International Journal for Parasitology: Parasites and Wildlife 13 (2020) 27–37

Contents lists available at ScienceDirect

International Journal for Parasitology: Parasites and Wildlife

journal homepage: www.elsevier.com/locate/ijppaw

"Weight of evidence" as a tool for evaluating disease in wildlife: An example assessing parasitic infection in Northern bobwhite (*Colinus virginianus*)

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ARTICLE INFO

Keywords:
Bobwhite
Conservation
Parasites
Weight of evidence
Wildlife disease

ABSTRACT

The potential of parasites to affect host abundance has been a topic of heated contention within the scientific community for some time, with many maintaining that issues such as habitat loss are more important in regulating wildlife populations than diseases. This is in part due to the difficulty in detecting and quantifying the consequences of disease, such as parasitic infection, within wild systems. An example of this is found in the Northern bobwhite quail (*Colinus virginanus*), an iconic game bird that is one of the most extensively studied vertebrates on the planet. Yet, despite countless volumes dedicated to the study and management of this bird, bobwhite continue to disappear from fields, forest margins, and grasslands across the United States in what some have referred to as "our greatest wildlife tragedy". Here, we will discuss the history of disease and wildlife conservation, some of the challenges wildlife disease studies face in the ever-changing world, and how a "weight of evidence" approach has been invaluable to evaluating the impact of parasites on bobwhite in the Rolling Plains of Texas. Through this, we highlight the potential of using "weight of the evidence" to better understand the complex effects of diseases on wildlife and urge a greater consideration of the importance of disease in wildlife conservation.

1. Introduction to the history of disease in wildlife conservation

Aldo Leopold, considered by many as the father of wildlife management, penned that disease was underestimated in wildlife conservation in his 1933 treatise "Game Management", a work that would go on to become a cornerstone for wildlife management in North America. Now in 2020, with climate change, habitat degradation, invasive species, and growing human populations eroding the wild heritage Leopold sought so fervently to protect, these words are more pertinent than the day they were written. Yet, it was only recently that wildlife diseases became a "New Frontier" in conservation (Fagerstone, 2014; Friend, 2014), likely due to the persistent paradigm that diseases are a natural regulatory mechanism of healthy populations with the ultimate outcome being one in which the host was not harmed (Elton, 1931; Lack, 1954). However, Leopold looked past this narrow view of diseases, stressing the need to consider the effects of factors such as microbes, parasites, contaminants, malnourishment, and any combination thereof, a perspective which was far ahead of its time. Today, prominent wildlife disease researchers have adopted similar views, with Wobeser (2006) defining disease as "any impairment that interferes with or modifies the performance of normal functions, including responses to environmental factors such as nutrition, toxicants, and climate; infectious agents; inherent or congenital defects; or combinations of these factors''. Hereafter, we will use this definition of disease as it accounts for the complexity associated with disease impacts on wildlife populations and may thus provide greater insight into the topic.

However, the history of diseases in the United States is long and varied (Fig. 1). Surges in disease reports, which were often initiated due to mortality being large and conspicuous (Plowright, 1988), were typically followed by an initiative that addressed the disease because the public demanded a response (Kadlec, 2002; Friend, 2014). Concerns for disease would eventually wane as other concerns took precedence. For example, people became more worried about chemical contaminants and heart disease after Rachel Carson published the seminal book "Silent

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https://doi.org/10.1016/j.ijppaw.2020.07.009
Received 24 June 2020; Received in revised form 27 July 2020; Accepted 27 July 2020
Available online 31 July 2020

