In 2014, trends indicate increases in the number of cases in the Cook Islands, Malaysia, Fiji and Vanuatu, with Dengue Type 3 (DEN 3) affecting the Pacific Island countries after a lapse of over 10 years.

An estimated 500,000 people with severe dengue require hospitalization annually, a large proportion of whom are children. About 2.5% of those affected die.

Transmission:



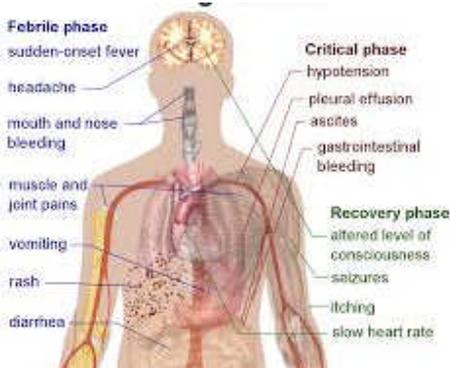
The *Aedes aegypti* mosquito is the primary vector of dengue. The virus is transmitted to humans through the bites of infected female mosquitoes. After virus incubation for 4–10 days, an infected mosquito is capable of transmitting the virus for the rest of its life.

Infected humans are the main carriers and multipliers of the virus, serving as a source of the virus for uninfected mosquitoes. Patients who are already infected with the dengue virus can transmit the infection (for 4–5 days; maximum 12) via *Aedes* mosquitoes after their first symptoms appear.

The *Aedes aegypti* mosquito lives in urban habitats and breeds mostly in man-made containers. Unlike other mosquitoes, *Ae. aegypti* is a daytime feeder with peak biting periods of early morning and evening before dusk. What's more, female *Ae. aegypti* bite multiple people during each feeding period.

Aedes albopictus, a secondary dengue vector in Asia, has spread to North America and Europe largely due to the international trade of used tires (a breeding habitat) and other goods (e.g. lucky bamboo). *Ae. albopictus* is highly adaptive and therefore can survive in cooler temperate regions of Europe. Its spread is due to its tolerance to temperatures below freezing, hibernation, and ability to shelter in microhabitats.

Clinical Signs:



Dengue fever is a severe, flu-like illness that affects infants, young children and adults with a relatively low mortality rate.

Dengue should be suspected when a high fever (40°C/ 104°F) is accompanied by two of the following symptoms: severe headache, pain behind the eyes, muscle and joint pains, nausea, vomiting, swollen glands or rash. Symptoms usually last for 2–7 days after an incubation period of 4–10 days following the bite from an infected mosquito.

Severe dengue is a potentially deadly complication **due to plasma leaking, fluid accumulation, respiratory distress, severe bleeding, or organ impairment.** Warning signs occur 3–7 days after initial clinical signs in conjunction with a decrease in temperature (below 38°C/ 100°F) and include: severe abdominal pain, persistent vomiting, rapid breathing, bleeding gums, fatigue, restlessness, blood in vomit. The next 24–48 hours of the critical stage can be lethal; proper medical care is needed to avoid complications and risk of death.

Treatment:

There is no specific treatment for dengue fever.

For severe dengue, medical care by physicians and nurses experienced with the effects and progression of the disease can save lives – decreasing mortality rates from more than 20% to less than 1%. Maintenance of the patient's body fluid volume is critical to severe dengue care.

Immunization:

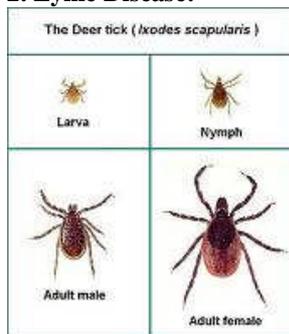
There is no vaccine to protect against dengue. WHO provides technical advice and guidance to countries and private partners to support vaccine research and evaluation. Several candidate vaccines are in various phases of trials.

Prevention and control:

At present, the only method to control or prevent the transmission of dengue virus is to combat vector mosquitoes through:

- preventing mosquitoes from accessing egg-laying habitats by environmental management and modification;
- disposing of solid waste properly and removing artificial man-made habitats;
- covering, emptying and cleaning of domestic water storage containers on a weekly basis;
- applying appropriate insecticides to water storage outdoor containers;
- use of personal household protection such as window screens, long-sleeved clothing, insecticide treated materials, coils and vaporizers;
- improving community participation and mobilization for sustained vector control;
- applying insecticides as space spraying during outbreaks as one of the emergency vector control measures;
- active monitoring and surveillance of vectors to determine effectiveness of control interventions.

2. Lyme Disease:



Lyme disease (LD) is an infection caused by *Borrelia burgdorferi*, a type of bacterium called a spirochete that is carried by deer (*Ixodes* spp) ticks. An infected tick can transmit the spirochete to both humans and animals via blood transfer during a bite. LD manifests itself as a multisystem inflammatory disease that affects the skin in its early, localized stage (characteristic “bull’s eye” lesion), and spreads to the joints, nervous system and, to a lesser extent, other organ systems in its later, disseminated stages.

Clinical signs of Lyme disease were first reported in medical literature in Europe in 1883. Over the years, various clinical signs of this illness have been noted as separate medical conditions: acrodermatitis, chronica atrophicans (ACA), lymphadenitis benigna cutis (LABC), erythema migrans (EM), and lymphocytic meningoradiculitis (Bannwarth’s syndrome). However, these diverse manifestations were not recognized as indicators of a single infectious illness until 1975, when LD was described

following an outbreak of apparent juvenile arthritis, preceded by a rash, among residents of Lyme, Connecticut.



Although LD is now the most common arthropod-borne illness in the U.S. (more than 150,000 cases have been reported to the Centers for Disease Control and Prevention [CDC] since 1982), its diagnosis and treatment can be challenging for clinicians due to its diverse manifestations and the limitations of currently available serological (blood) tests.

The prevalence of LD in the northeast and upper mid-west is due to the presence of large numbers of the deer tick’s preferred hosts - white-footed mice and deer - and their proximity to humans. White-footed mice serve as the principal reservoirs of infection on which many larval and nymphal (juvenile) ticks feed and become infected with the LD spirochete. An infected tick can then transmit the bacterium during its subsequent feedings.

While the LD spirochete, *Borrelia burgdorferi*, has been shown to infect other species of ticks, transmission mammalian hosts has only been reported via the deer tick (also known as the black-legged tick) and the related Western black-legged tick. In addition, studies have shown transmission of the spirochete does not begin until engorgement of the respective tick (approximately 36-48 hours following attachment).

Geographical Distribution:

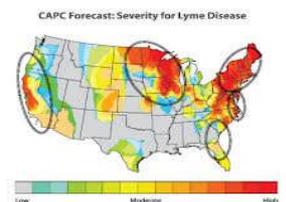
There are foci of Lyme borreliosis in forested areas of Asia, north-western, central and eastern Europe, and the USA.

In northern Africa, *B. burgdorferi sensu lato* has been identified in Morocco, Algeria, Egypt and Tunisia.

Lyme disease in sub-Saharan Africa is presently unknown, but evidence indicates it may occur in humans in this region. The abundance of hosts and tick vectors would favor the establishment of Lyme infection in Africa. In East Africa, two cases of Lyme disease have been reported in Kenya.

B. burgdorferi sensu lato-infested ticks are being found more frequently in Japan, as well as in northwest China, Nepal, Thailand and far eastern Russia. *Borrelia* has also been isolated in Mongolia.

