

Black Bear Population Health Monitoring in the Southeast

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Master of Science
Degree

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ABSTRACT

Recent growth of the American black bear (*Ursus americanus*) population in the southeast raises concerns about the potential spread of density-dependent diseases among bears, particularly sarcoptic mange. However, research on the health of bears in this area is limited. We analyzed samples from 169 bears in the region. We performed Knotts tests, skin scrapes, fecal floats; Canine adenovirus (CAV), canine parvovirus (CPV), canine distemper virus (CDV), and *Toxoplasma gondii* serology, *Trypanosoma*, *Rickettsia rickettsii*, and apicomplexan PCR, and identified ecto- and endoparasites found grossly or histologically. We found that 69% (63 of 91) had microfilaria; genetic analysis identified the microfilaria as a novel *Mansonella ozzardi*-like nematode. We performed 122 fecal floats and found 35 (28.7%) had *Baylisascaris transfuga*, 16 (13%) had Capillarid spp., 11 (9%) had nematode larva, 2 (1.7%) had *Ancylostoma* spp., and 4 (3.3%) had *Coccidia* spp. No *Sarcoptes scabiei* mites were identified on skin scrapes; however, *Ursicoptes americanus* mites were identified on six (6.4%). *T. gondii* antibodies were detected in 49.7% (78 of 157) of bears. CPV, CAV, and CDV antibodies were detected in 2.1%, 2.1%, and 18.8% of bears, respectively. Of the 113 bears tested, 10 were PCR-positive for *Babesia* (8.9%). All bears were PCR-negative for *Rickettsia rickettsii* and *Trypanosoma* spp. Collected ticks were morphologically identified; 64.5% of bears (69 of 107) were infested with *Dermacentor variabilis*, and 4.7% (5 of 107) were infested with *Ambylomma maculatum*, 0.9% (1 of 107) were infested with *Ambylomma americanum* and 0.9% (1 of 107) were infested with *Ixodes scapularis*. Additionally, endoparasites identified on autopsy or histology include *Macracanthorhynchus ingens*, *Gongylonema* spp., *Physaloptera* spp., *Dirofilaria immitis*, *Trichinella* spp., *Angiostrongylus vasorum*, *Pelodera* spp., and *Sarcocystis* spp. This research

will lay the foundation for future research on bear diseases in the southeast and aid wildlife managers with management decisions on free-ranging black bear populations.

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CHAPTER ONE

INTRODUCTION

The southern Appalachians are known for their abundant American black bear (*Ursus americanus*) population and the bears are a major draw for many visitors to the area. In addition to their cultural importance, black bears are also important ecologically. As long-lived, broad ranging omnivores black bears may serve as sentinels for the health of their environment. Though black bear populations had previously declined in the 1800s due to habitat loss and over-hunting, populations in the southeast have been increasing since the 1970s due to protection and habitat recovery (Humm and Clark 2021). This recent population growth has led to high densities of bears in the area. This increased bear density raises concerns about the potential spread of density dependent diseases among bears. However, there has been limited research on the health and diseases of bears in this area. One disease of concern for American black bears is sarcoptic mange. Sarcoptic mange, caused by the mite *Sarcoptes scabiei*, was previously thought to be rare in American black bears. However, cases of sarcoptic mange in black bears have been expanding throughout the eastern United States over the past decades (Niedringhaus et al. 2019b). Sarcoptic mange is highly contagious and can cause severe, potentially fatal, disease in black bears. Black bears with sarcoptic mange are often reported to have significant alopecia, skin crusting, and poor body condition (Schmitt et al. 1987; Peltier et al. 2018). In 2014 fifty-six bears were reported to have died or been euthanized due to sarcoptic mange in Pennsylvania (Peltier 2014). The cause of the emergence and expansion of sarcoptic mange in black bears is currently unknown.

Furthermore, there are a variety of other pathogens known to infect black bears. *Demodex ursi* and *Ursicoptes americanus* are two mites also known to cause mange in black bears. *Pelodera* spp., a rhabditid nematode, may also cause alopecia and dermatitis in bears. *Sarcocystis* spp., a tissue-cyst forming coccidia, has been associated with hepatitis, myositis, and encephalitis, in black bears in California, Nevada, and British Columbia. Prevalences of this parasite in black bears in these areas have reached up to 40% (Lee et al. 2021; Sinnott et al. 2022). Canine adenovirus-1 has also been associated with severe clinical signs, including ataxia, convulsions, and vomiting, in black bears, and was considered the cause of death for 28 black bears in a South Dakota Park in 1983 (Collins et al. 1984). Though, CAV antibodies have since been detected in black bears across the United States with no associated clinical signs (Dunbar et al. 1998; Bronson et al. 2014; Niedringhaus et al. 2019c). There are additionally, many pathogens that commonly infect black bears that rarely cause clinical disease, including *Baylisascaris transfuga*, *Toxoplasma gondii*, *Trichinella* spp., *Dirofilaria ursi*, and a variety of tick species. Many of these pathogens are also known to infect humans and domestic animals. *Toxoplasma gondii* and *Trichinella* spp., in particular, are known to cause severe disease in humans. Additionally, the tick species known to parasitize black bears are common vectors for a variety of zoonotic pathogens including *Rickettsia rickettsii*, *Borrelia burgdorferi*, *Ehrlichia* spp., *Babesia* spp., and *Anaplasma* spp. Increasing human-bear interactions due to increasing bear density and increasing urbanization, as well as black bears' role as a game species make the presence of potentially zoonotic pathogens of particular concern. Additionally, the risks some of these pathogens pose to bear, and potentially human health are not fully understood. Monitoring the black bear population for certain parasites and pathogens helps wildlife managers to better manage the population by identifying diseases of concern, as well as any risks to public health,

such as the presence of certain zoonotic pathogens. Furthermore, health monitoring will help establish a baseline that will allow researchers and wildlife managers to identify any changes in the health or pathogen prevalences in this population. This information is critical for making timely, informed decisions regarding population health.

CHAPTER TWO

LITERATURE REVIEW

Black bears are known to harbor a variety of parasites and pathogens, many of which are also known to infect humans and domestic animals. The presence of certain pathogens in black bear populations may have bear, human, and domestic animal health implications. The following is a summary of what is known about certain important diseases and parasites in free-ranging American black bears in the southeastern United States.

Ectoparasites

I. Sarcoptes Scabiei

Sarcoptes scabiei is a parasitic mite and the cause of scabies in humans, and sarcoptic mange in domestic and wild animals. The mite is transmitted through direct contact between infected and susceptible hosts, as well as through indirect contact through fomites or a shared environment. Once on a suitable host, the mite will burrow under the skin and cause pruritis, alopecia, and hyperkeratosis. Emaciation, secondary bacterial and fungal infections, and, eventually, death can occur in some hosts. *Sarcoptes scabiei* is distributed worldwide and can infect a large variety of mammalian species. In North America, *S. scabiei* primarily affects canids, including red foxes, coyotes, and wolves, and is considered endemic in many of these populations. Despite this, epizootics do occur and do have the potential to cause severe impacts in naïve populations (Niedringhaus et al. 2019a).

As stated previously, sarcoptic mange was previously considered rare in American black bears, however, cases have been increasing in the eastern United States since the 1990s. Cases have been reported in Pennsylvania, New York, West Virginia, Virginia, Arkansas, and

Oklahoma (Niedringhaus et al. 2019b). The cause for this emergence and expansion of sarcoptic mange in bears remains unknown. Clinical mange has been shown to not be associated with coinfections with canine adenovirus, canine parvovirus, canine distemper virus, *Toxoplasma gondii*, or *Trichinella spiralis* (Niedringhaus et al. 2019c). Additionally, genetic analysis of *S. scabiei* mites collected from bears suggests that there are multiple strains of *S. scabiei* circulating among black bears, and thus the emergence and expansion of this parasite is not due to a single bear-adapted strain (Peltier et al. 2017). In the southeastern United States cases of sarcoptic mange have only been confirmed in Virginia and West Virginia. Additionally, antibodies to *S. scabiei* have been detected in asymptomatic black bears in North Carolina. Bear density may play a role in the emergence of the disease as increased interactions with other mammalian hosts such as red foxes (*Vulpes vulpes*) and coyotes (*Canis latrans*), as well as with other black bears, due to high densities may result in increased transmission. Increasing bear populations have been reported in states with increasing cases of sarcoptic mange (Hristienko and McDonald 2007; Niedringhaus et al. 2019b). Black bears in the southern Appalachians may be at risk of the disease due to similar increasing populations (Hristienko and McDonald 2007).

II. *Ursicoptes americanus* and *Demodex ursi*

In addition to *S. scabiei*, two other mites have been implicated in cases of mange in American black bears, *Ursicoptes americanus* and *Demodex ursi*. *Ursicoptes americanus* is the cause of audycoptic mange in black bears. The mite was first described in a subclinical captive black bear in Kansas in 1970 (Yunker et al. 1980). Audycoptic mange was first reported in a free-roaming black bear in Idaho. The bear was reported to have lesions on its muzzle, head, neck, thorax, and front legs consisting of alopecia, lichenification, and exfoliation. The bear was recaptured 13 months later and, while *Ursicoptes* mites were still present, all lesions were

notably improved, possibly indicating the pathogen is self-regulating (Yunker et al. 1980). Further cases of audycoptic mange were reported in captive black bears in California and Virginia (Gillespie et al. 1984; Joyner et al. 2004). *Ursicoptes americanus* is also reported to have been found on free-roaming bears in Pennsylvania (Peltier 2014). There is still much unknown about *U. americanus*, including its significance for black bear populations.

Demodex ursi is the cause of demodectic mange in black bears. Demodectic mange was first reported in a single bear in Wisconsin with moderate alopecia in 1978 (Manville 1978). It has since been reported exclusively in Florida black bear populations (Forrester et al. 1993; Desch 1995; Foster et al. 1998). Similar to *U. americanus* there is still much unknown about *D. ursi* and how it may impact black bear populations.

III. Ticks

In addition to mites there are a variety of other ectoparasites known to infest American black bears. *Amblyomma americanum*, *Amblyomma maculatum*, *Dermacentor variabilis*, *Dermacentor albipictus*, *Dermacentor andersoni*, *Ixodes affinis*, and *Ixodes scapularis* have all been reported to parasitize black bears in North America (Jonkel and Cowan 1971; Rogers 1975; Yabsley et al. 2009). The presence and prevalence of tick species vary by season and region. In the southeastern United States all but *D. albipictus* and *D. andersoni* have been reported in black bears, with *D. variabilis* and *A. americanum* often having the highest prevalences (Forrester 1992; Yabsley et al. 2009; Nims and Durden 2011; Hertz et al. 2017; Lockwood et al. 2018). Additionally, these ticks can potentially transmit a variety of tick-borne pathogens, such as *Rickettsia rickettsii*, *Borrelia burgorferi*, *Erhlichia* spp., *Babesia* spp., and *Anaplasma* spp., some

of which are zoonotic. As long-lived species with the capacity to travel great distances, bears may play a role in the spread of ticks and tick-borne diseases.

Bacterial Diseases

I. *Rickettsia rickettsii*

Rickettsia rickettsii is a bacterial pathogen that is the cause of Rocky Mountain Spotted Fever (RMSF), an important zoonotic disease. *R. rickettsii* is most often spread by *D. variabilis* (the American dog tick), which commonly parasitize black bears across North America. Antibodies to *R. rickettsii* have been detected in black bears in Idaho and Maryland at low prevalences (2% and 6.6%) (Binninger et al. 1980; Bronson et al. 2014). Additionally, other *Rickettsia* spp., including *Rickettsia parkeri*, the causative agent of *R. parkeri* rickettsiosis, have been detected in a wide range of tick species parasitizing black bears throughout Georgia, Florida, Louisiana, New Jersey, and Kentucky (Yabsley et al. 2009; Leydet and Liang 2013; Chern et al. 2016; Lockwood et al. 2018).

Endoparasites

I. Protozoal Parasites

a. *Toxoplasma gondii*

Toxoplasma gondii is a protozoal parasite that infects a majority of bird and mammal species. *Toxoplasma gondii* is zoonotic and can cause significant disease in humans, primarily immunocompromised people and neonates. *Toxoplasma gondii* is spread through two primary methods: either through ingestion of sporulated oocysts that are shed in the feces of felids (the definitive host) and have contaminated food or water, or through the ingestion of bradyzoites that have encysted in the muscle of an intermediate host. *Toxoplasma gondii* has been monitored extensively in black bear populations throughout the U.S. since the 1970s, using a variety of

serological tests including IHAs, IFAs, ELISAs, LATs, and, primarily, MATs. Data from these studies show that bears have one of the highest seroprevalences of *T. gondii* of all animals (Dubey et al. 2021). This high seroprevalence may be attributed to increased exposure due to bears being omnivorous scavengers; Similar high seroprevalences have been reported in other omnivorous scavengers including raccoons, red foxes, and wild boars (Mitchell et al. 2006; Kornacka-Stackonis 2022). Serological surveys for *T. gondii* in black bear populations have been conducted in Alaska, California, Florida, Idaho, Kentucky, Maryland, Nevada, New Mexico, North Carolina, Oklahoma, Pennsylvania, Tennessee, and Wisconsin. Seroprevalences range from 5.7% (23 of 303 in Idaho) to 100% (4 of 4 in Florida), with an average seroprevalence of 56% (Dubey et al. 2021). Dubey et al., 2021 does an excellent job summarizing what is currently known about toxoplasmosis in bears. Since Dubey et al., 2021 has been published *T. gondii* has also been reported in bears in Wisconsin at a 53% seroprevalence (Sikes et al. 2022). As game species, black bears infected with *T. gondii* have the potential to cause toxoplasmosis in humans that ingest them. Cases of toxoplasmosis from the ingestion of wild game, including black bears, have been reported (Conrady et al. 2022). Furthermore, carcasses of black bears infected with *T. gondii* may also serve to spread *T. gondii* in the environment through scavenging. Clinical toxoplasmosis has only been reported once in black bears, in an 8-month-old orphaned black bear cub found in New Jersey reported to have neurological impairment. Histologic examination of the lungs, cerebrum, and cerebellum of the cub revealed multifocal necrosis associated with *T. gondii* cysts and tachyzoites (Huffman and Roscoe 2014).

b. *Babesia* spp.