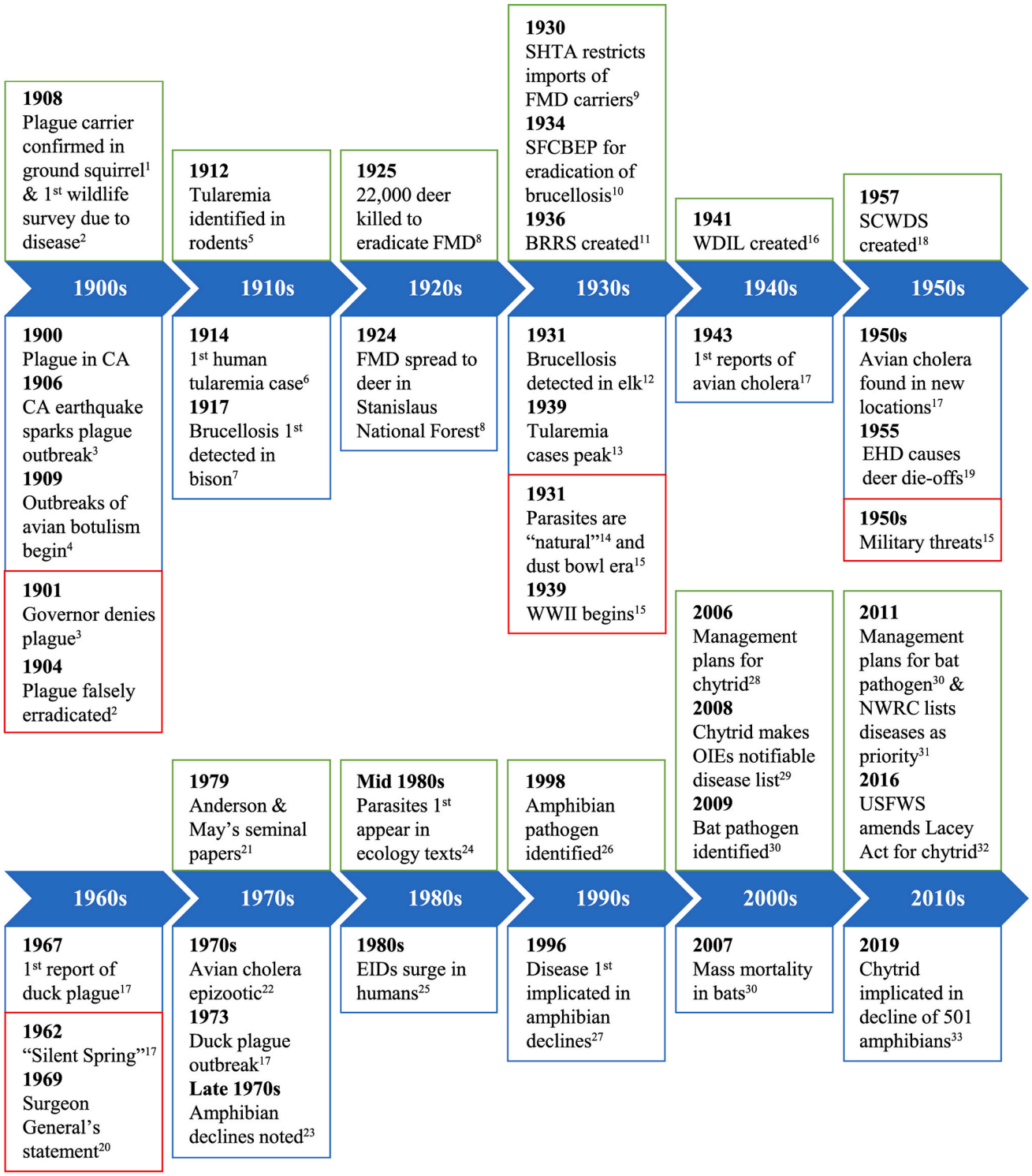

Fig. 1. Timeline depicting the history of wildlife diseases in the United States: blue boxes are for disease reports and outbreaks, green for improvements to disease research, and red for events that hindered disease research. Abbreviations: foot-and-mouth disease (FMD), Smoot-Hawley Tariff Act (SHTA), State-Federal Cooperative Brucellosis Eradication Program (SFCBER), Bear River Research Station (BRRS), Wildlife Disease Investigations Laboratory (WDIL), Southeastern Cooperative Wildlife Disease Study (SCWDS), epizootic hemorrhagic disease (EHD), World Organisation for Animal Health's (OIE), National Wildlife Research Center (NWRC), U. S. Fish and Wildlife Service (USFWS). References: 1. Antolin et al. (2002), 2. Creel (1941), 3. Anderson (1978), 4. Locke and Friend (1987), 5. McCoy and Chapin (1912), 6. Wherry and Lamb (1914), 7. Meagher and Meyer (1994), 8. Clements (2007), 9, Bachrach (1968), 10. Busch and Parker (1972), 11. USFWS (1991), 12. Tunnicliff and Marsh (1935), 13. Brooks and Buchanan (1970), 14. Elton (1931), 15. Brown (2007), 16. CDFW 2019, 17. Friend (2014), 18. SCWDS 2019, 19. Shope et al. (1960), 20. Cohen (2000), 21. Cross et al. (2013), 22. Samuel et al. (2007), 23. Carvalho et al. (2017), 24. Dobson and Hudson (1986), 25. Jones et al. (2008), 26. Berger et al. (1998), 27. Laurance et al. (1996), 28. Collins and Crump (2009), 29. OIE 2008, 30. Voyles et al. (2015), 31. Fagerstone (2014), 32. USFWS (2016), 33. Scheele et al. (2019). (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

Spring" in 1962 and the Surgeon General announced that we should "close the book on infectious disease" in 1969 (Cohen, 2000; Friend, 2014).