In the United Kingdom the number of laboratory confirmed cases of Lyme disease has been rising steadily since voluntary reporting was introduced in 1986 when 68 cases were recorded in the UK and Republic of Ireland combined. In the UK there were 23 confirmed cases in 1988 and 19 in 1990, but 973 in 2009 and 953 in 2010. Provisional figures for the first 3 quarters of 2011 show a 26% increase on the same period in 2010. It is thought, however, that the actual number of cases is significantly higher than suggested by the above figures, with the UK’s Health Protection Agency estimating that there are between 2,000 and 3,000 cases per year, (with an average of around 15% of the infections acquired overseas).



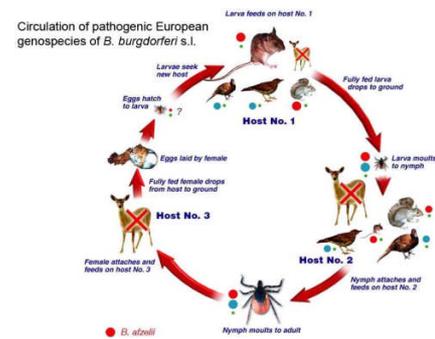
Many studies in North America have examined ecological and environmental correlates of Lyme disease prevalence. A 2005 study using climate suitability modelling of *Ixodes scapularis* projected that climate change would cause an overall 213% increase in suitable vector habitat by the year 2080, with northward expansions in Canada, increased suitability in the central U.S., and decreased suitable habitat and vector retraction in the southern U.S. A 2008 review of published studies concluded that the presence of forests or forested areas was the only variable that consistently elevated the risk of Lyme disease, and that other environmental variables showed little or no concordance between studies.

Lyme disease is the most common tick-borne disease in North America and Europe, and one of the fastest-growing infectious diseases in the United States. Of cases reported to the United States CDC, the ratio of Lyme disease infection is 7.9 cases for every 100,000 persons. In the ten states where Lyme disease is most common, the average was 31.6 cases for every 100,000 persons for the year 2005. Although Lyme disease has been reported in all states except Montana, about 99% of all reported cases are confined to just five geographic areas (New England, Mid-Atlantic, East-North Central, South Atlantic, and West North-Central).

The number of reported cases of the disease has been increasing, as are endemic regions in North America. Although Montana is the only state that has not reported a confirmed case of Lyme disease, in recent years there have been 5 to 10 cases a year of a disease similar to Lyme. It occurs primarily in pockets along the Yellowstone River in central Montana. People have developed a red bull's-eye rash around a tick bite followed by weeks of fatigue and a fever. Lyme disease prevalence is comparable among males and females. A wide range of age groups is affected, though the number of cases is highest among 10–19-year-olds. For unknown reasons, Lyme disease is seven times more common among Asians.

Transmission:

The spirochetal agent of Lyme disease, *Borrelia burgdorferi*, is transmitted to humans through a bite of a nymphal stage deer tick *Ixodes scapularis* (or *Ixodes pacificus* on the West Coast). The duration of tick attachment and feeding is a key factor in transmission. Proper identification of tick species and feeding duration aids in determining the probability of infection and the risk of developing Lyme disease.



Clinical Signs:

The early symptoms of LD can be mild and easily overlooked.

Most often, infected humans initially note an expanding rash (erythema migrans) which is thought to occur in 80% to 90% of all LD cases. An EM rash generally has the following characteristics:

- Often radiates outward from the site of the tick bite
 - Appears either as a solid red expanding rash or blotch or a central spot surrounded by clear skin that is in turn ringed by an expanding red rash (resembles a bull's-eye)
 - Appears an average of 1 to 2 weeks (range = 3 to 30 days) after disease transmission
 - Has an average diameter of 5 to 6 inches (range = 2 inches to 2 feet)
 - Persists for about 3 to 5 weeks
 - May or may not be warm to the touch
- Is generally neither painful nor pruritic



A dark, bruise-like appearance is more common on dark-skinned patients.

Additional symptoms may include joint pain, chills, fever, and fatigue. These symptoms may be brief, only to recur as a broader spectrum of symptoms as the disease progresses. As the LD spirochete continues spreading through the body, severe fatigue, a stiff, aching neck, and peripheral nervous system (PNS) involvement such as tingling or numbness in the extremities or facial palsy (paralysis) have been described.

The more severe, potentially debilitating symptoms of later-stage LD may occur weeks, months, or, in a rare cases, years following a tick bite and most often include severe headaches, painful arthritis/swelling of joints, cardiac abnormalities, and central nervous system (CNS) involvement leading to cognitive (mental) disorders.

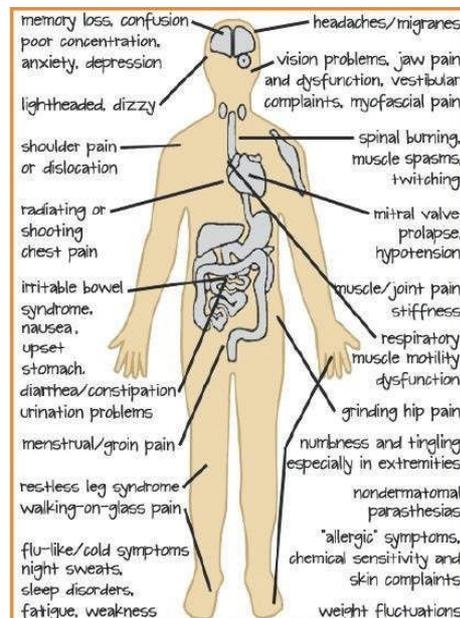
The following is a checklist of common symptoms seen in various stages of LD:

Localized Early (Acute) Stage:

- Solid red or bull's-eye rash, usually at site of bite
- Swelling of lymph glands near tick bite
- Generalized achiness
- Headache

Early Disseminated Stage:

- Two or more rashes not at site of bite
- Migrating pains in joints/tendons
- Headache
- Stiff, aching neck
- Facial palsy (facial paralysis similar to Bell's palsy)
- Tingling or numbness in extremities
- Multiple enlarged lymph glands
- Abnormal pulse



- Sore throat
- Changes in vision
- Fever of 100 to 102 F
- Severe fatigue

Late Stage:

- Arthritis (pain/swelling) of one or two large joints
- Disabling neurological disorders (disorientation; confusion; dizziness; short-term memory loss; inability to concentrate, finish sentences or follow conversations; mental "fog")
- Numbness in arms/hands or legs/feet

Treatment:

Antibiotic treatment of patients diagnosed as LD positive is generally curative with complete resolution of clinical signs.

Prevention & Control:



Larval and nymphal deer ticks often hide in shady, moist ground litter with adults above ground clinging to tall grass, brush, and shrubs. They also inhabit lawns and gardens, especially at the edges of woodlands and around old stone walls where deer and white-footed mice, the ticks' preferred hosts, thrive. Within the endemic range of *B. burgdorferi*, no natural, vegetated area can be considered completely free of infected ticks.

Deer ticks cannot jump or fly, and do not drop from above onto a passing animal. Potential hosts acquire ticks only by direct contact. Once a tick latches onto human skin, it generally climbs upward until it reaches a protected or creased area, often the back of the knee, groin, navel, armpit, ears, or nape of the neck.

Protective clothing includes a hat, long-sleeved shirts and long trousers tucked into socks or boots. Light-colored clothing helps with visibility of ticks for removal. In addition, Permethrin sprayed on clothing kills ticks on contact. Insect repellents with Picaridin, IR3535, DEET or Oil of Lemon Eucalyptus repel ticks as well.

