

Altered Behavior in Gopher Tortoises with URTD

For several years, SCWDS has been working with collaborators at the Jones Research and Ecological Center (JERC) at Ichuaway in Baker County, Georgia, and at the University of Georgia's D.B. Warnell School of Forestry and Natural Resources to investigate the distribution, prevalence, and impact of upper respiratory tract disease (URTD) in gopher tortoises. Tortoises with URTD have mild to severe nasal and ocular discharge, conjunctivitis, and swelling of the eyes and nares. At least two species of *Mycoplasma* (*M. agassizii* and *M. testudineum*) can cause URTD, which has been associated with gopher tortoise die-offs in Florida and also has been observed in Georgia, Louisiana, and Mississippi.

Previously, we reported that *M. agassizii* was present in numerous tortoise populations in Georgia, and that the prevalence was either very low (3% or less) or very high (96-100%), depending on the population (SCWDS BRIEFS Vol. 27, No. 1). In contrast, most populations had antibodies reactive to M. testudineum with seroprevalence ranging from 38% to 61%, and only one site had 0% prevalence. Tortoises at five sites tested positive for antibodies to both pathogens, and these were the only sites where we observed tortoises with clinical signs of Interestingly, tortoises at sites where URTD. clinical signs of URTD were not observed had against М. antibodies onlv testudineum. suggesting this organism may be of limited pathogenicity for gopher tortoises.

In an effort to better understand long-term impacts of URTD on tortoise populations, we conducted a home range and behavioral study of individual tortoises at Ichauway, where there is a historically high prevalence of *M. agassizii*, the most common pathogen associated with URTD.

At Ichauway, $\geq 92\%$ of the population was seropositive in 1997, and we found a similarly high prevalence in our recent study. The consistently high prevalence allowed us to evaluate the long-term implications of *Mycoplasma* exposure for a population, as well as the short-term consequences of recurrent, severe clinical disease.

In the late-1990s, Craig Guver (Auburn University) and collaborators investigated the home range size and behavior of gopher tortoises at JERC. Initial studies were focused in a 50 hectare (ha) area called Green Grove (GG), where the tortoise density was 1.3 /ha. At that time, the prevalence of antibodies against M. agassizii in tortoises at GG was 96%. Thirty tortoises in GG were outfitted with radio transmitters and temperature loggers. After a year of tracking, GG females had a mean home range size of 0.4 ha, whereas the mean for males was 1.1 ha. When we repeated this work in 2011, the *M. agassizii* antibody prevalence was 92%, the density had increased to 2.08 tortoises/ha, but the mean home ranges had not changed significantly (14 females: 0.81 ha; 16 males: 1.87 ha).

We also compared the behavior of tortoises in GG with mild or no signs of URTD with the behavior of severely affected tortoises from additional areas of JERC. Four females and six males with severe URTD were radio-tracked and temperature-logged from three to 334 days. Contrary to what has been hypothesized in the literature, some of the tortoises with severe disease traveled long distances, and some had large home ranges. The mean home range of severely affected tortoises (134.71 ha) was significantly larger than that of asymptomatic and mildly affected tortoises. but there was considerable variation among individuals with severe URTD (range= 0.09-489.69 ha). Severely

affected tortoises made many long distance movements (165 m to 3,498 m) over short periods of time, compared to the other group, in which the maximum movements ranged from 78.49 to 147.29 m. The longest documented movement in one day for a tortoise with severe URTD was 755 m.

We also detected а difference in thermoregulatory behavior (basking versus cooling in burrows) between the groups when we recorded the hourly temperature on the carapace of the tortoises. Data from individual tortoises were compared to the average of the study group to determine how often a particular tortoise exhibited abnormal thermoregulatory behavior (e.g., was outside of burrow basking when remaining individuals were inside burrows, or remained in a burrow cooling for unusually long periods of time). We found that tortoises in GG with mild or no signs of URTD had similar behaviors with overlapping temperature data, but temperature data of tortoises with severe URTD frequently deviated from that of the other tortoises. The greatest temperature range experienced by a tortoise with severe URTD was from 9.6°C to 42.6°C over the 311 days it was tracked.

In summary, gopher tortoises with severe URTD at JERC had significantly larger home ranges, traveled longer distances, and experienced greater deviation in carapacial temperatures than asymptomatic/mildly affected tortoises, suggesting they spent more time basking than tortoises with no clinical signs. Any of these behavioral changes could decrease condition and health.

Unfortunately, five of the ten tortoises with severe URTD died in their burrows or had to be euthanized humanely during the studv. Collectively, our data suggest that although severe URTD can alter behavior and kill individuals, this tortoise population remained stable despite a high prevalence of antibodies against M. agasizzii. Future work on disease recrudescence and recovery, as well as interactions with habitat quality, is needed to better understand the long-term consequences of URTD on tortoise populations. (Prepared by Jessica McGuire and Michael Yabsley)

Snake Fungal Disease

Since 2006, increasing numbers of free-ranging snakes in the eastern and midwestern United States have been diagnosed with severe fungal dermatitis, a syndrome known as Snake Fungal Disease (SFD). To date, SFD has been confirmed in Florida, Illinois, Massachusetts, Minnesota, New Jersey, New York, Ohio, Tennessee, and Wisconsin. Snakes with SFD may have scabs or crusty scales, subcutaneous nodules, abnormal molting, white opaque cloudiness of the eyes not associated with molting, and/or localized thickening and crusting of the skin. Lesions typically are most severe on the head, but distribution can vary. Infections have been found in the northern water snake (Nerodia sipedon), eastern racer (Coluber constrictor), rat snake (Pantherophis obsoletus species complex), timber rattlesnake (Crotalus horridus), massasauga (Sistrurus catenatus), pygmy rattlesnake (Sistrurus miliarius), and milk snake (Lampropeltis triangulum). Snake fungal disease is not known to affect any other orders of reptiles. The fungus is not transmissible to humans, pets (aside from captive snakes), or livestock.