Babesia are protozoal parasites in the phylum Apicomplexa that invade red blood cells and cause the disease babesiosis. Babesiosis is a tick-borne disease that is spread by Ixodid ticks.

There are numerous species in the genus *Babesia*, some of which can cause babesiosis in humans. In North America, *Babesia microti* is the primary cause of babesiosis in humans (Yabsley and Shock 2013). *Babesia* was first reported in American black bears in New Jersey in 2015 (41.8% prevalence) (Shaw et al. 2015). It has since been reported in black bear populations in northwestern NJ in 2015 and 2016 (9.2% and 7.1% prevalence, respectively), as well as in Oklahoma (6% prevalence), and North Carolina (16.7% prevalence) at lower prevalences than initially reported (Zolnik et al. 2015; Chern et al. 2016; Skinner et al. 2017; Westmoreland et al. 2019). Additionally, *Theileria*, a closely related protozoal parasite, was detected in one bear in North Carolina (Westmoreland et al. 2019). *Babesia* species detected in North Carolina and Oklahoma black bears were reported to be genetically similar to *Babesia* species found in raccoons and a maned wolf (*Chrysocyon brachyurus*) (Skinner et al. 2017; Westmoreland et al. 2019). There have been no reported cases of clinical babesiosis in American black bears, however clinical babesiosis has been reported in a captive Malayan sun bear (Zubaidah et al. 2020). There are numerous wildlife species that act as reservoirs for *Babesia* (Yabsley and Shock 2013). The role of American black bears as a potential reservoir of this disease is currently not fully understood.

c. *Trypanosoma* spp.

Trypanosoma spp. are protozoan parasites that infect a wide range of mammals. *Trypanosoma cruzi* is the cause of Chagas disease, a zoonotic disease of the heart and gastrointestinal tract that is potentially deadly. In the southeastern United States there have been several cases of autochthonous human infections. Additionally, *T. cruzi* has been detected in a wide range of wildlife in the southeast including raccoons, opossums, armadillos, skunks, coyotes, and bobcats (Bern et al. 2011; Busselman and Hamer 2022). *Trypanosoma cruzi* has not

been reported in black bears; however, *T. cruzi* has been reported in a captive polar bear in Mexico, that died of acute chagas disease (Jaime-Andrade G. et al. 1997). Additionally, *Trypanosoma evansi* has been reported to cause clinical signs including increased heart rate and reduced appetite in several captive Asiatic black bears (*Ursus thibetanus*) in Pakistan (Shahid et al. 2013). The role of American black bears as a potential reservoir of this disease, as well as how the disease may impact black bear populations in the southeast is currently not fully understood.

d. Coccidia spp.

Coccidia is a subclass of protozoal parasites in the phylum Apicomplexa. Coccidia are intracellular parasites that often reside in the gastrointestinal tract of their vertebrate hosts. *Eimeria albertensis* and *Eimeria borealis* were the first coccidian species described in black bears. Both were reported in black bears in Alberta, Canada in 1969, at 8% and 6% prevalences, respectively (Hair and Mahrt 1970). Since then, there have been no further reports of either in free-ranging black bear populations. More recently, a *Cystoisospora*-like parasite was detected in two bears with encephalitis surveyed in California and Nevada (Sinnott et al. 2022). *Cryptosporidium parvum*, a parasite with a coccidian lifecycle is known to cause gastrointestinal disease in a wide variety of animals, including humans, has been reported in one black bear cub in Virginia in 1999. The cub was found dead and *C. parvum* was identified in tissue sections of the small intestine (Duncan et al. 1999). Genetic analysis of the *C. parvum* isolate revealed that it was a new genotype, similar to *C. parvum* isolates from dogs (Xiao et al. 2000). Additionally, *Sarcocystis* spp., a tissue-cyst forming coccidia, has been reported in black bears. *Sarcocystis* spp. was first reported in black bears in the southeastern United States, where sporozoan cysts, tentatively identified as *Sarcocystis* spp., were found in the cardiac and skeletal muscle of six

bears (Crum et al. 1978). In 2008, a new species of *Sarcocystis*, *Sarcocystis ursusi*, was described in two black bears in Pennsylvania, and another unidentified *Sarcocystis* spp. was found in a third bear (Dubey et al. 2008). *Sarcocystis* spp. have also been reported in the muscles of free-ranging black bears in North Carolina, Florida, and Oregon at low prevalences (1.1-4.3%) (Dubey et al. 1998; Foreyt et al. 1999; Cheadle et al. 2002). *Sarcocystis* spp. associated with histopathologic lesions, including hepatitis, myositis, and encephalitis, have been reported in black bears in British Columbia at a much higher prevalence (40%) (Lee et al. 2021). *Sarcocystis* spp., including *S. neurona*, *S. canis*, and *S. felis*, have also been detected in the brains of bears with encephalitis in California and Nevada (Sinnott et al. 2022). Furthermore, *Sarcocystis* spp. have been associated with fatal protozoal hepatitis in two captive black bears in South Dakota and British Columbia (Zeman et al. 1993; Davies et al. 2011). The *Sarcocystis* spp. from the bear in British Columbia was reported to have 100% identity with *Sarcocystis canis* and *Sarcocystis arctosi*.

II. Helminths

a. Filarial Nematodes

Dirofilaria ursi is a filarial nematode of bears. *Dirofilaria ursi* was first described in a Japanese black bear (*Ursus Thibetanus Japonicus*) in 1941 (Yagmuti 1941). It has since been reported in a variety of bear species including Kamchatka brown bears (*Ursus arctos beringianu*), grizzly bears (*Ursus arctos horribilis*), and American black bears (*Ursus americanus*) (Rogers and Rogers 1976). *Dirofilaria ursi* is common in American black bear populations throughout North America and has been found in bears in Ontario, Quebec, Alberta, New Brunswick, British Columbia, New York, Alaska, Michigan, Minnesota, Wisconsin, Georgia, and Tennessee, with prevalence ranging from 1 to 100%. States and providences in the

northeastern side of the continent generally had high prevalences (94% in New York, 94.5% in Ontario, and 100% in Minnesota), while states and providences in the far west and southeast of the continent, generally had lower prevalences (31.8% in Alberta and British Columbia, and 17% in Georgia and Tennessee) (Choquette 1952; King et al. 1960; Rausch 1961; Jonkel and Cowan 1971; Rogers 1975; Frechette and Rau 1977; Manville 1978; Crum et al. 1978; Addison et al. 1978; Dies 1979; Duffy et al. 1994; Michalski et al. 2010; Catalano et al. 2015; Gwynn et al. 2017). Adults reside in subcutaneous tissue, as well as in the connective tissue surrounding organs, including the kidneys or the esophagus. Female *D. ursi* release first stage larvae (microfilaria) into the blood stream where they are taken up by a black fly (family Simuliidae) and transmitted to another suitable host (Addison 1980). Cases of dirofilariasis caused by *D. ursi* have been reported in humans (Beaver et al. 1987). No clinical signs have been reported in infected bears.

Another *Dirofilaria* species, *Dirofilaria immitis*, is also known to infect black bears. *Dirofilaria immitis*, or the canine heartworm, is a common parasite of wild and domestic canids. Microfilaria are ingested by mosquitoes while feeding on infected hosts and then, following maturation of *D. immitis* to L3 stage, the parasite is transmitted by infected mosquitoes to hosts. Adult parasites reside in the right ventricle and pulmonary arteries of the infected host. *Dirofilaria immitis* can cause significant diseases in canids and has also been reported to cause dirofilariasis in humans (Dantas-Torres and Otranto 2020). *Dirofilaria immitis* was first reported in a black bear in North Carolina in 1975; five adults were recovered from the right ventricle and pulmonary arteries of a hunter-killed black bear (Johnson 1975). *Dirofilaria immitis* has since been reported in other black bears in the southeastern United States at low prevalences (15% prevalence in Florida; 8% prevalence in bears surveyed from Virginia, West Virginia, North

Carolina, Tennessee, Georgia, and Florida) (Crum et al. 1978; Conti et al. 1983). Black bears were thought to be unsuitable hosts for *D. immitis* due to microfilaria being absent in adult female worms collected from the bear in North Carolina in 1975 (Johnson 1975). Microfilaria was similarly absent in adult female worms collected from bears in Florida (Conti et al. 1983). However, microfilaria have been detected in one worm collected from a bear in North Carolina in 1978 (Crum et al. 1978). Clinical signs have not been reported in infected black bears.

More recently, filarial nematodes genetically similar to *Mansonella ozzardi*, a human filarial nematode found in central and south America, have been reported in black bears. *Mansonella ozzardi*-like filarial nematodes were first reported in two American black bears in Minnesota in 2017. Both bears had microfilaria present on Wright–Giemsa-stained blood films. Sequencing of the filarial 5s RNA ITS region indicated that the samples from both bears were most similar to *Mansonella ozzardi* (98%) (Gwynn et al. 2017). *M. ozzardi*-like nematodes were then reported in Japanese black bears (*Ursus Thibetanus Japonicus*) in 2021. Sequencing of the 18S-ITS1 region indicated that 15 of 30 bears tested were infected with filarial nematodes most genetically similar to *M. ozzardi* (88-90%) (Masatani et al. 2021). The human filariid is transmitted by biting midges (*Culicoides* spp.) and black flies (Simuliidae) (Lima et al. 2016). *Manonsella* spp. adults reside in the connective tissue and peritoneal, pleural or pericardial cavities in humans and can cause mansonelliasis (Mediannikov and Ranque 2018). Symptoms of *M. ozzardi* induced mansonelliasis include fever, headache, articular pain, and ocular lesions, thought to be caused by migrating microfilaria (Lima et al. 2016). Little is known about the *M. ozzardi*-like parasite that infects bears, including its life cycle, anatomical location of the adult nematodes, how the parasites affect bears, and its zoonotic potential. Given that, further investigation into this novel filarial nematode is warranted.

b. *Baylisascaris transfuga*

Baylisascaris transfuga is an intestinal roundworm found in captive and free-ranging bears worldwide. The ascarid has been reported in all bear species except spectacled bears (*Tremarctos ornatus*) (Rogers and Rogers 1976). In North America *B. transfuga* is commonly found in wild black bear populations throughout the United States and Canada. It has been reported in bear populations in Alaska, Minnesota, Montana, Wyoming, Wisconsin, Virginia, West Virginia, Georgia, Tennessee, North Carolina, Florida, Quebec, Ontario, and Alberta. Prevalences range from 10 to 89% (Rausch 1961; Rogers 1975; Worley et al. 1976; Frechette and Rau 1977; Manville 1978; Crum et al. 1978; Dies 1979; Conti et al. 1983; Duffy et al. 1994; Jenness 1997; Foster et al. 2004; Catalano et al. 2015). Other species in the genus *Baylisascaris*, primarily *B. procyonis*, the raccoon roundworm, are known to cause visceral, neural, and ocular larval migrans (LM) in a variety of species, including humans (Wise et al. 2005). Disease caused by *Baylisascaris* spp. can be fatal. Larval migrans caused by *B. transfuga* has not been reported in humans, however, it has been reported in mice and Mongolian gerbils (*Meriones unguiculatus*) (Papini and Casarosa 1994; Papini et al. 1996; Sato et al. 2004). Additionally, fatal *Baylisascaris* larval migrans, caused by either *B. transfuga* or *B. procyonis*, was reported in Japanese Macaques (*Macaca fuscata fuscata*) housed near both American black bears and raccoons (Sato et al. 2005). This suggests that *B. transfuga* may have the potential to infect humans. Clinical signs have not been reported in black bears infected with *B. transfuga*; however, *B. transfuga* has been reported to cause disease in two other bears: First, in 1953 *B. transfuga* was reported to have caused the death of an unknown bear that had over one hundred nematodes in the intestine, stomach, oral cavity, and larynx (Rogers and Rogers 1976). Second, in 2012 a young European brown bear was diagnosed with granulomatous peritonitis upon

autopsy. The granulomatous peritonitis was determined to be caused by *B. transfuga* due to the presence of intralesional *Baylisascaris* ova and nematode fragments, as well as fifty-eight nematodes, morphologically identified as *Baylisascaris transfuga*, in the small intestine (Szczepaniak et al. 2012).

c. *Trichinella* spp.

