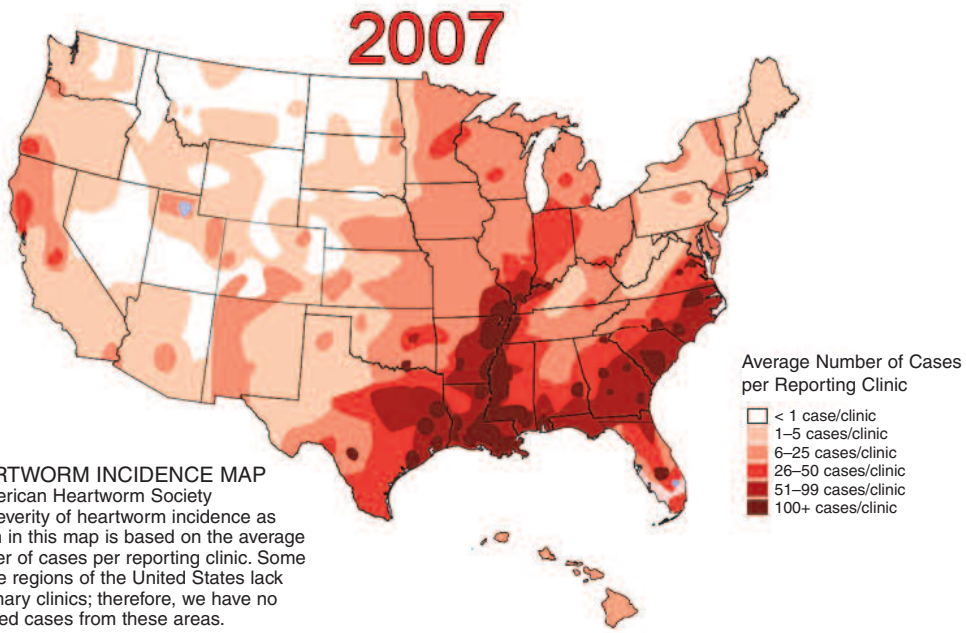
Changes in Vectors Creating an Emerging Heartworm Disease

Emerging Heartworm Disease: Part 1

Heartworm disease was first discovered in 1626 in Italy, reported in dogs in the United States in 1847, and documented in cats by the 1920s (T. Nelson, Heartworm: An emerging disease climactic conditions, reservoir, competent vectors all contributing to disease spread 2010). Heartworm preventive products are highly effective when administered per FDA-approved label directions (Merial Ltd. 2010). Current antigen tests used for disease diagnosis report very high sensitivity levels, ranging from 78 percent to 84 percent for low worm burdens to 97 percent for worm burdens of three or more. Private practitioners routinely use these tests (Atkins 2003). The FDA-approved adulticide treatment for heartworm disease provides 89.7 percent to 98.7 percent efficacy as based on laboratory and clinical field studies (Merial n.d.).

One may inquire why a disease that has been recognized for more than 350 years, that can be diagnosed using extremely sensitive diagnostic tests, that has an effective treatment, and that can be prevented using highly efficacious protocols can be classified as an emerging disease.

An emerging disease can be a disease that is newly recognized or one that demonstrates an increased incidence or geographic range (Nath, et al. 2006). Over the past decade, heartworm disease has demonstrated such an increased incidence and expansion of its geographic range. As indicated in **Figures 1, 2, and 3**, heartworm cases are on the rise. This increase has occurred in both endemic and non-endemic areas. Factors leading to this increase include changes in the vectors of transmission, reservoir host movement, and changes in climate.



HEARTWORM INCIDENCE MAP
© American Heartworm Society
The severity of heartworm incidence as shown in this map is based on the average number of cases per reporting clinic. Some remote regions of the United States lack veterinary clinics; therefore, we have no reported cases from these areas.

Figure 1. 2007 heartworm incidence in the United States.
Source: American Heartworm Society