Chrysosporium ophiodiicola, newly а characterized species of Chrysosporium, is consistently isolated from snakes with SFD. However, there is no conclusive evidence that C. ophiodiicola is the causative agent for this disease, and other fungal species have been isolated from Although affected snakes. Chrysosporium species routinely are isolated from normal reptile skin, certain species have been known to act as a primary pathogen. One example is "yellow fungus disease" of bearded dragons caused by the Chrysosporium anamorph of Nannizziopsis vriesii. Further research is needed to determine if C. ophiodiicola can act as a primary pathogen, or if it requires co-infection with other fungi to cause SFD. The role of environmental conditions, immunosuppression, genetic susceptibility, and other potential risk factors remain to be evaluated.

Chrysosporium ophiodiicola first was isolated and characterized in a captive black rat snake (*Elaphe obsoleta obsolete*). The snake was caught in Sparta, Georgia, and remained in captivity for four years before developing prolonged anorexia and slow-growing facial masses. The masses were removed surgically and were sent to a diagnostic lab for histopathology and culture. The fungal isolate did not match any of the known *Chrysosporium* species already identified, and the new species name was suggested from the Greek for snake, *ophio*.

One of the first reports of SFD in free-ranging snakes described four eastern massasauga rattlesnakes (*Sistrurus catenatus catenatus*) with facial swelling and disfiguration. *Chrysosporium ophiodiicola* was cultured from all four snakes, with no additional pathogens isolated. Three of the snakes were found dead near Carlyle, Illinois, during a routine survey in 2008. Another snake with similar lesions was found alive in the spring of 2010, treated with antifungals and supportive care, and was still alive at the time of the report.

Endangered New England populations of timber rattlesnakes also have been affected by SFD. In 2009, many of the snakes being monitored had lesions on the head, neck, and body resembling those on the massasaugas. Although *C. ophiodiicola* was cultured from these snakes, additional fungal species were isolated.

Questions remain about the impact of SFD impact on free-ranging snake populations. At this time, most reported incidents have been sporadic and involved individual snakes. The solitary nature of snakes makes surveillance and detection of diseased individuals difficult. Further monitoring is necessary to determine if SFD will continue to occur as occasional isolated cases, or if it will become more widespread in greater numbers of snakes. Although snake populations overall have not been affected by SFD to date, certain isolated subgroups have been impacted and appear threatened by the disease. The small and isolated New England timber rattlesnake populations are of particular concern. For example, the New Hampshire subpopulation decreased in size by 50% from 2006 to 2007, following infections consistent with SFD.

There is much more to learn about SFD at this point. The causative agent and pathogenesis of SFD, risk factors for infection, and its future impact on snake populations remain obscure. Surveillance programs and submission of samples to diagnostic laboratories are crucial to track the disease. Additional research and monitoring are vital to better understand SFD and to determine if management intervention is necessary or possible. (Prepared by Amanda Dolinsky, University of Illinois College of Veterinary Medicine, and Lisa Last)

Feral Swine Brucellosis Review Available

Commercial swine in the United States have been recognized as free of Swine Brucellosis (SB) since 2011; however, SB has historically been present in feral swine and recent surveys conducted by APHIS-Wildlife Services have found SB positive feral swine in 18 states. Based on data collected through the National Feral Swine Mapping System (www.feralswinemap.org), established populations of feral swine currently are present in 36 states. Feral swine carrying SB present a widespread and growing risk for disease transmission to commercial swine.

A review just published in the Journal Veterinary Microbiology summarizes brucellosis in feral swine and describes prospective genomic techniques that may be useful in understanding brucellosis epidemiology (Leiser et al., 2013. Feral swine brucellosis in the United States and prospective genomic techniques for disease epidemiology. Veterinary Microbiology 166:1-10). The paper reviews brucellosis in feral swine and wild boar, transmission of *B. suis* among feral swine, *B. suis* seroprevalence in feral swine, traditional methods of detecting and typing brucellae, and contemporary molecular genetic approaches to Brucella detection.

The development of contemporary molecular genetic approaches allows for high-resolution Brucella is a genetically genotyping studies. monomorphic genus, but with sufficient differences for population genetic analyses. New genomic techniques will facilitate studies of population-level relationships В. of suis, determination of the direction of SB movement, and an evaluation of B. suis dispersal in feral and domestic swine. These new advances should allow for a better understanding of B. suis transmission among feral swine and between feral and domestic swine, and they will help aid identify transmission routes and in development of measures to prevent transmission to domestic swine. (Prepared by Joe Corn)

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