Reducing the numbers of primary hosts on which the deer tick depends, such as rodents and other small mammals may, over time, help break the reproductive cycle of the deer ticks and their ability to flourish in suburban and rural areas. An unusual, organic approach to control of ticks and prevention of Lyme disease involves the use of domesticated guineafowl (voracious consumers of insects and arachnids).

Yard work is perhaps a more feasible method of reducing the deer tick population. Keep lawns mowed and edges trimmed; clear brush, leaf litter and tall grass around houses and at the edges of gardens and open stone walls; etc.

3. West Nile Virus:

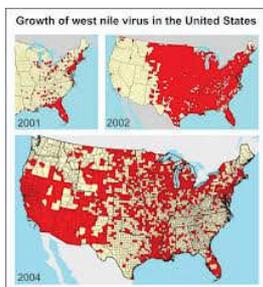
- West Nile virus can cause a fatal neurological disease in humans.
- However, approximately 80% of people who are infected do not exhibit clinical signs.
- West Nile virus is mainly transmitted via the bites of infected mosquitoes.
- The virus can cause severe disease and death in horses.
- Vaccines are available for use in horses but not yet available for humans.
- Birds are the natural hosts of West Nile virus.



West Nile Virus (WNV) has been shown to cause neurological disease and death in humans. Commonly found in Africa, Europe, the Middle East, North America and West Asia, WNV is maintained in nature in a cycle involving transmission between birds and mosquitoes.

West Nile Virus (WNV) is a member of the *flavivirus* genus and belongs to the Japanese encephalitis antigenic complex of the family *Flaviviridae*.

Outbreaks:



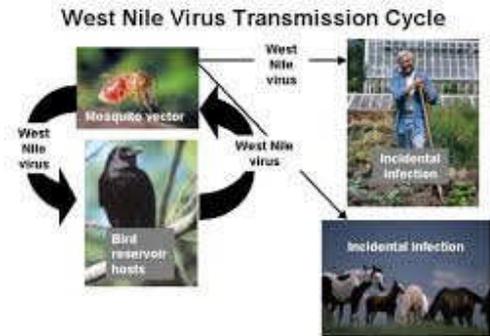
West Nile Virus (WNV) was first isolated in a woman in the West Nile district of Uganda in 1937. It was identified in birds (crows and columbiformes) in the Nile delta region in 1953. Prior to 1997, WNV was not considered pathogenic for birds; however, mutation of a virulent strain in Israel during that time manifested in presenting signs of encephalitis and paralysis among various avian species..

In 1999, WNV circulating in Israel and Tunisia was imported into New York producing a large and dramatic outbreak that spread throughout the continental United States in the following years. The outbreak (1999-2010) highlighted that importation and establishment of vector-borne pathogens outside their current habitat represent a serious danger to the world with rapid spread of the disease notable along bird migratory routes through Greece, Israel, Romania, and Russia. In its original range, WNV was prevalent throughout Africa, parts of Europe, Middle East, West Asia, and Australia.

Transmission:

Most commonly, the flavivirus is transmitted from infected fowl to mosquito during feeding. The pathogen then concentrates within the insect's salivary glands and passed into a subsequent host species during the mosquito's next blood meal.

A very small proportion of human infections have occurred through organ transplant, blood transfusions, and breast milk with one reported case of transplacental (mother-to-child) transmission. To date, no human-to-human route of WNV transmission through casual contact has been documented. Transmission of WNV to laboratory workers has been reported.



Clinical Signs:

About West Nile virus and humans

Human, animal immune systems usually destroy virus in bloodstream. About 80 percent of those with the virus have **no symptoms**.

Number of days it takes for symptoms to appear after being bitten: **3-14**

The virus is spread by mosquitoes that carry it from birds to humans.

Small Sources: Centers for Disease Control and Prevention

About 20 percent have mild symptoms:

- Fever
- Headache
- Body aches
- Skin rash
- Swollen lymph nodes

A severe case, 1 in 150, can result in death. The risk is highest for elderly children and people with impaired immune systems.

If virus survives in body, it can infect membranes around spinal cord and brain (encephalitis).

The Register

The diagram shows a cross-section of the human head and neck, highlighting the brain, spinal cord, and the protective membrane surrounding them. Labels include 'Membrane', 'Brain', and 'Spinal cord'.

Eighty percent of humans infected with WNV remain asymptomatic (exhibit no clinical signs of disease).

The remaining 20% report flu-like symptoms to include fever, headache, fatigue, myalgia (body aches), nausea, and vomiting with rare instances of a truncal skin rash and swollen lymph nodes following a 3 to 14 day incubation period.

Signs of severe disease (also called neuroinvasive disease, such as West Nile encephalitis or meningitis or West Nile poliomyelitis) include headache, high fever, stiff neck, stupor, disorientation, coma, tremors, convulsions, muscle weakness, and paralysis. It is estimated that approximately 1 in 150 persons infected with the West Nile virus will develop a more severe form of disease with immunocompromised and elderly patients remaining the most predisposed.

Treatment:

Treatment is supportive for patients with neuro-invasive West Nile virus, often involving hospitalization, intravenous fluids, respiratory support, and prevention of secondary infections. No vaccine is available for humans.

Vector and animal hosts:

WN virus is maintained in nature in a mosquito-bird-mosquito transmission cycle. Mosquitoes of the genus *Culex* are generally considered the principal vectors of WNV, in particular *Cx. Pipiens*. WNV is maintained in mosquito populations through vertical transmission (adults to eggs). Birds are the reservoir hosts of WNV.

In Europe, Africa, Middle East and Asia, mortality in birds associated with WNV infection is rare. In striking contrast, the virus is highly pathogenic for birds in the Americas with members of the crow family (*Corvidae*) particularly susceptible. Moreover, birds may be infected by a variety of routes other than mosquito bites, and different species may have different potential for maintaining the transmission cycle.

Prevention:

In the absence of a vaccine, prevention within the human population relies heavily upon education and reduction in means of exposure.