Trichinella is a genus of nematodes that infect mammals, birds, and reptiles and is found worldwide (Crisóstomo-Jorquera and Landaeta-Aqueveque 2022). It is spread through the consumption of raw or undercooked meat containing the encysted larvae. In humans, *Trichinella* is the cause of trichinosis, a disease which can cause significant clinical signs. Previously, all cases of trichinosis were attributed to *Trichinella spiralis*; however, there are now 10 recognized genotypes in the genus *Trichinella* (Pozio 2007). *Trichinella spiralis*, *Trichinella nativa*, *Trichinella murreli*, and *Trichinella* T6 have all been reported in black bears (Pozio et al. 2001; Hill et al. 2005; Harms et al. 2021). As omnivorous scavengers, bears may have increased exposure to *Trichinella*. Additionally, cannibalism has been suggested as a cause of spread among bears (Rogers and Rogers 1976). Bears' role as game species makes the presence of this zoonotic pathogen of particular concern. While undercooked pork was previously the most common cause of trichinosis in humans, improved regulations in the pork industry have led to a substantial decrease in cases; now trichinosis is most commonly caused by the consumption of raw or undercooked game meat (Rostami et al. 2017; Diaz et al. 2020). There have been numerous cases of human trichinosis caused by the consumption of undercooked bear meat. In fact, bear meat has been implicated as the cause of 60% (12 of 20) of trichinosis outbreaks in the U.S. between 1997 and 2012 (Rostami et al. 2017). Thus, *Trichinella* has been monitored extensively in black bear populations across North America. It has been detected in a wide range

of states and providences, including Alaska, New York, Pennsylvania, Virginia, West Virginia, Montana, Wyoming, California, Idaho, Wisconsin, Quebec, Arizona, New Brunswick, New Hampshire, Oregon, and Ontario, at prevalences ranging from 0.01% to 13.2% (Rausch et al. 1956; King et al. 1960; Harbottle et al. 1971; Worley et al. 1976; Zimmerman 1977; Frechette and Rau 1977; Le Count 1981; Duffy et al. 1994; Mortenson et al. 2014). In the southeastern United States, *Trichinella* has only been reported in one bear in West Virginia (Harbottle et al. 1971). Other studies have failed to detect *Trichinella* in black bears in Virginia, Tennessee, Georgia, North Carolina, and Florida (Crum et al. 1978; Conti et al. 1983). No clinical disease has been reported in black bears infected with *Trichinella* spp.

d. Hookworms

Hookworms are nematodes that reside in the small intestines and feed on the host's blood. Hookworms can cause significant clinical signs in their hosts including diarrhea, anemia, and death. There are five species of hookworms known to infect black bears. *Unicinaria yukonensis* and *Unicinaria rauschi* have been found in bears in Alaska, Alberta, British Columbia, Montana, Wyoming, and Quebec, reaching prevalences as high as 72.5% (Olsen 1968; Worley et al. 1976; Frechette and Rau 1977; Catalano et al. 2015). *Arthrocephalus lotoris*, a hookworm of raccoons, as well as *Ancylostoma caninum* and *Ancylostoma tubaeforme*, hookworms of domestic dogs and cats, have been found in black bears in the southeastern United States at prevalences ranging from 2 to 64% (Crum et al. 1978; Foster et al. 2004). Additionally, unidentified hookworm larvae were detected on a fecal float from one bear in Wisconsin (Manville 1978). Hookworms can cause significant disease in their hosts; however, there have been no reported cases of clinical disease in an infected black bear. This may be due to the relatively low intensities of infections. Intensities were highest in bears infected with *U. rauschi*

in Alberta and British Columbia, reaching up to 740 worms in one bear. Thus, *U. rauschi* may have the highest disease potential in black bears.

e. Lungworms

Metastrongeloidea is a superfamily of nematodes that commonly reside in the lungs of their host. There are three metastrongylid lungworms known to infect black bears: *Crenosoma petrowi*, *Crenosoma potos*, and *Crenosoma vulpis*. *Crenosoma* spp. have been found throughout black bear populations in eastern North America, most often at low prevalences (4.9-9%) (King et al. 1960; Crum et al. 1978; Addison et al. 1978; Conti et al. 1983). Black bear populations in Ontario have the highest recorded prevalence of *Crenosoma* spp. at 32.8% (Addison et al. 1978). *Crenosoma* spp. have been associated with pneumonia in one bear in New York, as well as a cough condition of one black bear cub in New Brunswick (King et al. 1960; Mahjoub et al. 2020).

III. Misc. endoparasites

There are a variety of other endoparasitic helminths also known to infect black bears. These include *Gongylonema pulchrum*, *Macracanthorhynchus ingens*, *Physaloptera* spp., and capillarid spp. *Gongylonema pulchrum* is a spirurid nematode that infects a wide range of mammals, including humans, and causes gongylonemiasis. Susceptible hosts are infected by ingesting intermediate hosts (beetles or cockroaches) infected with the third stage larvae. Adults reside in areas of the mucous membrane of the upper digestive tract, including the tongue and esophagus (Xiaodan et al. 2018). *Gongylonema pulchrum* has been reported in black bears in Pennsylvania, Virginia, West Virginia, Tennessee, North Carolina, Florida, and Georgia, with prevalences ranging from 33.1% to 54% (Chandler 1950; Crum et al. 1978; Conti et al. 1983).

The first report of *G. pulchrum* in a black bear occurred in 1950 in Pennsylvania. The bear was reported to be severely emaciated and had numerous *G. pulchrum* present in its tongue. Whether the poor body condition was related to the parasitic infection is unclear (Chandler 1950). No clinical disease has otherwise been reported in infected bears.

Macracanthorhynchus ingens is an acanthocephalan, or thorny-headed worm, that infects a wide range of mammals, including humans. Adult worms reside in the intestines of their host. Black bears and raccoons (*Procyon lotor*) are considered the primary definitive hosts. *Macracanthorhynchus ingens* has been found throughout eastern North America, however, in black bears it has only been reported in the southeastern United States (Richardson et al. 2017). *Macracanthorhynchus ingens* has been reported in bears in Virginia, West Virginia, Tennessee, North Carolina, Florida, and Georgia at prevalences ranging from 21% to 80.4% (Crum et al. 1978; Conti et al. 1983; Foster et al. 2004).

Physaloptera is a genus of spirurid nematodes that infect a variety of wild and domestic mammals. Adult worms reside in the stomach of their hosts and can cause vomiting, gastritis, and ulcers. *Physaloptera* spp. have been reported in black bear populations in the southeastern United States at prevalences ranging from 8% to 19.6% (Crum et al. 1978; Conti et al. 1983). *Physaloptera* has also been reported in bears in Ontario, Canada at a much lower prevalence (1.2%) (Addison et al. 1978). Bears are thought to be accidental hosts for *Physaloptera* due to their low prevalence in black bear populations (Crum et al. 1978).

Capillarids are a large group of nematodes that infect a variety of vertebrates. Most capillarid species are associated with a specific epithelial surface of their host. There are three species of capillarids known to infect black bears: *Aonchthea putorri*, a stomach worm, *Eucoleus aerophilis*, a lungworm, and *Pearsonema plica*, a worm of the urinary bladder. In black

bear populations, capillarids have been reported exclusively in the southeastern United States (Crum et al. 1978; Foster et al. 2004).

Pelodera spp. are free-living rhabditid nematodes that are commonly found in decaying organic matter. *Pelodera* larvae have been known to occasionally invade mammalian skin and cause dermatitis. *Pelodera* dermatitis has been reported in sheep, dogs, horses, cattle, and humans. *Pelodera* dermatitis was first reported in a black bear in Michigan, also infected with *Sarcoptes scabiei*, in 2008. The bear was reported to have severe hair loss, lichenification, and crusted skin (Fitzgerald et al. 2008). *Pelodera* spp. have since been reported in black bears in Minnesota, Pennsylvania, and New York, all with associated alopecia and dermatitis (Dykstra et al. 2012; Peltier et al. 2018; Rojas-Sereno et al. 2022). *Pelodera* dermatitis has not been reported in black bears in the southeast.

Viruses

There are several viruses known to infect black bears. These include canine adenovirus (CAV), canine parvovirus (CPV), canine distemper virus (CDV), and rabies. All four are important viruses for wild and domestic canids and can cause significant disease. Rabies is also zoonotic and is considered a significant public health risk, as it is 100% fatal once symptoms occur.

I. Canine Adenovirus

There are two serotypes of CAV: type 1 and type 2. Type 1 (CAV-1) is responsible for infectious canine hepatitis (ICH), while type 2 (CAV-2) is the cause of infectious tracheobronchitis. Both serotypes have been reported in black bears. The first confirmed case of CAV-1 in black bears was in 1983 in Georgia, where the virus was isolated from the tissues of two three-month old cubs reported to have ataxia, convulsions, vomiting, nystagmus, paddling of

legs, and excessive salivation before dying (Pursell et al. 1983). CAV-1 was also reported in 28 black bears with similar symptoms in a wildlife park in western South Dakota in 1983 (Collins et al. 1984). CAV-1 infection was confirmed by virus isolation, indirect fluorescent antibody (FA) and virus neutralization (VN) tests, and restriction endonuclease analysis (Whetstone et al. 1988). Antibodies to CAV-1 have since been reported in black bear populations in Florida, Maryland, and Pennsylvania at low prevalences (6-8.5%) (Dunbar et al. 1998; Bronson et al. 2014; Niedringhaus et al. 2019c). Clinical signs were not reported in these bears. Additionally, CAV-2 has been reported in black bears in Alberta and British Columbia (8% prevalence) (Philippa et al. 2004). Clinical signs were similarly not reported in these bears.

II. Canine Parvovirus

CPV causes severe gastrointestinal disease in dogs and is very contagious. Antibodies to CPV have been detected in black bears in Florida, Maryland, Pennsylvania, New Mexico, and Wisconsin, with prevalences ranging from 0.6-16% (Dunbar et al. 1998; Bronson et al. 2014; Stephenson et al. 2015; Bard and Cain 2019; Niedringhaus et al. 2019c; Sikes et al. 2022). No clinical signs have been reported in seropositive bears. Additionally, a novel parvovirus *Ursus americanus parvovirus* (UaPV), in the genus *Chaphamaparvovirus*, was detected in the liver and kidney of one bear in Nevada (Alex et al. 2020). The clinical significance of this parvovirus in bears is not fully understood.

III. Canine Distemper Virus

CDV is another highly contagious disease of canines. CDV can affect a variety of organs including the heart, gastrointestinal tract, respiratory system, and nervous system and can cause severe clinical signs. Antibodies to CDV have been detected in black bears in Tennessee,

Florida, Alberta, British Columbia, Maryland, California, Pennsylvania, and Wisconsin at prevalences ranging from 3 to 56.86% (Cook 1982; Dunbar et al. 1998; Philippa et al. 2004; Bronson et al. 2014; Stephenson et al. 2015; Niedringhaus et al. 2019c; Sikes et al. 2022). Clinical disease caused by CDV was reported in one black bear cub in Pennsylvania in 2011. The cub was reported to have neurological signs, including tremors and seizures, had hyperkeratotic footpads, and had lost its fear of humans. Histologic examination revealed encephalitis. CDV was diagnosed using immunohistochemistry (IHC) on the brain and footpad tissue, as well as reverse transcriptase PCR (RT-PCR) on brain tissue (Cottrell et al. 2013). No other cases of clinical CDV infections in black bears have been reported.

IV. Rabies

All mammals, including humans, are susceptible to the rabies virus, which causes severe neurological disease and is a significant public health concern. Bears are considered less susceptible to rabies than canines. Black and brown bears experimentally infected with the rabies virus required much larger amounts of the virus to induce clinical disease than their canine counterparts did. Additionally, experimentally infected bears rarely shed the virus in their saliva (Rausch 1975). Cases of rabies in black bears have been reported sporadically throughout North America since the 1940s. States and providences where cases have been reported include Alberta, Ontario, Quebec, Arizona, Montana, New York, Pennsylvania, Maryland, Virginia, Massachusetts, Iowa, and North Carolina (Tabel et al. 1974; Eng et al. 1989; Mansfield 1992; “Public Health Dispatch: Multiple Human Exposures to a Rabid Bear Cub at a Petting Zoo and Barnwarming -- Iowa, August 1999” 1999; “Update: Raccoon Rabies Epizootic -- United States and Canada, 1999” 2000; Blanton et al. 2006, 2008; Dyer et al. 2013; Ma et al. 2020). Multiple cases in Canada were reported to have been associated with aggressive behavior towards humans

(Tabel et al. 1974; Walroth et al. 1996). One black bear cub in Iowa was reported to have neurological signs several hours before dying of rabies infection (“Public Health Dispatch: Multiple Human Exposures to a Rabid Bear Cub at a Petting Zoo and Barnwarming -- Iowa, August 1999” 1999).