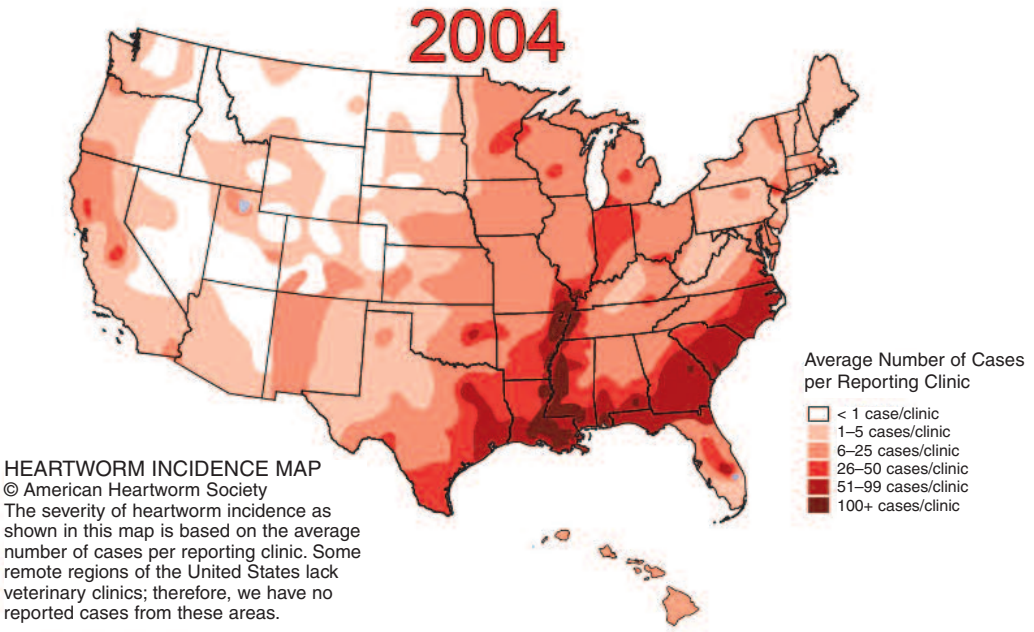


Figure 2. 2004 heartworm incidence in the United States.

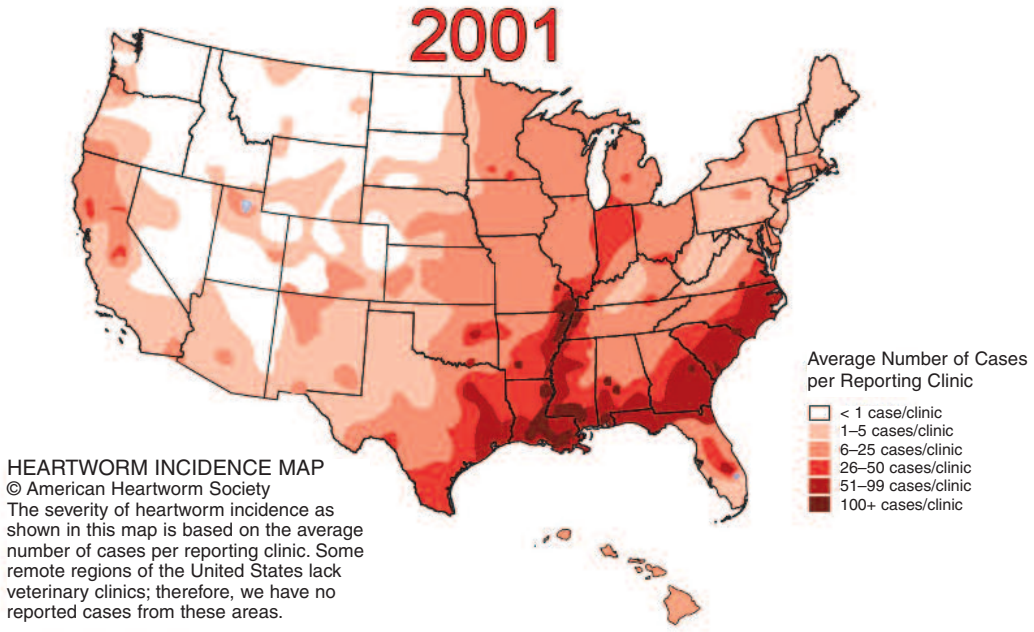


Figure 3. 2001 heartworm incidence in the United States.

Typical hosts for heartworms include dogs and cats with infections reported in other susceptible hosts, including ferrets, sea lions, seals, bears, raccoons, red pandas, coatimundis, wolverines, horses, muskrats, beavers, deer, and primates (Bowman and Atkins 2009).

Although previously confined to more southern climates, heartworm disease has now spread northward and westward. This spread involves expansion to nearly all climates where competent mosquito vectors reside (T. Nelson, What's new in heartworm disease? 2010).

Cases have been documented in all 50 states, and the disease is considered regionally endemic in 48 states, Puerto Rico, the U.S. Virgin Islands, and Guam (American Heartworm Society 2007). The highest infection rates in North America occur in dogs within 150 miles of the Atlantic and Gulf coasts and along the Mississippi River and its major tributaries. High rates of infection can be found in any area with large mosquito populations (American Heartworm Society 2007).

Case reports of heartworm disease are rising in both endemic and non-endemic areas (T. Nelson, Heartworm: An emerging disease climactic conditions, reservoir, competent vectors all contributing to disease spread 2010). According to Tom Nelson, former president of the American Heartworm Society, three things are needed for heartworms to be endemic in an area:

- a mosquito vector that is capable of transmitting the disease
- a reservoir host for infection
- favorable climatic conditions

Other considerations involve potential anthelmintic resistance issues, owner compliance issues, and the increased cases of feline heartworm disease. This publication addresses these and other issues that collectively may be causing this to be considered an emerging disease.