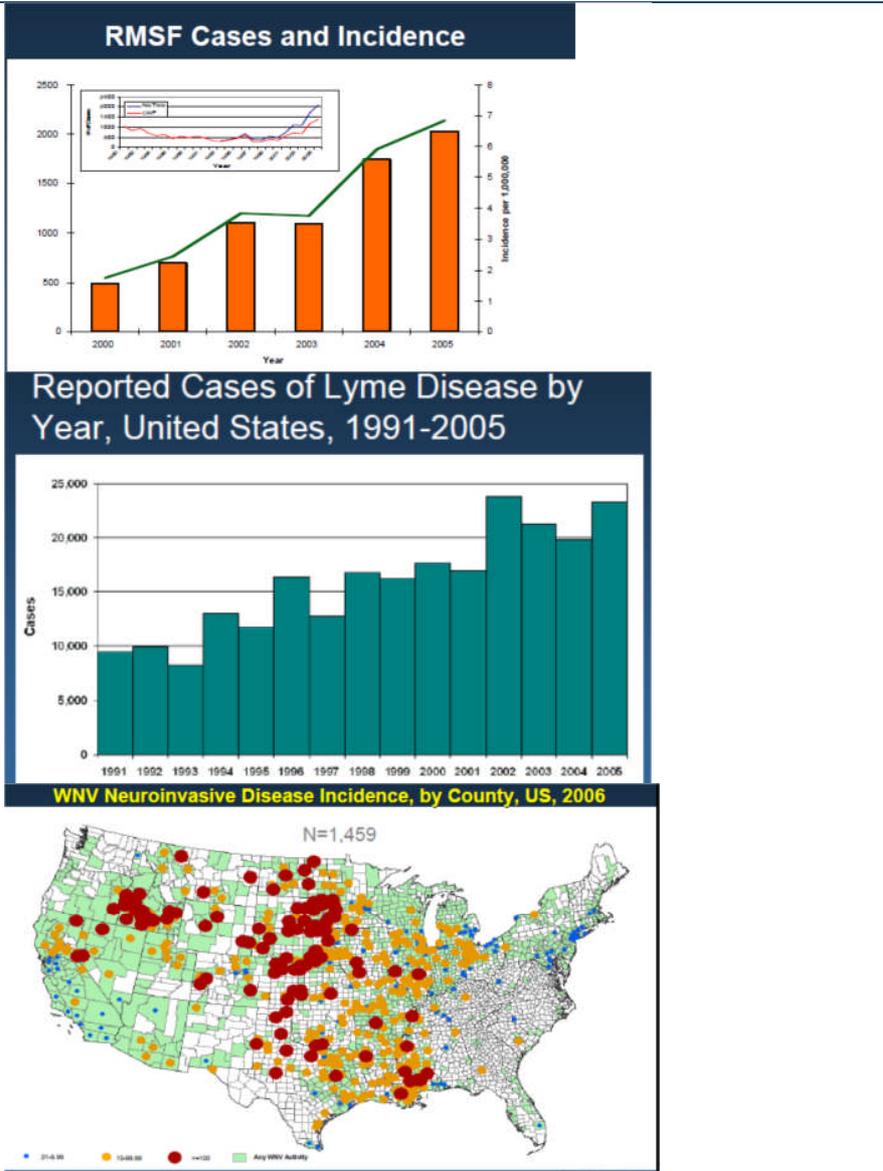
- Reducing the risk of mosquito transmission. Efforts to prevent transmission should first focus on personal and community protection against mosquito bites through the use of mosquito nets, personal insect repellent, by wearing light colored clothing (long-sleeved shirts and trousers) and by avoiding outdoor activity at peak biting times. In addition, community programs should encourage communities to destroy mosquito breeding sites in residential areas.
- Reducing the risk of animal-to-human transmission. Gloves and other protective clothing should be worn while handling sick animals or their tissues, and during slaughtering and culling procedures.
- Reducing the risk of transmission through blood transfusion and organ transplant. Blood and organ donation restrictions and laboratory testing should be considered at the time of the outbreak in the affected areas after assessing the local/regional epidemiological situation.
- Vector Control. Effective prevention of human WNV infections depends on the development of comprehensive, integrated mosquito surveillance and control programs in areas where the virus occurs. Emphasis should be on integrated control measures including source reduction (with community participation), water management, chemicals, and biological control methods.

Domestic Vector-Borne Diseases - At a Glance

Dengue and Dengue Hemorrhagic Fever: Spread by mosquitoes, dengue viruses infect up to 100 million people annually. Puerto Rico experienced its largest outbreak in 2010 and Florida has reported local cases for the first time in 75 years. CDC is working to advance diagnostics, improve patient survival, find new methods of mosquito control and develop innovative vaccines. Read CDC's [Dengue Update](#) for more.

Lyme and other tick-borne diseases: [Tick-borne diseases](#), including [Lyme disease](#) and [Rocky Mountain spotted fever](#) are serious public health problems, infecting tens of thousands within the U.S. each year. CDC is working closely with local communities, developing innovative control approaches and researching improved diagnostics.

West Nile and other mosquito-borne viruses: [West Nile virus](#) swept across the U.S. in less than 10 years, causing over 12,700 cases of severe disease. CDC leads [ArboNET](#), an innovative system to monitor mosquito-borne infections in humans, mosquitoes, birds and other animals. This information allows CDC and states to quickly respond, preventing further cases. CDC and its partners implemented screening of the U.S. blood supply for WNV, preventing 3,000-9,000 transfusion-transmitted infections.



To further illustrate the rise of vector-borne diseases in the United States, read the article below on the recent re-emergence of Dengue fever along the Florida coastline!

[Vector-Borne Diseases Growing as Threat to U.S. Public Health: Climate Change and Travel Linked to Illness](#)
 Source: *The Nation's Health* September 2010 vol. 40 no. 7 1-21



Vector-borne diseases (VBDs) account for 16 % of the estimated global burden of communicable diseases

Last fall, an old resident returned to the beaches of Florida, though it certainly was not welcome and officials are determined to see its visit cut short.

The unwanted visitor is mosquito-borne dengue, which made headlines this summer after public health officials found that 5 percent of Key West residents showed recent exposure to the virus. The problem began late last year with 27 reported cases, prompting immediate response from local health and mosquito control

officials. Winter came and went with no more reported cases, and health officials hoped that it meant the end of dengue's stay. But April saw another case with more after that, finally leading to the headline-grabbing Key West study, which began after the initial cases in 2009.

"These people had not traveled outside of Florida, so we need to determine if these cases are an isolated occurrence or if dengue has once again become endemic in the continental United States," said Harold Margolis, chief of the dengue branch at the Centers for Disease Control and Prevention.

With the last reported Florida dengue outbreak in 1934, its recent re-emergence has health officials worried that with the right conditions, the virus could gain a new foothold in its old stomping grounds. Health officials also worry that outbreaks such as the dengue one are part of a growing trend that could be poised to get worse — that changing climates, more travel and urbanization mean vector-borne, once-tropical diseases such as dengue and West Nile virus are not only here to stay, but are on the move.

"This is not going away," said Peter Hotez, president-elect of the American Society of Tropical Medicine and Hygiene. "I actually think that this is a bigger threat than many of the biodefense pathogens that we're spending huge amounts of money on. Dengue and other vector-borne diseases are a true homeland security threat."

Experts have long warned that warmer temperatures due to climate change will make it easier for vector-borne diseases to spread. For example, a study in the April issue of *Environmental Health Perspectives* on the northward expansion of a species of tick and the emergence of Lyme disease in Canada said the tick's movement is "possibly facilitated by a warming climate."

Vector-borne diseases are also taking advantage of easy travel, both domestically and globally. A 2007 study in the *New England Journal of Medicine* on mosquito-borne chikungunya fever, which affects a person's joints, said the disease was diagnosed in an "unprecedentedly" large number of people returning to Europe and the United States from islands in the Indian Ocean where a 2006 outbreak occurred. The authors noted that the "chikungunya outbreak is an example of the abrupt expression of vector-borne diseases in the global village."

And in some cases, researchers cannot yet find the reason for a vector-borne outbreak. For example, a 2005 study in the *New England Journal of Medicine* on the tick-borne Rocky Mountain spotted fever said the disease was in the midst of its third emergence since 1920, with more than 1,500 cases reported in 2004. While the authors said Rocky Mountain spotted fever can no longer be considered only a rural or southern disease, theories attributing its emergence to "suburban development, changes in recreational activities or long-term changes in climate have not withstood careful investigation."

Back in Florida, Carina Blackmore, DVM, PhD, state public health veterinarian with the Florida Department of Health, said the source of the recent dengue outbreak is "certainly" travel-related. She said the strain found in Key West ultimately originated in Mexico, with the likely scenario being a person transporting the virus to the Keys and infecting the local mosquito population. Blackmore noted that dengue was once endemic in Florida, but with housing and mosquito control advancements in the 1950s, the disease lost its hold.

"We have the environmental conditions for these diseases," Blackmore told *The Nation's Health*. "But it's human behavior that's different now than 50 years ago, so it's difficult for these diseases to get re-established."

Blackmore said she is more concerned with vector-borne diseases that have animal hosts, such as Rift Valley fever, which infects livestock and can be transmitted to humans via contact with an infected animal's blood or organs. The mostly Africa-based virus, which has human symptoms similar to West Nile virus, has not been detected in Florida, but she said officials are watching and preparing for it.