Toxins

As opportunistic scavengers, black bears may accidentally ingest a variety of toxins. Repeated exposure to toxins through the ingestion of contaminated plants, animals, or garbage, may lead to bioaccumulation in black bears, making them an excellent sentinel for environmental toxins. One toxin that bears are often exposed to is lead (Pb). Large game hunters frequently utilize Pb ammunition which releases metal fragments into the tissues of their harvested animals. Thus, bears may be exposed through the ingestion of discarded remains of hunter harvests. Black bears in Quebec were reported to frequently scavenge moose viscera left by hunters, and Pb exposure increased in female bears as harvest density increased, as well as with age (Legagneux et al. 2014; Brown et al. 2022). In Yellowstone Pb was detected in the blood of black bears at levels higher than what is considered safe for humans, but did not increase during the hunting season (Rogers et al. 2012). Despite the high levels of Pb exposure, clinical signs of toxicosis have not yet been reported in bears.

Bears may be exposed to pesticides through direct ingestion, ingestion of contaminated food or vegetation, or ingestion of contaminated prey. Pesticides (DDE, DDT, DDD, and Dieldrin) and mercury were detected at low levels in fat and muscle samples from eight black bears in Idaho in 1972 (Benson et al. 1974). Additionally, a variety of anticoagulant rodenticides, including brodifacoum, bromadiolone, difethialone, difenacoum, chlorophacinone, and

diphacinone, have been detected in the livers of bears surveyed in California (McMillin et al. 2018; Clothier et al. 2022). Bromethalin, a neurotoxic rodenticide, was also detected in the brain and adipose tissue of three bears in California in 2021 (Rudd et al. 2021). No clinical signs were reported in these bears, however toxicosis due to pesticides have been reported in black bears. Anticoagulant rodenticide toxicity was reported in two black bears in same neighborhood in Georgia three years apart. The first case was a sow that emerged from her den in March 2019 and collapsed dead (Nasim et al. 2020). The second case was a yearling found lethargic and recumbent for several days before dying. High levels of bromadiolone and brodifacoum were detected in both bears' livers (Teo et al. 2023). Additionally, neurotoxic rodenticide toxicity was reported in one yearling black bear in Ontario in 2021. The bear was reported to have ataxia and unilateral epistaxis. Desmethylobromethalin detected in the adipose tissue by LC-MS/MS confirmed bromethalin toxicity (Cox et al. 2021). Furthermore, toxicosis due to other toxins have also been reported in black bears. Four bears (a sow and three yearlings) found dead in Pennsylvania in 2016 were diagnosed with Yew toxicosis. All four bears had large amounts of English yew (*Taxus baccata*), an evergreen shrub, in their stomachs. Yew contains alkaloid toxins called taxines which attack the cardiovascular system and can cause cardiac arrest and acute death (Mee et al. 2020). Bears in Michigan and New Hampshire were diagnosed with theobromine toxicosis caused by chocolate consumption in 2010 and 2014, respectively. All five bears were found dead near bait piles, and the four bears in New Hampshire all died of cardiovascular collapse (Beringer et al. 2016). Ethylene glycol toxicity was identified as the cause of death in one cub in Tennessee, due the presence of renal tubular necrosis and intratubular calcium oxalate crystals (Sheldon et al. 2022).

Conclusions and Objectives

There have been a multitude of parasites, pathogens, and toxins detected in bears. This is unsurprising as omnivorous, opportunistic scavengers, bears may have increased exposure to these pathogens and toxins, making them an excellent sentinel for the health of their environment. Despite this, there have been limited studies on black bear health in the southeast. The objective of this study is to monitor the black bear population in East Tennessee and the surrounding areas for the presence of certain parasites and pathogens using a variety of diagnostic techniques including PCR, serology, skin scrapes, and fecal floats. The results of this study will help wildlife managers make more informed management decisions regarding bear, human, and domestic animal health. It will also create a baseline for future health monitoring and research in this area.

CHAPTER THREE

MATERIALS AND METHODS

For this study, blood, serum, fecal, skin scrape, and ectoparasite samples were collected from bears between March and December 2022. Bears sampled for this study include black bears undergoing relocation in the Great Smoky Mountains National Park (GSMNP), cubs and yearlings undergoing rehabilitation at Appalachian Bear Rescue (ABR), euthanized ‘conflict’ bears, roadkill bears, and hunter-harvested bears. Additionally, blood, serum, and fecal samples collected from GSMNP, ABR, and euthanized ‘conflict’ bears in 2021 were also included in this study.

To determine the presence of several blood-borne pathogens, DNA was extracted from whole blood using Qiagen DNeasy Blood and Tissue Kits following the manufacturer’s instructions. Nested PCR was performed using RLB 3.1 and 5.1 primers, which test for the presence of apicomplexan parasites, including *Babesia* spp. and *Hepatozoon* spp. Thermocycling was completed following the protocol described in Yabsley et al., 2006. To test for *Trypanosoma* spp. D75 and D76 primers were used and thermocycling was completed as described by Madeira et al., 2009. And to test for *Rickettsia rickettsia*, Rr190.602n and Rr190.70p primers were used and the thermocycling protocol outlined in Saracho Bottero et al., (2015) was followed. A negative DNA extraction, and positive and negative PCR controls were used for all PCR tests. PCR products were loaded onto a 1.5% agarose gel and gel electrophoresis was performed. PCR products with bands at the correct size were cleaned with Exosap (Thermo Fisher Scientific) and sent for Sanger sequencing at UT’s Division of Biological sequencing. The sequences were then entered into a BLAST search in GenBank to determine the species.

A modified agglutination test (MAT) was used to test serum for the presence of *T. gondii* antibodies. Serum samples were serially diluted from 1:25 to 1:3200 to determine concentration cutoff. Samples that were $\geq 1:25$ concentration were considered seropositive. Additionally, serum from 48 black bears were sent to Cornell for CAV, CDV, and CPV serology.

Fecal floats were performed using Sheather's sugar solution and centrifugation as described in Chapman et al., 2021. Slides were analyzed under a microscope and parasite eggs and larvae were morphologically identified.

Skin scrapes were taken from bears sampled in 2022. If the bears had any areas of alopecia the skin scrape was taken there, otherwise a random spot on each bear was chosen. A size 10 scalpel blade was then used to scrape the skin until a little bit of blood appeared. The scraped skin was then placed on microscope slide with a drop of mineral oil, covered with a cover slip, and analyzed under a microscope for the presence of mites, fleas, or lice. Other ectoparasites, such as ticks, that were opportunistically found on the bears were collected and morphologically identified.

To assess for the presence of microfilaria, blood samples were collected in EDTA tubes from bears sampled in 2022. Centrifugation, 2% formalin, and methylene blue were used to perform Knott's tests as described by Chapman et al. 2021. Slides were analyzed under a microscope and microfilaria were measured and counted. DNA was extracted from several positive samples and PCR was performed using filarial ITS F and R primers. The DNA extraction and PCR protocols were performed as described earlier.

Thirty-five bears were brought in for autopsy. Fifteen of these bears were euthanized nuisance bears, 7 were roadkill, 7 were euthanized for health reasons, and 2 were found dead.

Four of the bears autopsied did not have their cause of death recorded. Additionally, the viscera of two hunter harvested black bears were also analyzed. Bears were examined internally and externally for any abnormalities, lesions, or parasites. The heart and GI tract were cut open to look for the presence of parasites. Any parasites found were preserved in 70% EtOH and morphologically identified. Additionally, samples of lung, diaphragm, skeletal muscle, lymph node, brain, heart, liver, spleen, kidney, and skin (as well as tongue, urinary bladder, pancreas, thymus, eye, ovary, testicle, bone marrow, and sciatic nerve in some bears) were fixed in 10% neutral-buffered formalin, routinely processed for histology, embedded in paraffin, sectioned at 4 μm , and stained with hematoxylin and eosin (HE). Histology slides were analyzed under the microscope for lesions or parasites.

The association of age and sex with positive test results for each item were examined using a Chi square test. Statistical significance was identified at the level of 0.05. Analyses were conducted in SAS 9.4 TS1M8 for Windows 64x (SAS institute Inc., Cary, NC).

CHAPTER FOUR

RESULTS

Most bears sampled originated from east Tennessee, including the Great Smoky Mountains National Park (GSMNP) and surrounding areas. However, ABR rescues bears from across the southeast and several ABR cubs originated from other states: six cubs were from Kentucky, two from Alabama, and two from South Carolina. Additionally, some of the bears sampled from the GSMNP originated from the North Carolina side of the park. Therefore, the bears sampled in this study represent the black bear population from east Tennessee and the surrounding states.

Of the 169 bears sampled, 77 were females and 92 were males. Repeat samples were taken from a total of 31 bears. Twenty-two of these were ABR bears, which were sampled during both intake and release exams. The additional 9 were GSMNP bears that were either recaptured or brought in for autopsy following their initial capture. Seventy-three were juveniles (neonates, cubs, or yearlings), 93 were adults, and age was not determined for 3 bears. The three bears with undetermined ages were excluded from age analyses. ABR yearlings that were 2 years old by the time of release were considered 'juveniles' for our analysis.

Skin scrapes were performed on a total of 94 bears. *Sarcoptes scabiei* was not found on any. However, *Ursicoptes americanus* was found on 6 of the bears; none had any significant alopecia. There were no significant differences between sexes ($P = 0.9141$, $\chi^2 = 0.01$) or age categories ($P = 0.4554$, $\chi^2 = 0.56$). One-hundred and seven bears were examined for ticks. Ticks were discovered on 71 (66.4%) of them. Sixty-nine were infested with *Dermacentor variabilis* (64.5%), 5 were infested with *Amblyomma maculatum* (4.7%), 1 was infested with *Amblyomma*

americanum (0.9%), and 1 was infested with *Ixodes scapularis* (0.9%) (Table 1) (All tables and figures are located in the appendix). Adults had higher prevalences of ticks (72.7%) as well as specifically *Dermacentor variabilis* (71.2%) than juveniles did (53.9%, and 51.3%, respectively). The differences were marginally statistically significant ($P = 0.0489$, $\chi^2 = 3.88$; $P = 0.04$, $\chi^2 = 4.22$).

DNA was extracted from the blood of 113 bears and *Trypanosoma* spp., *Rickettsia rickettsii*, and apicomplexan parasite PCR was performed. None were positive for *Trypanosoma* spp. or *Rickettsia rickettsii*. Ten (8.9%) were positive for the presence of apicomplexan parasites (Table 2). Sequencing revealed all 10 were infected with *Babesia* spp. There was no statistically significant difference ($P = 0.2935$, $\chi^2 = 1.10$) between sexes, however there was a statistically significant difference ($P = 0.007$, $\chi^2 = 7.10$) between age categories; 3% of adults were positive, while 17.8% of juveniles were positive. Genetic analysis of the *Babesia* spp. indicated that samples from three juveniles were 99.75-100% similar to *Babesia coco* isolates identified in raccoons in Texas (Accession number: MN013190.1). Samples from three more bears were 100% aligned with *Babesia microti*-like isolates also from raccoons in Texas (Accession number: MN011935.1). One sample was 100% similar to a *Babesia* spp. isolate also from a raccoon from Texas (Accession number: MN013191.1) (Modarelli et al. 2020). The remaining three positives were 99.76-100% similar to *Babesia* spp. isolates also isolated from raccoons in the United States (Accession number: MK580473) (Garrett et al. 2019).

Serum from 157 bears were tested for the presence of *Toxoplasma gondii* antibodies using a modified agglutination test (MAT). Seventy-eight bears were positive, giving a seroprevalence of 49.7% (Table 3). There was no statistically significant difference between the seroprevalence in males and females ($P = 0.2316$, $\chi^2 = 1.43$). There was a statistically significant

difference between the seroprevalence in adults and juveniles ($P = <0.0001$, $\chi^2 = 17.90$). Adults had a seroprevalence of 64.7% and juveniles had a seroprevalence of 30.4%. Two adult bears tested positive for *T. gondii* antibodies on their initial capture but tested negative 2 months later. This change in seropositivity may indicate that titers in bears decrease quickly without reinfection. Both bears were considered positive for this analysis.

Serum samples from 49 black bears were sent for CAV, CDV, and CPV serology. Samples from two separate bears were unable to be tested for CAV and CDV, and CAV and CPV, respectively, due to insufficient quantity and toxicity. Antibodies to CAV were detected in one juvenile male, antibodies to CPV were detected in one juvenile female, and antibodies to CDV were detected in nine bears, giving seroprevalences of 2.1%, 2.1%, and 18.8%, respectively (Table 3). There was no statistically significant difference between CDV seroprevalence in males vs females or in adults vs juveniles ($P = 0.5161$, $\chi^2 = 0.42$; $P = 0.3291$, $\chi^2 = 0.95$).