The Life Cycle of the Heartworm

The completion of the lifecycle of the heartworm requires an infected host, a mosquito, and a susceptible host. Two important points to remember about this life cycle include stages at which preventives have action and stages at which adulticides have action. The macrocyclic lactone class of heartworm preventives is effective only on the L_3 to L_4 tissue stages. The juvenile, previously called the L_5, is actually the sexually immature adult heartworm state. This stage is not affected by heartworm preventives.

Adulticide therapy has efficacy against juvenile and adult heartworms 4 months of age or older (Merial n.d.). It is very important to realize that this allows a window of 2 months in which the microfilaricide has no effect on the microfilaria and the adulticide has no effect on the pre-adult. This may explain some treatment failures, resulting in increased case numbers. See **Figure 4**.

Figure 5 depicts the life cycle typically associated with heartworms in the dog and cat. Microfilaria are produced as L_1 within 24 hours after the mating of adult male and female heartworms. After release into circulation and ingestion by a female mosquito, the L_1 then undergoes two separate molts over an 8- to 17-day period. This transformation is temperature-dependent, and if the temperature is not appropriate, molting may not occur. The infective L_3 larvae are deposited into the subcutaneous tissues via hemolymph (mosquito saliva) of the susceptible host as the mosquito takes another blood meal before laying her eggs. Another molt (L_3 to L_4) takes place approximately 3–12 days later. The L_4 tissue larvae take approximately 2 months to complete their final molt into the juvenile stage (L_5). In dogs, it takes 6–7

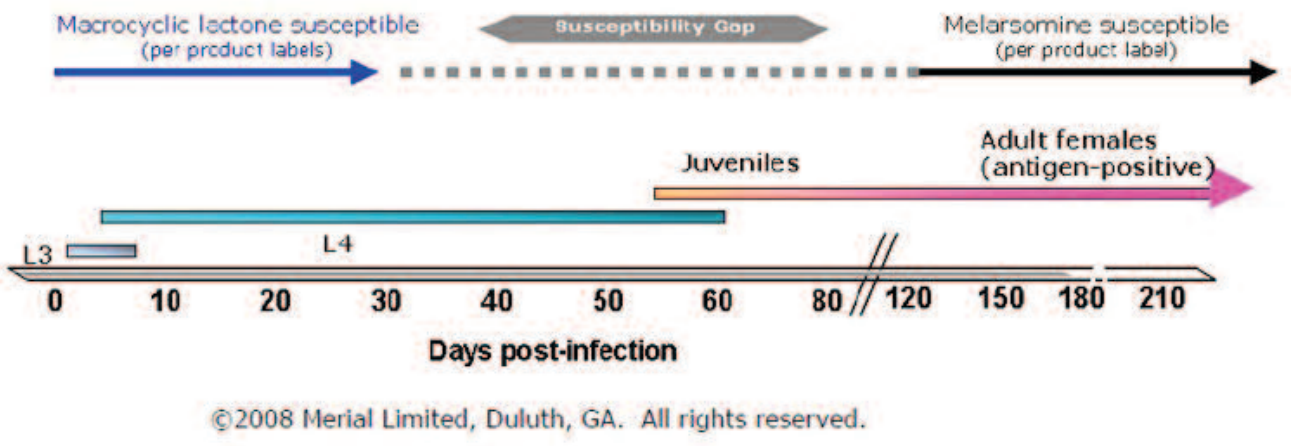


Figure 4. Susceptibility gap of heartworm treatment.