Other U.S. communities have been confronting dengue for a while now. In Brownsville, Texas, health officials are constantly on watch for the disease, said Art Rodriguez, director of the health department in Brownsville, which sits on the U.S.-Mexico border. A study in a 2007 issue of CDC's *Emerging Infectious Diseases* journal found that past dengue infection had been detected in 40 percent of Brownsville residents. The last reported cases in which health

officials believe residents acquired dengue locally was in 2005, Rodriguez said, noting that the health department has significantly restructured its vector-borne disease surveillance in the last three years. Central to the department's strategy is trapping mosquitoes after rainfalls and sending specimens to the state health department for testing.

Rodriguez said the trapping not only provides data on the species and density of mosquitoes, but helps pinpoint hotspots and gauge if, when and where to use chemical pesticides. Regarding susceptibility to dengue, Rodriguez said there tends to be a disparity between those with central air conditioning and heating and those without. But because Brownsville is home to many low-income residents, prevention education is targeted citywide, from TV spots to leafletting neighborhoods.

"It's under control until the next rainfall, and then you can scratch all that hard work we did and we're back at ground zero," Rodriguez told *The Nation's Health*. "It's a constant state of alert."

Low-income communities are much more susceptible to vector-borne diseases, often due to poor housing conditions and weak community infrastructures. In a 2008 article in the *Public Library of Science Neglected Tropical Diseases* journal, Hotez, of the American Society of Tropical Medicine and Hygiene, called the spread of certain tropical and vector-borne diseases inside the United States "neglected infections of poverty."

Regarding the dengue outbreak in Florida, Hotez said "it's not too much of a stretch to believe that it could emerge along the Gulf Coast," however, he said the biggest determinant of its spread will be poverty.

"My concern is the poorest people on the Gulf Coast," Hotez told *The Nation's Health*. "First we had (Hurricane) Katrina, then the oil disaster, and dengue, in my opinion, could be the third threat."

Despite data that show vector-borne diseases pose a threat, President Barack Obama in his 2011 fiscal year federal budget proposed eliminating funding for CDC's vector-borne disease program. In a letter to key members of the House and Senate, APHA and fellow public health advocates said the proposal has the "potential to leave America vulnerable to some of the world's deadliest diseases."

Vector-Borne Disease Control and Prevention



Vector control interventions have a proven track record of successfully reducing or interrupting disease transmission, particularly in areas that are highly prone.

New strategies for prevention and control of vector-borne diseases are emphasizing "Integrated Vector Management" – as an approach that reinforces linkages between health and environment, optimizing benefits to both. IVM strategies are designed to achieve the greatest disease control benefit in the most cost-effective manner, while minimizing negative impacts on ecosystems (e.g. depletion of biodiversity) and adverse side-effects on public health from the excessive use of chemicals in vector control. Rather than relying on a single method of vector control, IVM stresses the importance of first understanding the local vector ecology and local patterns of disease transmission, and then choosing the appropriate vector control tools, from the range of options available.



These include environmental management strategies that can reduce or eliminate vector breeding grounds altogether through improved design or operation of water resources development projects as well as use of biological controls (e.g. bacterial larvicides and larvivorous fish) that target and kill vector larvae without generating the ecological impacts of chemical use.

At the same time, when other measures are ineffective or not cost-effective, IVM makes judicious use of chemical methods of vector control, such as indoor residual sprays, space spraying, and use of chemical larvicides and adulticides; these reduce disease transmission by shortening or interrupting the lifespan of vectors.



IVM provides a framework for improved personal protection/preventive strategies that combine environmental management and chemical tools for new synergies (e.g. insecticide-treated nets (ITNs)). For example, trials using insecticide-treated bed nets in some malaria-endemic African countries have shown very substantial reductions in child and infant mortality. IVM also supports effective, accessible and affordable disease diagnosis and treatment within the framework of a multi-disease control approach.

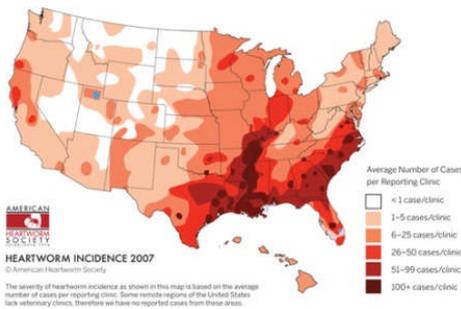
IVM requires a multi-sectoral approach to vector-borne disease control. For instance Health Impact Assessments of new infrastructure development, e.g. water resource, irrigation and agriculture, can help identify potential impacts on vector-borne disease upstream of major policy decisions so effective action may be taken.

IVM is not a panacea. However, in many settings, the use of IVM strategies has yielded sustainable reductions in disease and transmission rates. In addition, certain IVM field experiences have been documented as cost-effective in terms of disease control, and potential generators of economic co-benefits in terms of development and growth – although more work needs to be done linking health and economic outcomes.

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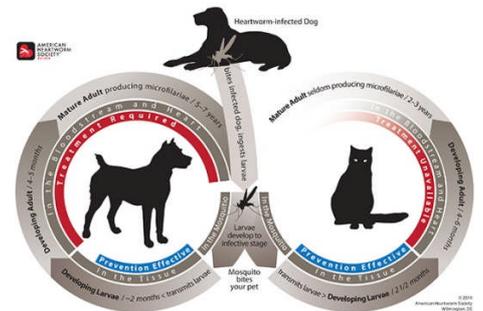
The Pet Corner: Vector-Borne Diseases

Heartworm Disease



Heartworms are parasites that primarily infect the circulatory system of dogs, cats, and ferrets, resulting in a serious and potentially fatal disease.

Heartworm larvae (microfilaria) are indirectly transmitted between hosts (from animal to animal) via mosquitoes (vector).

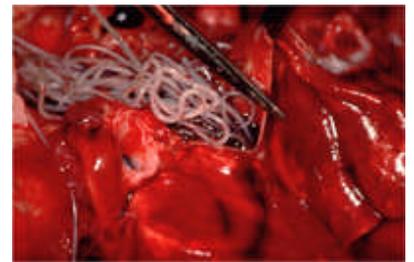


Unfortunately, heartworm disease, although with primary “hot spots” in tropical and sub-tropical climates, has grown to become a global threat with cases reported in each of the 50 United States. What’s more, hosts do not develop protective immunity against the disease despite exposure, meaning all dogs, cats, and ferrets remain susceptible regardless of age, sex, or living environment.

Signs of heartworm disease



Early onset infection is often subclinical. As the disease progresses, clinical signs observed in canine patients include a mild, persistent cough, reluctance to move or exercise, fatigue after only moderate exercise, reduced appetite and weight loss. Those observed in the feline population are often non-specific, mimicking many other feline diseases. Chronic clinical signs include vomiting, gagging, difficulty or rapid breathing, lethargy and weight loss.



Signs associated with the first stage of heartworm disease, as the parasites enter a blood vessel and are carried to the pulmonary arteries, are often mistaken for feline asthma or allergic bronchitis, when in fact they are actually due to a syndrome newly defined as Heartworm Associated Respiratory Disease (HARD).

Preventing heartworm disease



With heartworm disease, prevention is key! From daily and monthly tablets/chewables, monthly topicals and a six-month injectable product (available only for dogs), there are a variety of convenient, inexpensive, and highly effective means to protect your family pet. Each of these medications interrupts the parasites' life cycle prior to adult worm migration to pulmonary vasculature, thereby preventing disease.