Fecal floats were performed for a total of 122 bears. Parasite eggs or larvae were discovered on 48 (39.3%) of these. Thirty-two had *Baylisascaris transfuga* eggs and an additional three, that were negative on fecal flotation, had *B. transfuga* adults on autopsy. These three were considered *B. transfuga* positive for our analysis. Thus, thirty-five bears (28.7%) were positive for *B. transfuga*. Sixteen bears (13.1%) had capillarid spp. eggs, 11 (9%) had nematode larvae, 2 (1.6%) had *Ancylostoma* spp. eggs, 2 (1.6%) had unidentified coccidia, 1 (0.8%) had *Eimeria* spp., and 1 (0.8%) had *Cryptosporidium* spp. (Table 4). A higher percentage of juveniles were positive for *B. transfuga* (51.2%) than adults (16.9%) and the difference was statistically significant ($P = <0.0001$, $\chi^2 = 15.69$). Similarly, more juveniles (25.6%) also tested positive for capillarid spp. than adults (6.5%) did, which was also statistically significant ($P = 0.0032$,

$\chi^2=8.70$). There was no statistically significant difference between the sexes for *B. transfuga*, capillarid spp., nematode larva, or coccidia ($P= 0.5874$, $\chi^2= 0.29$; $P= 0.1747$, $\chi^2= 1.84$; $P= 0.1753$, $\chi^2= 1.84$; $P= 0.2491$, $\chi^2= 1.33$). There was also no statistically significant difference between age categories for nematode larvae and coccidia ($P= 0.4850$, $\chi^2= 0.49$; $P= 0.6458$, $\chi^2= 0.21$).

Thirty-five bears were autopsied and the viscera of two additional hunter-harvested black bears were analyzed. Stomachs and intestines were analyzed for the presence of parasites in all 37 of these bears. Adult nematodes morphologically consistent with *Baylisascaris transfuga* were found in the intestines of 9 of these bears, nematodes morphologically consistent with *Macracanthorhynchus ingens* were found in 2, and 1 *Physaloptera* spp. adult was found in the stomach of 1 adult female black bear. There was no statistically significant difference between sexes or age categories for *B. transfuga* ($P= 0.0771$, $\chi^2= 3.12$; $P= 0.832$, $\chi^2= 0.05$).

Additionally, the hearts of all of these bears were also analyzed for the presence of parasites. The heart from an additional hunter-harvested black bear was also analyzed for parasites. Two adult *Dirofilaria immitis* were found in the right atrium, right ventricle, and pulmonary artery of one yearling male black bear. Tongues were analyzed grossly and histologically for the presence of parasites on 22 bears. Nematodes morphologically consistent with *Gongylonema* spp. were found in 3 bears (Figure 1). Additionally, a cyst consistent with *Sarcocystis* spp. was found in the tongue of one adult male bear (Figure 2)(Mehlhorn and Frenkel 1980). Diaphragms from 20 bears were analyzed using squash prep and histology for the presence of *Trichinella* spp. *Trichinella* spp. was found in the diaphragm of one adult female black bear. *Trichinella* spp. was also found encysted in the skeletal muscle and tongue of the same bear (Figure 3). The lungs of 33 of the bears were analyzed histologically for the presence of parasites or lesions. Severe

eosinophilic and granulomatous pneumonia with intralesional eggs and larvae was noted in one adult female black bear (Figure 4). PCR using NC1 and NC2 primers, and sequencing revealed the eggs and larvae were 95% similar to *Angiostrongylus vasorum*, or the French heartworm, isolates from Europe, including GU45375.1. No significant lesions or parasites were noted in the lungs of the other bears. Skin samples from 27 bears were analyzed histologically for presence of microscopic lesions or parasites. One *Pelodera* spp. nematode was found in the skin of one male yearling with mild alopecia on the flanks (Figure 5). Mild hyperkeratosis and follicular atrophy were noted, but there was no associated inflammation. Autopsy and histology results are summarized in Table 5.

Knott's tests were performed on 91 bears. Microfilaria was found on a total of 63 (69%) of these bears (Table 5). Microfilaria sizes ranged from 175 to 300µm long and 2.5 to 6.25µm wide, with most being between 225 and 275µm long and 2.5 to 5µm wide. PCR using filarial ITS primers was performed a select number of positive samples. PCR samples sent to sequencing came back as 85.57% similar to *Mansonella ozzardi*. Adults (86.2%) had a significantly higher prevalence of microfilaria than juveniles (38.7%) ($P = <0.0001$, $\chi^2 = 21.57$). There was no significant difference between the sexes ($P = 0.9164$, $\chi^2 = 0.01$).

Additionally, samples of liver and brain from one yearling male black bear, and one adult female black bear were sent to University of California, California Animal Health and Food Safety System, Davis, California for toxicology tests. The yearling was euthanized due to neurological impairment, while the adult female had been found dead with no obvious cause of death apparent. Trace amounts of desmethylbromethalin, the toxic metabolite of bromethalin, a neurotoxic rodenticide, was detected in the brain of the yearling with a vacuolar leukoencephalopathy, and flunixin, a non-steroid anti-inflammatory drug (NSAID) used

commonly in large animal medicine, was detected by liquid chromatography- mass spectrophotometry (LC/MS) in the liver of the adult bear.

Forty bears died over the course of this study. Four bears were hunter-harvested, and the histories on an additional four were not recorded. The most common cause of death was euthanasia due to human-bear conflict. Sixteen bears were euthanized due to ‘conflict’ activity, including breaking into cabins and cars, entering a public restroom, and approaching humans. Seven of the euthanized ‘conflict’ bears had human food and garbage in their stomachs, indicating habituation. The second most common cause of death was vehicular collision. Seven bears were found dead after being hit by a car, and an additional two were euthanized due to traumatic injuries caused by vehicular collisions. Four more bears were also euthanized due to health concerns, including traumatic injuries due to falling from a tree, a possible pneumothorax, bone disease that inhibited climbing, and seizures due to Bromethalin toxicity. One cub died shortly after it had been rescued by ABR. The cub was emaciated and had microphthalmia with ocular dysgenesis and selenium deficiency. Two additional bears were found dead with no obvious cause of death apparent grossly. The first was too autolyzed to determine the cause of death. The second was a 12yr old female black bear that had significant eosinophilic granulomatous pneumonia with intralesional eggs and larvae. PCR and sequencing confirmed *Angiostrongylus vasorum*, or French heartworm, infection.

CHAPTER FIVE

DISCUSSION

Health monitoring revealed that the southeastern black bear population harbors many diseases and parasites, including two parasites, *M. ozzardi*-like nematodes and *A. vasorum*, not previously reported in the southeast. Other notable results include the first putative case of *Pelodera* spp. in a black bear in the southeast, the second report of *Trichinella* spp. in a black bear in the southeast, and the second report of fatal Bromethalin toxicity in a black bear. These findings demonstrate how health monitoring can help identify new potential threats to black bear, and other potential hosts, including human, health. Other findings in this study will be used to create a baseline that will allow future researchers to identify any changes in health or pathogen prevalences in this population. These findings highlight the importance of health monitoring as a tool for wildlife management, and the need for further research and monitoring.

Notably, no *Sarcoptes scabiei* were detected on any bears in this study. *Ursicoptes americanus*, another mite also known to cause mange in black bears, was found on six bears. Previous reports of *Ursicoptes americanus* in black bears have been associated with alopecia, lichenification, and exfoliation. Other cases report no clinical signs in infested bears (Yunker et al. 1980). None of the positive bears in this study had any significant alopecia, though two had mild facial alopecia; one around the eyes, and another on the forehead. The reason for some bears having severe clinical signs while others do not is unclear. In this study only one to three mites were found on each bear, thus lack of clinical mange in these cases may be attributed to the relatively low intensity of the infestations.

Additionally, several tick species were recovered from bears in this study including, *Dermacentor variabilis*, *Ixodes scapularis*, *Amblyomma americanum*, and *Amblyomma maculatum*. *Dermacentor variabilis*, the American dog tick, was by far the most common tick identified on black bears. *Dermacentor variabilis* was identified on 64.5% of all bears examined. Additionally, *D. variabilis* made up 96.8% (691/714) of all ticks collected. This high prevalence of *D. variabilis* in black bears is consistent with previous reports in the southeast. The low prevalences of *Ixodes scapularis* (0.9%) and *Amblyomma americanum* (0.9%), however, were not consistent with previous reports in the southeast, which typically report high (30-94%) prevalences (Yabsley et al. 2009; Nims and Durden 2011; Hertz et al. 2017). The reasons for these low prevalences are unclear. The range of *Amblyomma maculatum*, the gulf coast tick, previously did not extend into eastern Tennessee. The presence of *A. maculatum* in the east Tennessee black bear population indicates a northward expansion of their range, likely due to global climate change. *Amblyomma maculatum* is known to harbor the apicomplexan parasite *Hepatozoon* spp. and the bacterium *Rickettsia parkeri*, both known to cause disease in wild and domestic mammals. *Rickettsia parkeri* can also cause *Rickettsia parkeri* rickettsiosis in humans. Thus, the expansion of the range of *A. maculatum* may put more wild and domestic animals, and humans at risk of disease. The prevalence of *A. maculatum* in the east Tennessee black bear population is still lower than what has been reported in states within their traditional range, including Florida and Georgia which reported prevalences ranging from 13 to 50% (Yabsley et al. 2009; Nims and Durden 2011; Hertz et al. 2017). Adults were marginally more statistically likely to be infested with ticks (and specifically *D. variabilis*) than juveniles were. The reason for this difference may be due to increased exposure as adults will have had more time to encounter ticks than juveniles.

Despite the high prevalence of *D. variabilis*, the vector for *Rickettsia rickettsii*, the bacterial agent that causes Rocky Mountain Spotted Fever (RMSF), none of the bears were PCR positive for *R. rickettsii*. The bacteria has yet to be reported in a black bear, however, antibodies to *R. rickettsii* have been reported in bears in Idaho and Maryland (Binninger et al. 1980; Bronson et al. 2014). Additionally, other *Rickettsia* spp., including *Rickettsia parkeri* the causative agent of *R. parkeri* rickettsiosis, have been detected in a wide range of tick species parasitizing black bears throughout the eastern United States (Yabsley et al. 2009; Leydet and Liang 2013; Chern et al. 2016; Lockwood et al. 2018). Ticks in this study were not tested for the presence of tick-borne pathogens, therefore it is unclear how prevalent the bacteria is in ticks parasitizing black bears.

While no *R. rickettsii* was detected in any of the bears, another tick-borne pathogen, *Babesia* spp., was found in 8.9% of bears tested. The 8.9% prevalence of *Babesia* spp. in southeastern bears is consistent with what has previously been reported in New Jersey (9.2% and 7.1%), Oklahoma (6%), and North Carolina (16.7%) (Zolnik et al. 2015; Chern et al. 2016; Skinner et al. 2017; Westmoreland et al. 2019). Though, these are all much lower than the 41.8% prevalence initially reported in American black bears in New Jersey in 2015 (Shaw et al. 2015). Juveniles in this study had a significantly higher prevalence of *Babesia* spp. than adults did, while in a previous study adults and yearlings had a significantly higher prevalence than cubs (Shaw et al. 2015). Shaw et al. (2015) suggested that as bears get older, they have more chances to be exposed to ticks harboring the parasite. The reason for juveniles having a higher prevalence in this study is unclear. The relatively small sample of positive bears in this study may have skewed our results. Previous studies have reported *Babesia* spp. in black bears to be genetically similar to *Babesia* spp. found in raccoons and a maned wolf (Skinner et al. 2017; Westmoreland

et al. 2019). Similarly, in this study all *Babesia* sequences from bears were >99% similar to sequences previously identified in raccoons from North America (Garrett et al. 2019; Modarelli et al. 2020). It is unclear whether *Babesia* spp. poses any health risks to black bears. No clinical disease was observed in any of the positive bears in this study, nor has it been reported in previous studies. However, clinical babesiosis has been reported in a captive Malayan Sun bear (Zubaidah et al. 2020). Furthermore, while many wildlife species act as reservoirs for *Babesia*, the role of bears as a reservoir of this parasite, including potentially zoonotic *Babesia* species, also remains unclear and warrants further research.

Another vector-borne parasite, *Trypanosoma* spp., was not detected in any bears in this study. *Trypanosoma* species have not been identified in American black bears, however they have been found in a captive polar bear and several captive Asiatic black bears, all with clinical signs of disease (Jaime-Andrade G. et al. 1997; Shahid et al. 2013). Additionally, *Trypanosoma* spp. has been detected in a wide range of wild mammals including raccoon, coyotes, skunks, and bobcats, in the southeast. How *Trypanosoma* may affect black bears remains unknown, as well as the role, if any, bears play as a potential reservoir of this parasite.

Serological tests revealed that around half of the 157 bears tested in this area have antibodies to *Toxoplasma gondii*. This seroprevalence is unsurprising as black bears are known to have one of the highest seroprevalences of *T. gondii* of all animals. In fact, this seroprevalence is on the lower end of what is typically reported in black bear populations in North America (Dubey et al. 2021). Specifically, it is lower than the 74% seroprevalence previously reported in east Tennessee (Ammar et al. 2020). This relatively low seroprevalence may, in part, be due to the relatively large percentage of juvenile bears sampled (44.8% of bears tested were juveniles, compared to 36% in the previous study). Adults in our study were significantly ($P = <0.0001$)

more likely to test positive than juveniles, which is consistent with previous studies (Dubey et al. 2021). Adults in this study had a seroprevalence of 64.7%, which aligns more with previous reported seroprevalences in this area. While juveniles had a seroprevalence of 30.2%. This increase in seroprevalence with age is likely due to increased exposure, as older bears will have had more opportunities to consume *T. gondii* oocysts or bradyzoites through scavenging. Additionally, three of the juveniles that tested positive were neonates, two of which were siblings. These positives may have been due to maternal antibodies rather than an active infection. It is notable that *T. gondii* is zoonotic and thus, its presence in this population may pose a health risk to those who consume hunter-harvested bear meat.