Despite this, progress has been made regarding wildlife disease research and conservation, particularly considering that prior to 2000 wildlife diseases were largely ignored unless they affected game species or livestock (Fagerstone, 2014; Cunningham et al., 2017; Preece et al., 2017). For example, the lessons learned from chytridiomycosis, which has been implicated in the decline or extinction of 501 amphibian species (Scheele et al., 2019), facilitated a quicker response to white-nose syndrome in bats, with the pathogen being identified and management plans in place within four years (Voyles et al., 2015). Then in 2011, the National Wildlife Research Center (NWRC) listed wildlife diseases as a priority for the first time on their research needs assessment (Tobin et al., 2012; Fagerstone, 2014). Unfortunately, the wildlife diseases that still gain the most attention either cause massive die-offs, affect game species, domestic animals, or humans (Deem et al., 2001; Friend, 2014; Polley and Thompson, 2015); meanwhile diseases with less obvious but substantial effects are overlooked (Wobeser, 2006; Wood and Johnson, 2015). Moreover, the interactions between disease and other variables are largely unknown, and while our understanding of the ecological influence of parasites has improved, it is still very much incomplete (Hernández and Peterson, 2007; Kendall et al., 2010; Tompkins et al., 2011; Wood and Johnson, 2015).

The growing human presence has also been implicated in the spread and proliferation of wildlife diseases through environmental contaminants, land use changes, shifts in animal populations, and climate change (Daszak et al., 2000, 2001; Deem et al., 2001; Dobson and Foufopoulos, 2001). Some of these factors, such as environmental contaminants, are directly correlated with intensification of wildlife diseases (Borošková et al., 1995; Bichet et al., 2013), whereas others like climate change have more insidious effects (Lafferty, 2009). In Table 1, we provide a broad overview of the various ways in which human activities may exacerbate the impact of wildlife diseases, citing specific examples of each. While this overview is by no means a comprehensive account of the subject, it serves to emphasize the potentially increased ecological role of wildlife diseases, and heightened importance of disease management in the Anthropocene. This is especially true considering the potentially catastrophic effects of diseases may be obscured by the intricate associations present in wild systems (McCallum, 2000; Friend et al., 2001). But how then, do we approach the problem of unraveling these complex interactions and determine the true impact of diseases on wildlife?

2. Weight of evidence and its uses

In order to determine the ultimate impacts of diseases on wildlife, it is necessary to assess specific effects on population attributes through randomized and controlled studies. However, as discussed in the previous section, the impacts of diseases are often discreet, dynamic, and influenced by multiple interacting variables. Furthermore, correlative associations may be the only evidence that diseases are influencing a particular system and designing and implementation of empirical studies to assess these impacts for every population potentially affected by disease may not be possible. It is therefore necessary to determine whether further study of a system potentially at risk of being compromised by disease is needed.

Weight of evidence (WOE) is "… an inferential process that assembles, evaluates, and integrates evidence to perform a technical inference in an assessment" used by the U.S. Environmental Protection Agency for a variety of assessments (Suter et al., 2017). The WOE approach is also effective for evaluating scenarios *post hoc*, where data is typically limited and/or only correlative (Forbes and Calow, 2002). Consequently, WOE may provide a valuable tool to investigate other multivariate problems outside of risk assessment, such as determining the causes of wildlife population declines. Adaptations of WOE have been effectively used in this regard by researchers investigating the effects of multiple stressors on aquatic systems (Lowell et al., 2000; Adams, 2005; Burkhardt-Holm and Scheurer, 2007).