And to answer the impending question of “do I need to administer preventatives year round,” the best answer is “yes,” particularly in this climate. The reasoning behind my response is that it only takes one mosquito to cause this devastating disease. In my opinion, spending \$5-10 monthly is miniscule compared to \$1500 in treatment costs if your dog is diagnosed as seropositive. Also, there is currently no medication approved for treatment in cats save for actual surgical removal of the parasite from the heart/lungs.

For additional information, visit the American Heartworm Society's website at <http://www.heartwormsociety.org/>

Cats versus Dogs	Cats	Dogs
Parasite	<i>Dirofilaria immitis</i>	<i>Dirofilaria immitis</i>
Transmission	Mosquito	Mosquito
Susceptibility to infection	Lower than dogs - 61% to 90% of cats exposed to infective larvae become infected.	Very high - virtually 100% of dogs exposed to infective larvae become infected.
Longevity of worms	2-3 years	5-7 years
Ectopic infections	Not uncommon	Occasionally
Number of worms	Usually less than 6, 1-2 worms most common	Not uncommon to find more than 30
Single-sex infections in meso- to high-endemic areas	Common	Unusual
Microfilaremia	<ul style="list-style-type: none"> • Transient (Lasts about 1 month) • Seen in less than 20% of naturally infected cats 	<ul style="list-style-type: none"> • Persistent • Very common (80%-90%)
Organ with greatest pathology	Lungs	Heart and lungs
Clinical importance of small worm burdens	Potentially fatal	Clinical importance depends on the size of the dog, the size of the worm burden, and exercise level
Diagnosis	Complex	Relatively simple
Treatment	<ul style="list-style-type: none"> • None approved 	<ul style="list-style-type: none"> • 1 compound approved
Compounds for prevention	4 approved in US	Several approved in US

West Nile Virus

Vaccination is recommended to protect horses from this deadly mosquito-borne disease

Overview

Mosquitoes transmit a myriad of pathogens, such as Eastern Equine Encephalitis (EEE) and West Nile virus (WNV).¹ In mammals, WNV causes an inflammation of the brain and spinal cord (called encephalomyelitis), and up to 40% of infected horses ultimately die.²

Although WNV has long been recognized throughout the world, the first cases in North America weren't diagnosed until 1999. The virus then spread rapidly throughout the United States and Canada, infecting birds, humans, horses, and other mammals. As of 2012 more than 25,000 horses have been infected since 1999.^{2,3}

The number of WNV cases has decreased steadily following the identification of the WNV and the prompt manufacture of vaccines against the disease.

How WNV Spreads

The WNV is maintained in the wild bird population and, just like with Eastern and Western equine encephalitis viruses, is spread between birds by mosquitoes.³ Birds are therefore referred to as the "natural reservoir" for WNV, because high levels of the virus circulate in their bloodstream. Horses and humans most often become infected after being bitten by a mosquito infected by feeding on an infected bird. Once an infected mosquito bites a horse, the virus enters the horse's bloodstream and spreads to the spinal cord and brain, causing a widespread inflammation.⁴

Unlike birds, infected humans, horses, and other mammals do not have high virus levels in their blood. These animals are called dead-end hosts because they can become infected with the virus, but do not spread the disease to mosquitoes. WNV can also be spread via mechanical transmission, such as through a blood transfusion or vertical transmission from mare to foal.⁶



The American Association of Equine Practitioners recommends vaccinating all horses against West Nile virus.

Clinical Signs

Once a horse is infected, the virus multiplies in the bloodstream, crosses the blood-brain barrier, and invades the central nervous system (brain and spinal cord). Clinical signs of disease typically develop three to 15 days after the horse was initially exposed to the virus.

Classic signs of WNV-infected horses include fever, ataxia (wobbly gait), stumbling, hind limb weakness, lethargy, off feed, recumbency (with the inability to rise), muscle tremors, teeth grinding, dysphagia (inability to swallow), head pressing, signs of colic, a flaccid (limp) paralysis of the lower lip, aimless wandering, excessive sweating, behavior changes, and convulsions or even coma.¹

Diagnosis

Any horse exhibiting abnormal behavior or neurologic signs (such as ataxia or muscle trembling) should be examined by a veterinarian as soon as possible. It's important to rule out other neurologic diseases such as rabies, equine protozoal myeloencephalitis, other viral encephalitides, the neurologic form of equine herpesvirus-1, botulism, wobbler syndrome, among others.

A veterinarian can perform blood tests to help diagnose WNV in horses with clinical signs consistent with infection.⁷

When interpreting test results, veterinarians will also consider a horse's vaccination status, as some tests are incapable of distinguishing between infected and vaccinated horses. Good recordkeeping regarding vaccine history is recommended. While waiting for the test results, veterinarians typically initiate symptomatic treatment.

Treatment

No specific treatment or cure for horses infected with the WNV exists. Veterinary care includes administering anti-inflammatory drugs and, if necessary, intravenous fluids. Supportive care is very important for infected horses to ensure adequate food and water intake, to protect the horse from self-inflicted injuries, and to prevent pressure sores in recumbent horses.

Some veterinarians have attempted treating WNV-infected horses with antiviral drugs such as interferon and passive antibody products. Published clinical trials demonstrating this approach's efficacy or safety are lacking at present, but this is an important and actively researched topic in both human and veterinary medicine.

Prognosis

Compared to humans, who rarely die from WNV, the case fatality rate in horses is higher (as mentioned, approximately 40% in nonvaccinated horses). Recumbent horses are at higher risk of dying than infected horses that remain standing, and older horses reportedly have higher fatality rates.

That said, many infected horses will recover completely following infection (60%), which means that infection with WNV is not a "death sentence." Unfortunately, approximately 40% of horses that recover from the infection experience residual clinical signs such as gait and behavior

abnormalities. In addition, a small percentage (10%) of horses can suffer a relapse after the initial recovery.

Prevention

Although the number of infected horses has declined since the spread of the virus throughout the United States from 1999 through 2003, WNV remains an important disease in unvaccinated horses.^{3,4}

The American Association of Equine Practitioners recommends vaccination of all foals and horses against WNV.^{7,8} Yet some owners elect not to vaccinate their horses against WNV. This may be, at least in part, to the fact that owners do not consider WNV a "real" threat.⁹

Prevention through vaccination of equids against WNV and mosquito control is the most important way to minimize a horse's chances of becoming infected.

In the northern regions of the United States, veterinarians recommend vaccinating horses in the spring prior to peak mosquito levels. In the south, where mosquito populations are present year-round, horses might be vaccinated more frequently.

In addition to geographic location, age

and exposure play important roles in deciding how often to vaccinate horses. Studies have shown that the WNV vaccine has a substantial effect on preventing disease.

Minimizing mosquito populations near your horses by eliminating mosquito breeding and resting areas will make it more difficult for the insects to bite and infect horses and the people who care for them.

For example, reduce or eliminate sources of stagnant or standing water, remove muck from areas near the horses, stable your horses during peak mosquito periods (i.e., dawn and dusk), use equine-approved mosquito repellants, place fans inside the barns or stalls to maintain air movement (mosquitoes don't fly well in wind), keep weeds and grass trimmed, and avoid using incandescent bulbs inside stables at night. Instead, place incandescent bulbs away from the stables to attract mosquitoes to areas away from horses.¹

Finally, discourage wild birds from roosting near or in your stables. Report any dead birds—particularly crows, blue jays, magpies, owls, and hawks—to your local Department of Health. 🍌

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Authored by Stacey Oke, DVM, MS; reviewed by Justa Traub-Dargatzis, DVM, MS, Dipl. ACVIM

Equine Borreliosis (Lyme Disease)

Source: aaep.org; *The Horse*

Lyme disease is caused by a spiral-shaped bacterium called *Borrelia burgdorferi* that is spread to some mammals via the bite of specific hard-bodied ticks. Also known as borreliosis, it is widely considered the most important insect-borne bacterial infection in North America, and the question remains: Do ticks transmit the bacterium to horses and cause disease, as they do to humans and dogs, or is this a disease presumed also to affect horses simply because they coexist in the same environments as the ticks?