Furthermore, serology tests also indicated bears in this area have been exposed to three viruses that can cause significant disease in wild and domestic canids, though at prevalences much lower than what has been reported for wild canids in the area (Gates et al. 2014). CAV-1 antibodies were detected in one black bear of 47 tested, giving a seroprevalence of 2.1%. This is consistent with previous reports from Florida, Maryland, and Pennsylvania, which reported similar low seroprevalences (6-8.5%) (Dunbar et al. 1998; Bronson et al. 2014; Niedringhaus et al. 2019c). CAV-1 has also been isolated from bears with clinical signs, including ataxia, convulsions, vomiting, and death, in Georgia and South Dakota (Pursell et al. 1983; Collins et al. 1984). No clinical signs were seen in the seropositive bear in this study. Additionally, no lesions consistent with CAV-1 infection were found either of the two bears with serology that were also subject to postmortem exam. However, these results do confirm the presence of this virus in the Tennessee black bear population. Given the disease potential of this virus in black bears, further monitoring is warranted.

CPV antibodies were similarly detected in only one bear of 48 tested, giving a seroprevalence of 2.1%. Previous studies in New Mexico, Wisconsin, and California have reported similar low prevalences (0.6- 6%) (Stephenson et al. 2015; Bard and Cain 2019; Sikes et al. 2022). However, other studies, including one in Florida, have reported slightly higher prevalences ranging from 12.2 to 16% (Dunbar et al. 1998; Bronson et al. 2014; Niedringhaus et al. 2019c). No clinical signs have been reported in seropositive bears. Similarly, no clinical signs were seen in the seropositive bear in this study. Additionally, no lesions consistent with CPV infection were seen in either of the two bears with serology that were also subject to postmortem exam.

CDV antibodies were detected in 9 of 48 bears tested, giving a seroprevalence of 18.8%. Seroprevalences of CDV in black bear populations range greatly from 3 to 56% (Cook 1982; Dunbar et al. 1998; Philippa et al. 2004; Bronson et al. 2014; Stephenson et al. 2015; Niedringhaus et al. 2019c; Sikes et al. 2022). Our reported seroprevalence is slightly lower than a previous report from Tennessee, which reported a seroprevalence of 29.2% (Cook 1982). However, it is higher than what has been reported in Florida (8%) (Dunbar et al. 1998). There has been one case of clinical distemper in a black bear (Cottrell et al. 2013). No clinical signs were seen in any of the positive bears in this study. Additionally, no lesions consistent with CDV infection were seen in either of the two bears with serology that were also subject to postmortem exam.

Interestingly, both the CAV-1 and CPV positive bears were juveniles, and six of the nine CDV positive bears were juveniles, two of which were siblings. Three of the CDV positive bears, including the two siblings, were neonates. Thus, these positives may have been a result of maternal antibodies. Three of the CDV positive cubs, including the two siblings, were also

positive for *T. gondii*. The CAV-1 positive cub was negative on its initial test but tested positive three months after intake, suggesting the cub became infected while housed at ABR.

Surprisingly, none of the three other cubs housed with this bear tested positive for CAV-1.

Fecal flotations revealed a number of parasites. The most prevalent parasite found on fecal floatation was *Baylisascaris transfuga*. This is unsurprising as *B. transfuga* is a common parasite of bears, found in black bear populations across North America. Prevalences of *B. transfuga* in black bear populations range greatly from 8 to 89% (Rausch 1961; Rogers 1975; Worley et al. 1976; Frechette and Rau 1977; Manville 1978; Crum et al. 1978; Dies 1979; Conti et al. 1983; Duffy et al. 1994; Jenness 1997; Foster et al. 2004; Catalano et al. 2015). The 28.69% prevalence seen in this population is consistent with a previous southeastern study which found a 23% prevalence of *B. transfuga* in black bears in Florida (Foster et al. 2004). Though these are both lower than other reports from the southeast which have reported 50 and 53% prevalences (Crum et al. 1978; Jenness 1997). Additionally, *Baylisascaris transfuga* adults were found in the intestines of 3 bears on autopsy that tested negative on fecal floatation. The lack of *B. transfuga* eggs on fecal flotation may be attributed to the nematodes not shedding eggs at the time the fecal sample was collected. Thus, the estimated prevalence in this study may be lower than the actual prevalence in the population. Juveniles in this study were significantly more likely to be positive for *B. transfuga* than adults. Notably though, 9 of the 19 juveniles that tested positive were housed together at ABR and may have become infected through the accidental ingestion of eggs from the other bears' feces. Thus, prevalences of *B. transfuga* in juveniles may have been artificially elevated. Furthermore, there was one adult female bear that had *Baylisascaris* spp. eggs on fecal flotation, but no adults were found on autopsy. It is unknown why adult nematodes were not observed on autopsy. It is possible they were simply missed

during the autopsy process. Another possibility is that the bear obtained the eggs through the ingestion of fecal matter from another host, such as a raccoon. Other scavengers in this area have been shown to shed *Baylisascaris* spp. eggs despite not harboring the nematode (Gerhold et al. 2018).

The next most prevalent parasite found on fecal floatation was capillarid spp. Thirteen percent of bears tested positive for capillarid spp. Capillarid spp. have been reported exclusively in bears in the southeastern United States and prevalences range greatly from 5 to 73.2% (Crum et al. 1978; Conti et al. 1983). Juveniles were significantly more likely to test positive than adults. Similar to *B. transfuga* this may, in part, be attributed to 11 of the positive cubs being housed together at ABR.

Nematode larvae were found on the fecal floats of 11 bears. Nematode larvae may indicate lungworm infections in bears. There are 3 lungworm species that are known to infect black bears: *Crenosoma petrowi*, *Crenosoma potos*, and *Crenosoma vulpis*. *Crenosoma* spp. have been associated with pneumonia and cough conditions in black bears (King et al. 1960; Mahjoub et al. 2020). Though no clinical signs were observed in the positive bears, this finding may indicate the presence of potentially pathogenic parasites in this black bear population. However, these nematode larvae may also be parasites of the plants or animals consumed by the black bears and thus are simply incidental findings. Further investigation is needed to understand the significance of this finding.

Ancylostoma spp. eggs were found on fecal flotations of two bears. *Ancylostoma* spp. have been detected in bears in the southeast at prevalences ranging from 2 to 64% (Crum et al. 1978; Foster et al. 2004). No clinical signs have been reported in infected bears and were similarly not seen in positive bears in this study.

Coccidia oocysts were found on fecal floats from 4 bears. Intestinal coccidia have been sporadically reported in black bears across the United States at low prevalences. Coccidia oocysts from two of the bears were unable to be identified. Coccidia from one bear was identified as *Eimeria* spp., which has been reported in bears in Alberta, Canada at 6 to 8% prevalences (Hair and Mahrt 1970). Coccidia from another bear was identified as *Cryptosporidium* spp., which has been reported in one bear in Virginia that was found dead (Duncan et al. 1999). Other coccidia species have been associated with clinical disease in black bears including a *Cystoisospora*-like parasite, which was associated with encephalitis, and *Sarcocystis* spp. which has been associated with hepatitis, myositis, and encephalitis in California, Nevada, and British Columbia, as well as fatal protozoal hepatitis in two captive bears in South Dakota and British Columbia (Zeman et al. 1993; Davies et al. 2011; Lee et al. 2021; Sinnott et al. 2022). Notably, in these areas *Sarcocystis* spp. have been reported in black bears at much higher prevalences (33.3-40%) than other coccidia species. *Sarcocystis* spp. have also been reported in black bear in eastern states as well as Oregon, at much lower prevalences (1.1-4.3%) (Dubey et al. 1998; Foreyt et al. 1999; Cheadle et al. 2002). While *Sarcocystis* spp. oocysts were not identified on fecal flotations, a *Sarcocystis* spp. cyst was found histologically in the tongue of one bear that died due to vehicular collision. Unfortunately, no fecal sample was collected from this bear, therefore it is unclear whether the bear was shedding sporocysts. No clinical signs were noted on any of the coccidia positive bears in this study. Additionally, no histopathologic lesions were noted on the *Sarcocystis* spp. positive bear.

Numerous other parasites were also found grossly on autopsy or on histology slides of tissues from autopsied bears. *Macracanthorhynchus ingens* was found in the intestines of two bears. *Macracanthorhynchus ingens* has been reported exclusively in black bears in the

southeast. Previous reports indicate a much higher prevalence (21-80.4%) than what was found (5.4%) in this population (Crum et al. 1978; Conti et al. 1983; Foster et al. 2004). Additionally, *Physaloptera* spp. was also found in the stomach of one bear. *Physaloptera* spp. has been previously reported at similar low prevalences in the southeast, as well as in Ontario (Crum et al. 1978; Addison et al. 1978; Conti et al. 1983). *Gongylonema* spp. was found in the tongues of 3 of 22 autopsied black bears, giving a prevalence of 13.6%. This prevalence is much lower than what has previously been reported in black bears in southeastern states, as well as Pennsylvania, which reported prevalences ranging from 33.1 to 54% (Chandler 1950; Crum et al. 1978; Conti et al. 1983). One bear with numerous *Gongylonema* spp. found in its tongue was reported to be severely emaciated (Chandler 1950). Whether the poor body condition was related to the parasitic infection remains unclear. Otherwise, no clinical signs have been reported in infected bears. Similarly, no clinical disease was seen in the three infected bears in this study.

The most notable finds on autopsy include *Trichinella* spp., *Pelodera* spp., *Angiostrongylus vasorum*, and *D. immitis*. *Trichinella* spp. was found in the diaphragm, tongue, and skeletal muscle of one bear. There has been only one report of *Trichinella* spp. in a black bear in the southeast, in one bear in Virginia (Harbottle et al. 1971). Other studies in the southeast have failed to detect *Trichinella* spp. in black bears (Crum et al. 1978; Conti et al. 1983; Sheldon et al. 2022). This finding in our study confirms the presence of *Trichinella* spp. in the black bear population, and potentially other host populations, in Tennessee. Since *Trichinella* spp. is zoonotic and spread through the consumption of undercooked meat of an infected animal, the presence of *Trichinella* spp. in a game species is of particular concern. This finding demonstrates the need for further surveillance in this area.

This is the first report of putative *Pelodera* spp. in a black bear in the southeast. Previous reports in Michigan, Minnesota, Pennsylvania, and New York, have all been associated with alopecia and dermatitis in bears (Fitzgerald et al. 2008; Dykstra et al. 2012; Peltier et al. 2018; Rojas-Sereno et al. 2022). This bear did have some alopecia on its flank, but no associated inflammation was seen histologically. Mild hyperkeratosis and follicular atrophy was seen histologically, though whether it is associated with the nematode is unclear.

This is also the first report of *Angiostrongylus vasorum* in a black bear, as well as the first report of *A. vasorum* in Tennessee. *Angiostrongylus vasorum* is a metastrongylid nematode that infects wild and domestic canids. In North America it is endemic to the Canadian island of Newfoundland but has recently been expanding to other regions including Nova Scotia, Prince Edward Island, and West Virginia (Smith and Threlfall 1973; Conboy 2011; Kistler et al. 2014; Priest et al. 2018; Mahjoub et al. 2022). It is considered the most pathogenic lungworm of canids and can cause severe cardiorespiratory disease in infected dogs (Taubert et al. 2009). Based on the severity of the associated pneumonia *A. vasorum* likely played a role in the death of this bear. The detection of this parasite in Tennessee demonstrates an expansion of its range, placing more wild and domestic mammals, including bears, at risk of disease. The population level impacts of this parasite on bears, as well as other accidental hosts, is unknown and warrants further study.

This is the fourth report of *D. immitis* in American black bears. All previous reports have also been in the southeast (North Carolina, Florida, and Tennessee) (Johnson 1975; Crum et al. 1978; Conti et al. 1983; Sheldon et al. 2022). Clinical signs have not been reported in bears, and similarly were not seen in the yearling in this study.

The presence of other filarial nematodes was examined for using Knott's tests. *Microfilaria* was found in the blood of 69% of bears tested. Sequencing revealed these nematodes to be

Mansonella ozzardi-like filarial nematodes. Blood samples that were not sent to sequencing contained microfilaria with morphology consistent with those sent to sequencing and thus, were considered *M. ozzardi*-like nematodes as well. This is the first report of *Mansonella ozzardi*-like filarial nematodes in black bears in the southeast, and only the second report in the United States. The prevalence seen in this black bear population is even higher than the 50% prevalence reported in black bears in Japan (Masatani et al. 2021). Adults had a significantly higher prevalence than juveniles, likely due to increased exposure. Notably, the size of the *Mansonella ozzardi*-like microfilaria (225-275µm x 2.5-5µm) overlaps with the size of *Dirofilaria ursi* microfilaria (198-242µm x 4-5.5µm) (Michalski et al. 2010). Thus, previous reports of *D. ursi* in black bears based solely on microfilaria morphology, without genetic components, may have misidentified *Mansonella ozzardi*-like microfilaria as *D. ursi*. Thus, the prevalence of this parasite in the broader United States black bear population is not fully understood. Furthermore, many other aspects of this parasite also remain unknown, including its life cycle, anatomical location of the adult nematodes, how the parasites affect bears, and its zoonotic potential. Given that, further investigation into this novel filarial nematode is warranted.