For instance, Burkhardt-Holm and Scheurer (2007) employed a WOE approach to identify potential causes for the decline in brown trout (*Salmo trutta*) by using an adaptation of the WOE framework developed by Forbes and Calow (2002). In this, they used a series of 7 questions to assess the plausibility, exposure, correlation, threshold, specificity, experiments, and then removal of the variable of interest. These 7 questions promote a rigorous way of evaluating data that does not discount the plausibility of factors for which there may be only limited and/or correlative data. This method allowed Burkhardt-Holm and Scheurer (2007) to overcome uncertainty and confounding variables to positively identify proliferative kidney disease as the most likely cause of brown trout declines in half of their study areas. However, despite the success of WOE based studies in assessing population stressors in aquatic systems, to our knowledge, similar evaluations are lacking in terrestrial environments, which are also subject to an array of complex and variable stressors.

Here, the seven questions proposed by Burkhardt-Holm and Scheurer (2007) (Fig. 2a) were modified to specifically address disease(s) in wildlife populations (Fig. 2b). The original questions were robust and widely applicable, and this allowed us to adhere closely to the original framework, with 1 question remaining unchanged and 4 others only being rephrased to incorporate the disease aspect. However, 2 of the questions were modified to better evaluate the potential impacts of diseases given the complex nature and specific characteristics of the topic. Namely, thresholds of infection in diseases are often difficult to discern and concrete thresholds are typically unavailable. Thus, question 4 is now used to determine whether there is an apparent threshold in which the disease elicits an observable or quantifiable response in the individual host, and this leads to question 5, which considers if this

Table 1
Examples for various anthropogenic factors and their influence on wild systems.

Organism	Anthropogenic Factor	Result	Reference
Sparrows	Pollution	Trace metals increase susceptibility to malaria	Bichet et al. (2013)
Rats	Pollution	Chronic exposure to lead at low concentrations leads to immunosuppression	Bendich et al. (1981)
Timber rattlesnakes	Habitat fragmentation	Inbreeding depression and pathogenic fungal outbreak	Clark et al. (2011)
Lesser Antillean bullfinch	Habitat fragmentation	Increased prevalence of two blood parasites	Perez-Rodriguez et al., 2018
Bumble bees	Habitat fragmentation	Decreased genetic diversity and increased pathogen prevalence	Cameron et al. (2011)
Christmas Island rat	Animal translocation	Introduction of black rats causes outbreak of trypanosomiasis and eventual extinction	Wyatt et al. (2008)
Bighorn sheep	Introduced diseases	Livestock diseases hinder conservation efforts due to lack of resistance in bighorn sheep	Singer et al. (2001); Clifford et al. (2009)
Harbor seals	Climate change	Migratory changes in harp seals cause exposure to phocine distemper virus	Jensen et al. (2002)
Great pond snail	Climate change	High ambient temperatures cause reduced immune defense	Seppälä and Jokela, 2010