Many adult horses in specific geographic areas are seropositive for Lyme disease (i.e., have antibodies in their blood against the bacterium), indicating they are either currently infected or have been infected with *Borrelia*, according to clinicians and researchers such as Thomas

J. Divers, DVM, Dipl. ACVIM, Dipl. ACVECC, professor of medicine and chief of Large Animal Medicine at Cornell University's College of Veterinary Medicine.

Here we'll review the key advances that have been made over the past 10 years and whether equine

borreliosis is a real threat to horses or if, as some researchers contend, Lyme disease poses no threat to horses at all.

What is Lyme Disease?

Biting ticks of the genus *Ixodes* can spread Lyme disease to a variety of mammals. Researchers think humans, dogs, cats, and, presumably, horses are the most commonly infected mammals.

Lyme disease only occurs in areas of the world where both the ticks and reservoir mammalian hosts (typically small rodents) coexist. In North America domestic animals and humans are most commonly diagnosed with Lyme disease along the



Once an infected tick transmits *B. burgdorferi* to a susceptible mammal, the bacteria multiply and begin to migrate through the mammal's body.

eastern seaboard, upper Midwest, Texas, and on the Pacific coast (California).

Ticks hatch uninfected from eggs as larvae. The larvae or nymphs feed on infected reservoir hosts and become infected with *B. burgdorferi*. The bacteria reside in the midgut of infected ticks and are subsequently transmitted back to mammals when any life stage of the infected ticks feeds on animals. Many infections are acquired in the spring and fall, when juvenile or adult ticks feed on mammals the most.

Once an infected tick transmits *B. burgdorferi* to a susceptible mammal, the bacteria multiply and migrate through the mammal's body. The bacteria appear to prefer traveling and residing in the skin, fascia (bands of fibrous connective tissue that bind muscles and organs), and perineural (surrounding nerves) tissues, often winding up in synovial (joint) membranes, where the host's immune system is least likely to recognize and neutralize them. The sneaky spirochete bacteria can also, although less frequently, wiggle their way into the heart, kidneys, and meninges—the thin tissue that covers the brain.

There is currently no approved Lyme vaccine for use in horses, although laboratory testing indicates the canine Lyme vaccine is used in some horses, says Divers.

Signs of the Lyme

Lyme disease can affect any body system, but in horses the musculoskeletal system and possibly the nervous system appear to be favorite target sites for *B. burgdorferi*. The most common clinical signs include:

- Stiffness;
- Mild to moderate lameness in multiple limbs;
- Muscle or nerve pain; and
- Behavioral changes (e.g., dullness).

Other signs of equine borreliosis include chronic weight loss, skin hypersensitivity

and resentment to being touched, uveitis (inflammation in the interior of the eye), and, rarely, joint swelling.

"Most horses show no obvious signs of infection," says Divers. Infected horses can show signs so subtle that attributing things such as mild changes in gait to Lyme disease can be extremely challenging.

Divers adds that a veterinarian shouldn't confuse Lyme disease with anaplasmosis, another tick-borne disease that can cause fever, low blood platelet counts, leg edema (swelling), lethargy, jaundice, and sometimes muscle wasting or incoordination.

Diagnostic Dilemmas

Diagnosing equine borreliosis is challenging due to the vague, nonspecific, and variable clinical signs, and the limitations of available tests. Most of the available tests are based on measuring antibodies—produced by the horse's immune system

against *B. burgdorferi* after tick transmission of the bacterium—that are circulating in the horse's bloodstream. The two main tests are an enzyme-linked immunosorbent assay (ELISA) and a Western Blot.

Divers says, "Some ELISA tests only provide information regarding antibody levels produced against either whole (bacterium) cell or specific antigens, but do not differentiate between past and current infections." It is therefore important to note that the Western Blot and C-6 ELISA SNAP test are the only tests that should be used in horses vaccinated against Lyme disease (i.e., using a canine vaccine not approved for use in horses), as these are the only tests capable of differentiating between antibodies produced in response to the vaccine versus antibodies produced after natural exposure to *B. burgdorferi*.

Divers and colleagues reported in 2008 that an ELISA SNAP kit marketed for dogs

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Facts and Fallacies of LYME DISEASE

is a reasonable stall-side test to screen horses for *Borrelia* antibodies, rather than submitting samples to a laboratory. The advantages of this SNAP test are speed, convenience, and cost; however, there is a moderate chance of false negative results. If a veterinarian strongly suspects a horse has Lyme disease, he or she should test the horse using traditional lab methods.

Scientists describe this further in the free article, "Validation of an in-clinic enzyme-linked immunosorbent assay kit for diagnosis of *Borrelia burgdorferi* infection in horses," available online at www.jvdi.org/cv/reprint/2013/321.

Trick or Treat?

Based primarily on his research involving experimentally infected ponies, Divers recommends treating horses if they have clinical signs and laboratory findings consistent with Lyme disease and if other

diseases have been ruled out.

Treating Lyme disease using antibiotics is advocated and theoretically curative; however, it is not as easy as it sounds. Long-term administration might be required to clear the infection, and this can become expensive. And even following a prolonged course of antibiotic therapy, infection can persist or reinfection can occur.

At present, the first choice antibiotic is intravenous tetracycline (5-7.5 mg/kg/day) for 28 days. The most common treatment, however, is doxycycline at 5-10 mg/kg orally every 12 hours for at least a month.

Fight the Bite!

Since ticks must be attached to the horse for approximately 24 hours to successfully transmit *B. burgdorferi*, aggressive tick avoidance is one of the best ways to protect horses from Lyme disease.

Other preventive measures: Groom and inspect your horse daily for ticks, particularly on the lower limbs and under the mane and tail. Remove and destroy attached ticks, and apply an antibiotic ointment to bite sites if there are skin lesions.

While there is no approved vaccine for use in horses, a 2003 study by Divers and colleagues on experimentally infected ponies showed that a recombinant outer-surface protein A (rOspA) vaccine is effective in preventing Lyme disease.

Take-Home Message

Divers concludes: "Determining if a horse's clinical signs are due to Lyme disease is based upon relating those signs to the preferred location of the organism (i.e., noting damage has occurred in synovial membranes, skin, and nervous system as seen in experimentally infected ponies), serologic or PCR testing, response to treatment, and, most importantly, ruling out other diseases."

According to Divers, treating serologically positive horses that do not meet the other criteria is likely unwarranted. Proper duration of treatment is unknown in field cases because reinfection and duration of infection prior to treatment vary.

The great majority of properly selected and treated horses have clinical improvement, but serum antibodies can persist. Unfortunately, recurrence of clinical signs following treatment is not unusual. ♣

Equine Protozoal Myeloencephalitis (EPM)

Source: aaep.org; UGA-CVM

EPM is the most commonly diagnosed neurologic disease in horses

Overview

Equine protozoal myeloencephalitis (EPM) is a potentially fatal neurologic disease in horses caused by *Sarcocystis neurona* or *Neospora hughesi*.¹ *S. neurona* is more common than *N. hughesi* and more research has been devoted to *S. neurona*; however, both are important and can cause EPM.²

Horses inadvertently become infected with *S. neurona* by ingesting opossum feces containing the parasite's sporocysts. The sporocysts migrate from the horses' gastrointestinal system via the blood and lymphatic system and cross the blood-brain barrier.³ The definitive host of *N. hughesi* is not currently known, but horses are believed to become infected by ingesting sporocysts.