Toxicology tests performed on brain and liver samples from two bears revealed that one bear, that was found dead, had flunixin, a non-steroid anti-inflammatory drug (NSAID) commonly used in equine medicine, in its liver. While, this NSAID has been reported to cause renal failure and gastric ulceration in dogs (Elwood et al. 1992; Vonderhaar and Salisbury 1993), no lesions consistent with flunixin toxicity were identified in the bear. Therefore, the role of this NSAID in the bear's death is unclear. It is worth noting that this same bear had *A. vasorum* eggs and larvae in its lungs, *Trichinella* spp. in its muscles, and *B. transfuga* in its intestines, and thus there were likely many contributing factors in this bear's death. The other bear, that was euthanized due to

seizures, had trace amounts of Bromethalin, a neurotoxic rodenticide, in its brain. Vacuolation of the white matter of the brain seen on histology confirmed the Bromethalin toxicity diagnosis. This is the fourth report of fatal rodenticide toxicity in a black bear. All four cases have occurred within the past four years and three have occurred in the southeastern United States, with two occurring in the same neighborhood (Nasim et al. 2020; Cox et al. 2021; Teo et al. 2023). The two previous cases in the southeast were both caused by bromadiolone and brodifacoum, two anticoagulant rodenticides (Nasim et al. 2020; Teo et al. 2023). While, the other case, in Ontario, was also caused by the neurotoxic rodenticide Bromethalin (Cox et al. 2021). These cases may suggest that rodenticide toxicity is more common in bears than previously thought. Trace amounts of anticoagulant and neurotoxic rodenticides have been commonly detected in black bears throughout California, indicating high exposure rates, though clinical signs are rarely seen (McMillin et al. 2018; Clothier et al. 2022). It is unclear what factors lead to some bears displaying clinical signs while others do not.

The most common cause of death for bears in this study was euthanasia due to human-bear conflict. Human food and garbage often found in the stomachs of these euthanized conflict bears indicate habituation due to the intentional or unintentional feeding of black bears. Furthermore, none of the autopsied conflict bears had any underlying lesions that may help explain their unusual behavior, such as encephalitis. This further indicates that the bears' conflict activity was likely the result of habituation. The second most common cause of death in this study was trauma due to vehicular collision. This is consistent with a previous study in this area, which also found vehicular collision to be a common cause of death among bears (Sheldon et al. 2022). This cause of death is also human mediated. In fact, non-hunter related human-caused deaths, including euthanasia due to conflict activity, vehicular collisions and Bromethalin toxicity,

accounted for over half (65%) of the deaths in this study. These may not be indicative of what is the most common cause of death in the entire bear population in this area, as hunter-killed, roadkill, and euthanized conflict bears are more easily available for study than bears that die of other causes. However, it does demonstrate that human-bear interactions are already common in this area and are likely to increase as bear populations do. Thus, in addition to increased spread of diseases and parasites, increased human-bear conflict activity may also pose a threat to bear health as densities continue to grow.

CHAPTER SIX

CONCLUSION

The results of this study show that there are a wide variety of parasites and diseases present in this black bear population. While no *Sarcoptes scabiei* were detected, other pathogens also known to cause significant disease in black bears including *Ursicoptes americanus*, *Sarcocystis* spp., CAV-1, and CDV were. While no clinical disease was observed in any of the positive bears in this study, previous reports indicate that these pathogens can cause severe disease in bears. Additionally, the presence of *A. vasorum* was found in one bear that had severe pneumonia. This parasite is highly pathogenic in dogs and given the severity of the associated lesions in this bear, may be highly pathogenic to bears as well. The population level impacts of these pathogens are unknown. Thus, further surveillance of these diseases is warranted.

Other notable pathogens detected include *Toxoplasma gondii* and *Trichinella* spp. Both are zoonotic and are spread by the consumption of undercooked meat from infected animals. The presence of these in this area may pose a risk to humans that consume hunter-harvested meat. Hunters in the area should be aware of their presence as well as the precautions that should be taken to avoid infection. Furthermore, there were several pathogens present whose zoonotic potential is not fully understood, including *Babesia* spp., *M. ozzardi*-like nematodes, and *B. transfuga*. Some *Babesia* species, primarily *B. microti* in north America, are known to cause clinical babesiosis in humans (Yabsley and Shock 2013). It is unclear whether the *Babesia* species harbored by raccoons and black bears can cause babesiosis in humans, but they may have zoonotic potential. There is much unknown about the *M. ozzardi*-like nematode prevalent in this black bear population, including its zoonotic potential. *Mansonella ozzardi* can cause clinical

mansonelliasis in humans (Lima et al. 2016). Thus, as a closely related species the *M. ozzardi*-like nematodes may have a similar potential to cause disease in humans. Other species in the genus *Baylisascaris*, primarily *B. procyonis*, have been known to cause potentially fatal visceral, neural, and ocular larval migrans in humans (Wise et al. 2005). While there have been no reported cases of larval migrans caused by *Baylisascaris transfuga* in humans, it has been reported mice and Mongolian gerbils (*Meriones unguiculatus*) (Papini and Casarosa 1994; Papini et al. 1996; Sato et al. 2004). Additionally, macaques housed near raccoons and black bears at a zoo in Japan were reported to have fatal *Baylisascaris* larval migrans caused by either *B. transfuga* or *B. procyonis* (Sato et al. 2005). This suggests that *B. transfuga* may have the potential to cause disease in humans. Additionally, the bears harbored many pathogens known to cause significant disease in domestic dogs including CAV-1, CPV, CDV, *Ancylostoma* spp., *D. immitis*, *A. vasorum*, and capillarid spp. Many of these pathogens are also carried by other peridomestic species, including raccoons, coyotes, and red foxes. Raccoons, in particular, harbor many of the same parasites as bears, including *Babesia* spp. and *M. ingens* among others. Their presence in the black bear population likely indicates a presence in the wider scavenger population in this area.

As bear populations continue to grow and urbanization continues to increase, bears may be pushed further into more urban environments. Human-bear interactions are already common in this area, as demonstrated by the numbers of ‘conflict’ bears in this study and are likely to increase. Interactions between bears and domestic animals, as well among bears will likely also increase. All leading to more opportunities for disease spread, as well as increased conflict. This population health survey will provide essential information to help wildlife managers make

informed management decisions to best protect bear, human, and domestic animal health. It will also lay the groundwork for future black bear health studies in this area.

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APPENDIX

Table 1. Ectoparasites found grossly or by skin scrape on black bears in the southeast sampled 2021-2023

Parasite spp.	Pos/Total	% (95% confidence interval)
Ticks		
<i>Dermacentor variabilis</i>	69/107	64.5% (55.42-73.55%)
<i>Amblyomma maculatum</i>	5/107	4.7% (0.67-8.67%)
<i>Amblyomma americanum</i>	1/107	0.9% (0.0-2.76%)
<i>Ixodes scapularis</i>	1/107	0.9% (0.0-2.76%)
Mites		
<i>Ursicoptes americanus</i>	6/94	6.4% (1.44-11.32%)

Table 2. PCR results for bloodborne pathogens in black bears sampled from the southeast in 2021-2023

Pathogen	Pos./Total	% (95% confidence interval)	Sample
<i>Babesia</i> spp.	10/113	8.9% (3.6-14.1%)	Whole blood
<i>Trypanosoma</i> spp.	0/113	0%	Whole blood
<i>Rickettsia rickettsii</i>	0/113	0%	Whole blood

Table 3. Serology results for *T. gondii*, CAV, CDV, and CPV in black bears from the southeast sampled in 2021-2023

Pathogen	Pos./Total	% (95% confidence interval)	Method
<i>Toxoplasma gondii</i>	78/157	49.7% (41.9-57.5%)	MAT
CAV	1/47	2.1% (0.0-6.25%)	Serology
CDV	9/48	18.8% (7.71-29.79%)	Serology
CPV	1/48	2.1% (0.0-6.12%)	Serology

Table 4. Fecal floatation results for black bears in the southeast sampled 2021-2023

Parasite species	Pos./Total	% (95% confidence interval)
<i>Baylisascaris transfuga</i> *	35/122	28.7% (20.66-36.71%)
Capillarid spp.	16/122	13.1% (7.1-19.1%)
Nematode larvae	11/122	9.0% (3.9-14.1%)
<i>Ancylostoma</i> spp.	2/122	1.6% (0.0-3.89%)
<i>Eimeria</i> spp.	1/122	0.8% (0.0-2.4%)
<i>Cryptosporidium</i> spp.	1/122	0.8% (0.0-2.4%)
Unidentified coccidia	2/122	1.6% (0.0-3.89%)
Total coccidia	4/122	3.3% (0.12-6.4%)

*Three bears that were negative on fecal flotation, but had *B. transfuga* adults found on autopsy were considered positive for *B. transfuga*

Table 5. Nematode and protozoal parasites found by autopsy, Knott's tests, histology, or squash prep in black bears in the southeast sampled 2021-2023

Parasite spp.	Pos./Total	% (95% confidence interval)	Method	Location
Microfilaria*	63/91	69% (59.8-78.7%)	Knott's test	Blood
<i>B. transfuga</i>	6/37	16.2% (4.34-28.1%)	Autopsy	GI
<i>Gongylonema</i> spp.	3/22	13.6% (0.0-27.98%)	Histology/Autopsy	Tongue
<i>M. ingens</i>	2/37	5.4% (0.0-12.69%)	Autopsy	GI
<i>Physaloptera</i> spp.	1/37	2.7% (0.0-7.93%)	Autopsy	Stomach
<i>D. immitis</i>	1/37	2.7% (0.0-7.93%)	Autopsy	Heart
<i>Trichinella</i> spp.	1/20	5% (0.0-14.55%)	Histology/Squash prep	Diaphragm, tongue, skeletal muscle
<i>Angiostrongylus vasorum</i>	1/33	3% (0.0-8.88%)	Histology	Lung, lymph node
<i>Pelodera</i> spp.	1/26	3.9% (0.0-11.24%)	Histology	Skin
<i>Sarcocystis</i> spp.	1/28	3.6% (0.0-10.45%)	Histology	Tongue