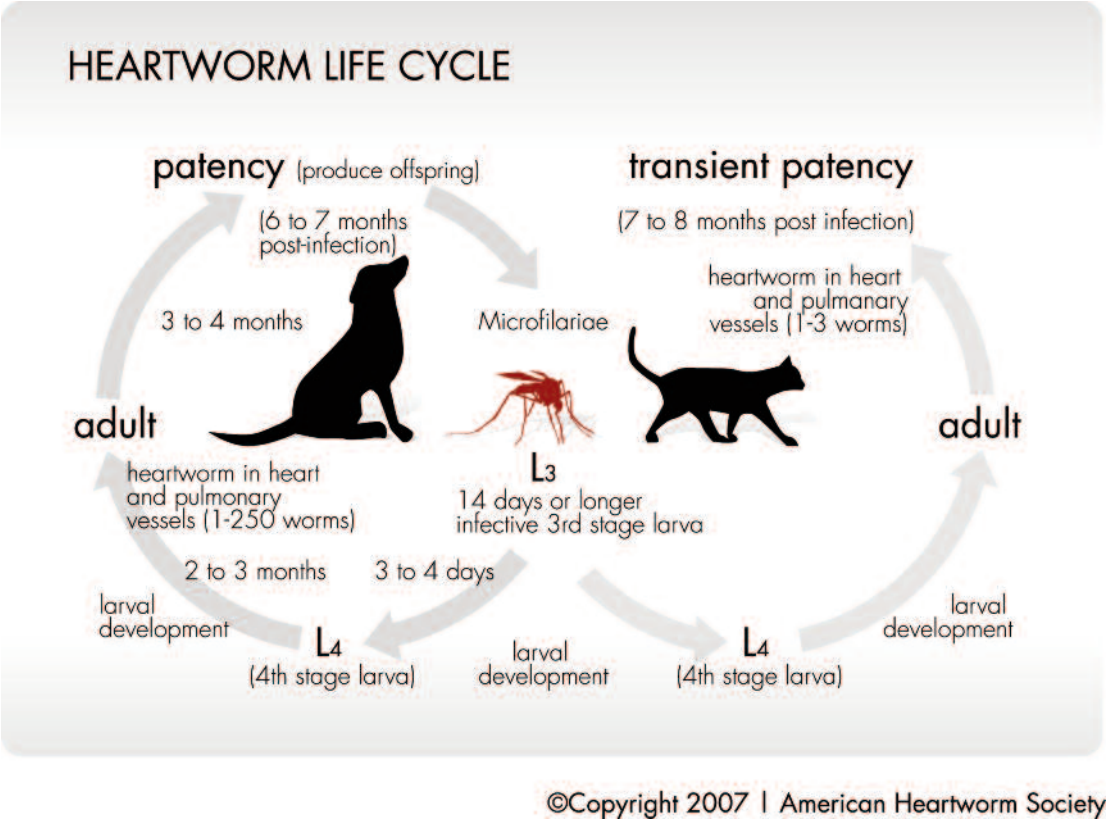


Figure 5. Life cycle of the heartworm.

months for the infection to mature from microfilaria to mature adult, which may live 5–7 years. In cats, it takes approximately 8 months for the infection of L_3 tissue larvae to mature, with the adult heartworm living 2–3 years.

More than 70 species of mosquitoes have been reported capable of transmitting *Dirofilaria immitis*. Twenty-four species are believed to be naturally infected with *D. immitis* in the United States. Different species of mosquitoes are more prevalent in particular geographic locations. *Aedes*, *Anopheles*, and *Psorophora* mosquitoes were commonly described as vectors for *D. immitis* (Hampshire 2005).

Mosquito trapping near the Texas Gulf Coast has determined that *Psorophora* and *Aedes* were found most commonly. Hurricanes and other storm activity in 2000 and 2001 were blamed for introducing saltwater mosquito species such as *Ochlerototatus taeniorhynchus*. This mosquito may have adversely affected the *Aedes* mosquito species in the region (Hampshire 2005).

According to the Mosquito and Vector Management District of Santa Barbara County, the Western Knot Hole Mosquito (*Aedes sierrensis*) is the primary vector of dog heartworm disease in the Pacific Coast region of the United States and parts of Canada, where they appear to be more abundant in heavily wooded areas. Adult Knot Hole mosquitoes are more active at sunset, but they are also active during the daytime.

In 1987, the Asian Tiger mosquito (*Aedes albopictus*) arrived in Texas via car tires from Japan. This mosquito is a day feeder and only requires small containers of water to replicate. It has an incredible ability to adapt to new climates and has very few natural predators. It was implicated as a carrier of flaviviruses (dengue serotypes 1, 2, 3, and 4), Japanese encephalitis, West Nile, and yellow fever viruses. It was initially found in Delaware, Florida, Georgia, South Carolina, Alabama, Mississippi, and Louisiana, but it is now believed to have migrated northward through the Rocky Mountains (American Heartworm Society 2007).

As the presence of mosquito vectors for heartworm disease increases, so do the number of potential vectors for human vector-born diseases. Community-wide mosquito control using pesticides was very common in the 1950s and 1960s. In the 1970s, due to sensitivity to chemicals and human-related sickness, some chemical means of vector control were discontinued in lieu of non-chemical methods. The resulting increase in mosquito populations likely caused an increase in heartworm disease in dogs (Bowman and Atkins 2009).

Conclusion

Canine heartworm disease continues to cause significant morbidity and mortality to the animals of the United States. Despite excellent preventive products, sensitive diagnostic testing, and effective adulticide therapies, the case numbers continue to escalate. Documentation of heartworm disease has demonstrated increased incidence and expansion of geographic range. We reviewed the effect of new vectors relative to the emergence of heartworm disease in dogs and cats. In Extension Publication 2644, we will review the relationship of reservoir hosts and climate changes on the emergence of heartworm disease. Please talk to your veterinarian for prevention and treatment recommendations for your particular situation.

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