Researchers currently estimate that 50-85% of horses in North America have antibodies in their blood against *S. neurona*, which indicates they have been exposed to the parasite but not necessarily infected.⁴ Not all horses that ingest the sporocysts become infected. Although EPM is currently the most commonly "diagnosed" neurologic disease in horses, it is still quite rare: EPM incidence is estimated to be less than 1%.

Signs of EPM

After being ingested, the sporocysts can damage virtually any region of the brain or spinal cord. The signs of disease depend on where the sporocysts "land" in the brain and/or spinal cord. A key feature of EPM is that the neurologic signs are usually asymmetric (i.e., worse on one side of the body than the other). Signs can be as mild as a slight decrease in performance or as severe as seizures and collapse.

EPM can mimic many other neurologic diseases, including wobbler syndrome (cervical stenotic instability), West



Veterinarians often perform neurologic exams in the early stages of EPM diagnosis.

Nile virus, the neurologic form of equine herpesvirus-1, rabies, and Eastern or Western equine encephalitis, among others.⁵

Master of Disguise

The current "gold standard" test is finding the parasite in the brain and/or spinal cord, which can only be done posthumously. In live horses there are four types of tests available for diagnosing *S. neurona*.⁶ When performed on serum (blood) samples, the Western blot, indirect fluorescent antibody test (IFAT), and enzyme-linked immunosorbent assay (ELISA) simply indicate if a horse has been exposed to *S. neurona*. These three tests do not directly provide proof the horse is currently infected.

If those same three tests are performed using a cerebrospinal fluid sample (CSF, the fluid that bathes the brain and spinal cord), a positive result raises the suspicion that the horse in question is actively infected.

The fourth test, the DNA-based "PCR" test, attempts to detect genetic material from *S. neurona*. If the parasite damaged the spinal cord or brain but was then cleared by the horse's immune system, the horse can still exhibit clinical signs of disease, but it will no longer have detectable parasitic DNA. Thus, a negative PCR test doesn't mean the horse did not, at some point in the past, have EPM.

Experts warn that no single test is perfect and that all test results must be interpreted together with clinical signs. Clearly, diagnosing EPM continues to be challenging; therefore, veterinary researchers continue to modify and improve the current EPM tests in an attempt to minimize the chances of obtaining either a false negative or false positive result.^{7,8} Further, separate tests need to be performed if *N. hughesi* is suspected.

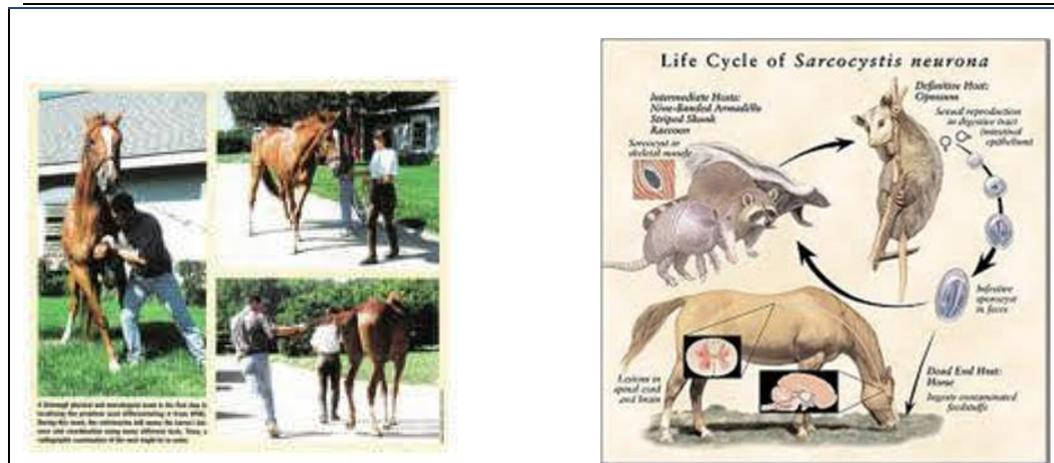
Treatment

Treatment should only be instituted once all other neurologic diseases have been ruled out and if diagnostic tests are highly suggestive of EPM. The three treatment options for horses diagnosed with EPM include (1) a sulfadiazine-trimethoprim-pyrimethamine combination; (2) diclazuril; and (3) ponazuril.^{9,10}

These FDA-approved treatments are indicated for horses infected with *S. neurona*. Infected horses might also be treated with nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroids, dimethylsulfoxide (DMSO), vitamin E, folic acid, and/or complementary and alternative therapies.

Prognosis

Early diagnosis and treatment are key factors that contribute to an acceptable outcome. Says Martin Furr, Dipl. ACVIM, PhD, an associate professor of medicine at



Virginia Tech's Virginia-Maryland Regional College of Veterinary Medicine:

- About 70% of properly treated cases improve;
- One-third of cases that improve achieve a complete recovery;
- Early treatment improves the chances of a complete recovery; and,
- Less severe cases have the best chance for a complete recovery.

Prevention

The microscopic parasites that cause EPM are hardy, and the sporocysts are resistant to even the most intense disinfectants. Prevention is therefore aimed at minimizing contact between horse and opossum feces. Potential EPM risk factors have been reported, including:^{11,12}

- **Age** The highest risk of infection occurred in horses aged 1-5 years;
- **Location** Horses on farms with previously infected horses had a higher risk of developing EPM;
- **Seasonal effects** Fewer EPM cases occur in winter;
- **Stress** An increased risk of EPM associated with stressful events;

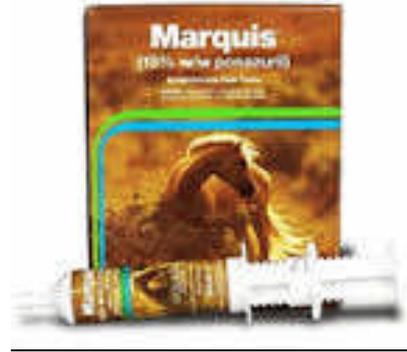
■ **Natural water source** Presence of water sources (creek or river) on farms provided a preferred habitat for opossums;

■ **Food storage** Securing feed and water sources from opossum fecal contamination is important to limit exposure; and

■ **Cats** As a known intermediate host for *S. neurona*, cats have been identified as a risk factor for EPM. But the authors of one study suggest that, while it is possible cats play a role in the natural occurrence of EPM, more research is needed before banning cats from barns.¹³

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About the Editor

I am an equine veterinarian from the great state of Georgia who currently serves as the OIC for the Ft Huachuca Veterinary Treatment Facility.

Recently tasked by our hospital command to "revamp" the RWBACH hospital public information sites, I, with the assistance of our RWBACH health-care providers, will strive to provide you with the most up-to-date, relevant information as it pertains to your overall health and well-being as well as that of your four-legged counterparts. Check back often as I post new articles weekly!

As always, feedback is appreciated on all posts. If you know of news or events that would interest other MEDCOM readers or if there is a topic you would like discussed in the next issue, please contact me via the e-mail address listed below.

Thank you in advance and I hope you enjoy this week's newsletter!

CPT Lauren Baldwin, DVM