*Likely *Mansonella ozzardi*-like filarial nematode

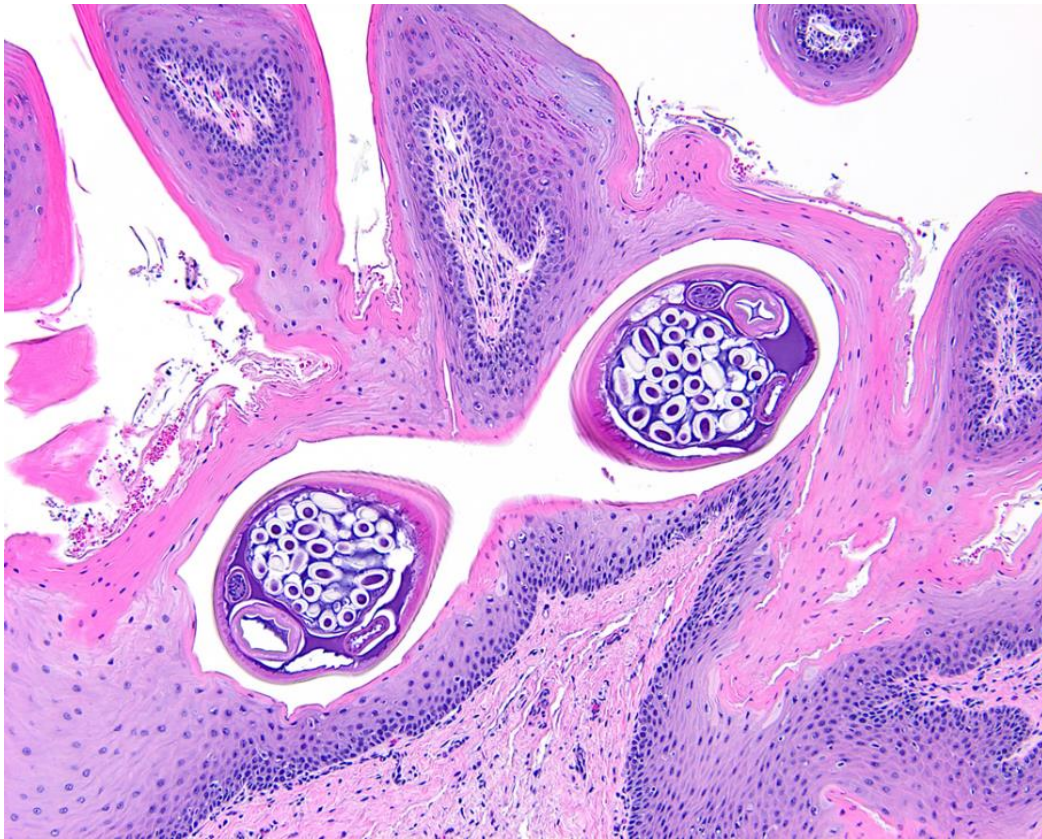


Figure 1. Adult *Gongylonema* spp. in the tongue of a black bear

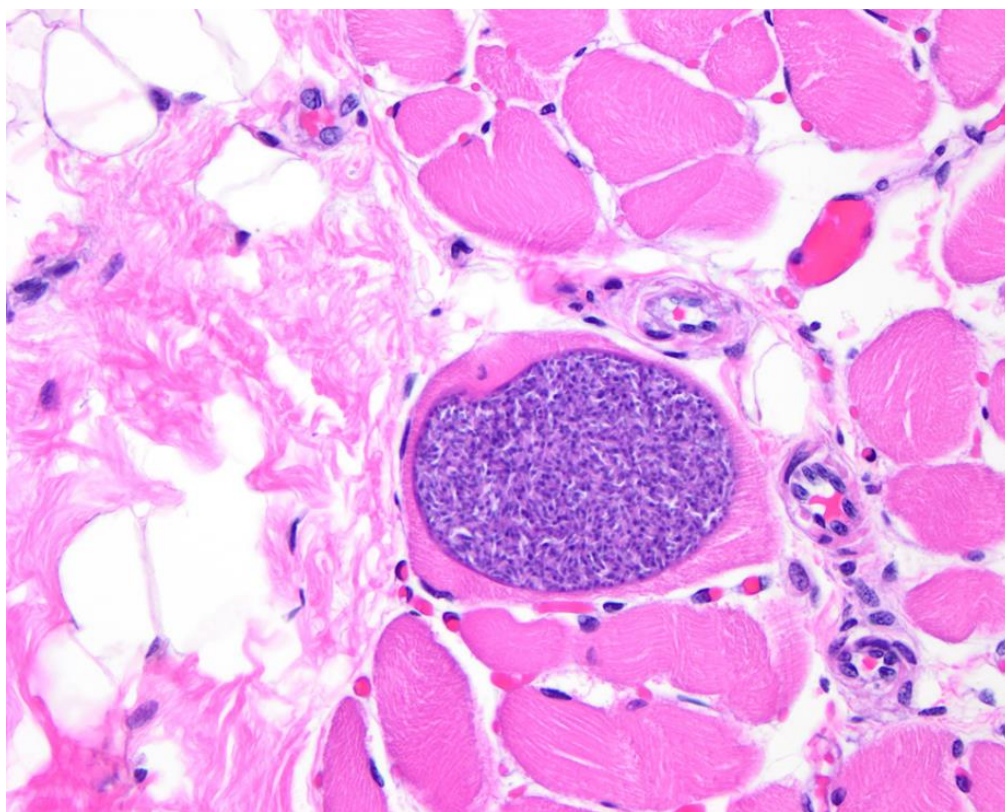


Figure 2. A *Sarcocystis* spp. cyst in the striated muscle of the tongue of a black bear

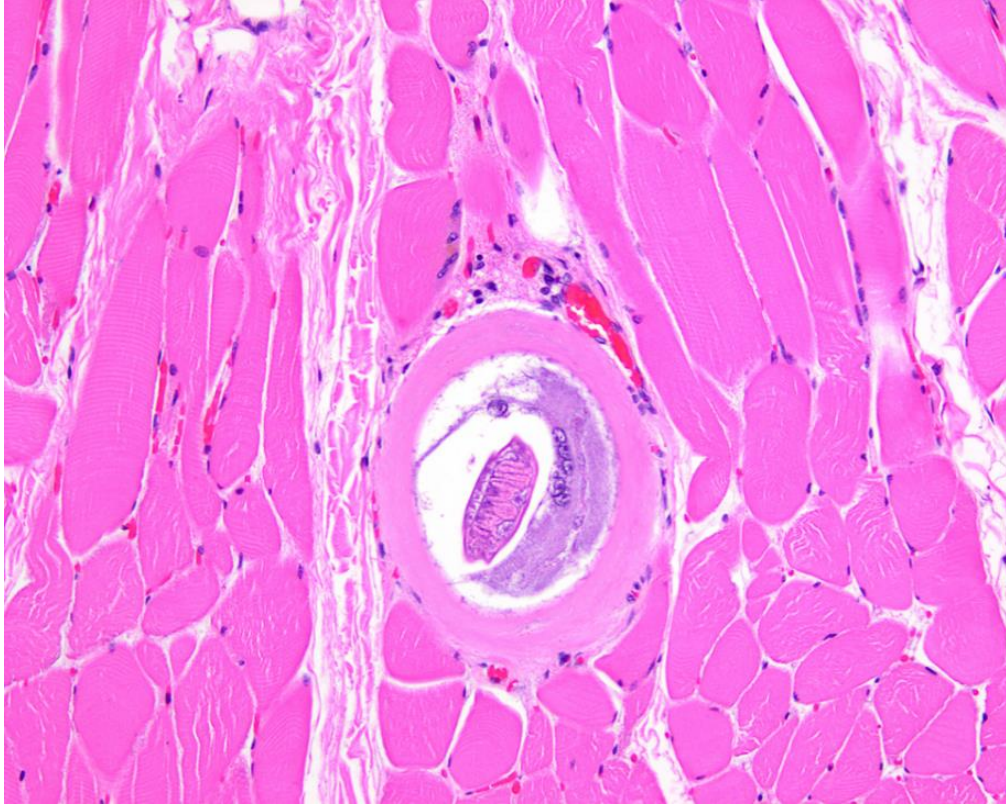


Figure 3. *Trichinella* spp. larva encysted in the tongue of a black bear

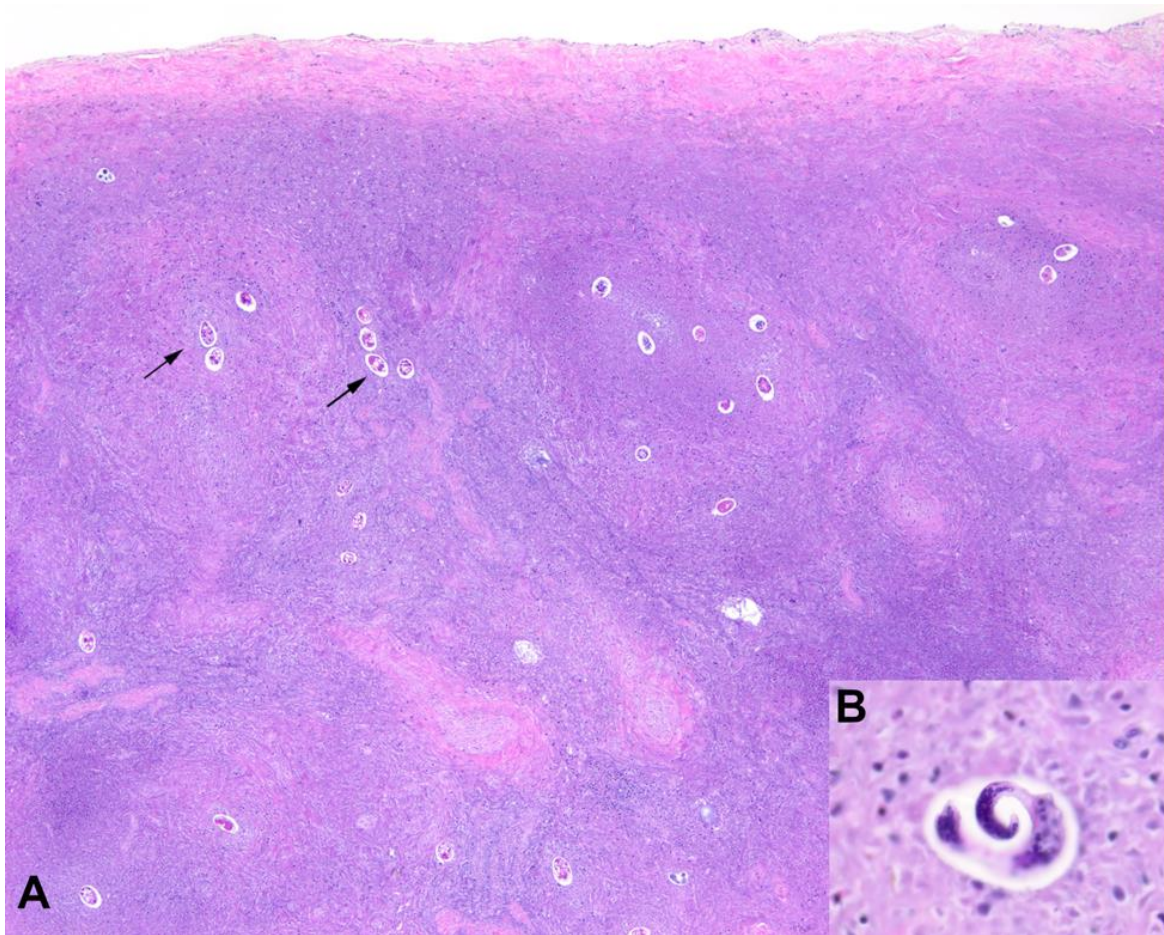


Figure 4. Severe eosinophilic and granulomatous pneumonia affecting normal lung parenchyma with numerous intralesional *Angiostrongylus vasorum* eggs (arrows) in a black bear from Tennessee (A). Larvated *A. vasorum* egg in lung tissue (B)

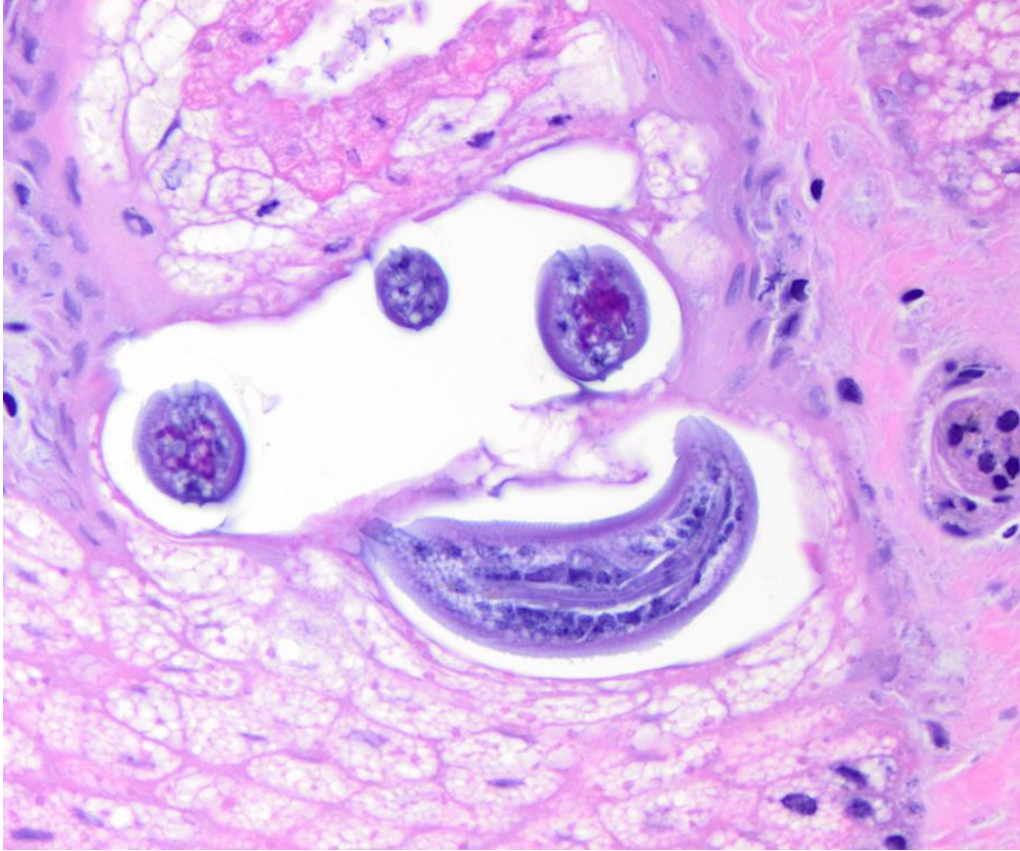


Figure 5. *Pelodera* spp. larva in the skin of a black bear

VITA

Kathleen “Katie” Riese was born April 16, 1998, in Lancaster, Pennsylvania, and grew up in Chattanooga, Tennessee. After graduating from Ooltewah Highschool in 2016, Katie was accepted to the University of Tennessee, Knoxville, where she majored in Wildlife and Fisheries Science with a concentration in Wildlife Health. While pursuing her undergraduate degree she became a member, and later the secretary and treasurer, of the Wildlife Disease Association UT student chapter, which sparked her interest in wildlife disease research. Also while in undergraduate she had the opportunity to become an undergraduate research assistant and work with Dr. Wesley Sheley on her research regarding *Batrachochytrium salamandrivorans* (Bsal) in salamanders, which further solidified her decision to pursue a career in wildlife disease research.

Katie graduated with her Bachelor of Science in May 2020, and was accepted into UT’s Comparative and Experimental Medicine program as a Masters student in August 2021. Katie’s graduate research focused on the parasites, diseases, and causes of mortality in the southeastern black bear population. The goal of her research was to help wildlife managers and set a foundation for future black bear health research. After graduation she will continue to work in wildlife research as a research technician working with Dr. Richard Gerhold and Dr. Michelle Dennis at the University of Tennessee College of Veterinary Medicine.