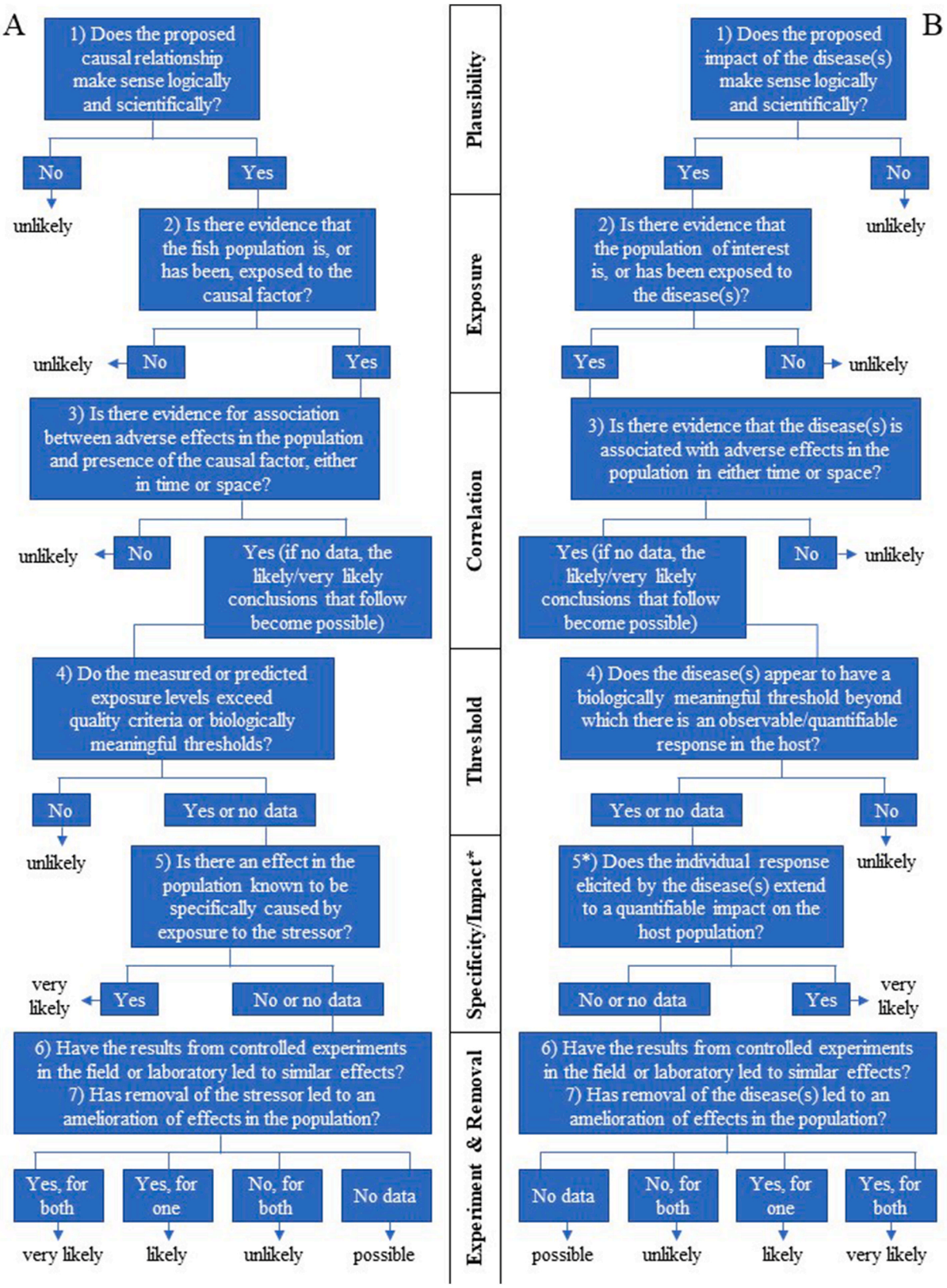

Fig. 2. Flow diagrams showing a weight of evidence framework using the (A) 7 questions proposed by Burkhardt-Holm and Scheurer (2007) and the (B) modified questions for addressing disease(s) in wildlife.

effect may be impactful at the population level. Structuring the questions in this way permits a better evaluation of the impacts a disease may have on a populations as highly pathogenic diseases present at a low frequency may have less of an impact on a population than a widespread disease with subtle symptoms (McCallum and Dobson, 1995).

3. Bobwhite, their decline, and the possible role of parasites

Bobwhite are an iconic species and one of the most extensively studied and heavily managed gamebirds (Rosene, 1969; Scott, 1985; Hernández et al., 2013). While bobwhite declines have been noted since the late 1800s (Stoddard, 1931; Rosene, 1969), systematic surveys of bird abundance in the 1960s reinforced the severity and significance of these declines (Sauer et al., 2013), as did documentation of widespread localized extinctions of bobwhite (Brennan et al., 2007; Palmer et al., 2012). This has led to a great deal of effort to determine and mitigate factors contributing to bobwhite declines, and many consider habitat loss and degradation to be the primary threat to bobwhite across their range (Palmer et al., 2012; Hernández et al., 2013). However, bobwhite are also influenced by other factors, including predation (Rollins and Carroll, 2001), weather (Lusk et al., 2002), contaminants (Ertl et al., 2018), diseases (Peterson, 2007), and climate change (Guthery et al., 2000), which interact to exert a cumulative effect on bobwhite populations (Hernández and Peterson, 2007; Hernández et al., 2013).

While bobwhite have been undergoing a general decline since the late 1800s, more recently, researchers have documented bobwhite populations faltering in places that have long been considered strongholds for the species (Brennan et al., 2007; Rollins, 2007). One of these areas is the West Texas Rolling Plains, a region where the dominant land use type (rangeland) is conducive to quail management and where land may often be managed for bobwhite (Rollins, 2007; Hernández and Guthery, 2012). Despite this, in 2010 bobwhite populations in the Rolling Plains failed to irrupt during a year where plentiful rainfall and quality habitat led to predictions of a quail boom. This led to the launch of Operation Idiopathic Decline (OID), a collaborative research effort to investigate the impact of contaminants and diseases on bobwhite from the region. During OID, researchers from major Texas universities collaboratively surveyed bobwhite in the Rolling Plains and found a high prevalence of eyeworms (*Oxyspirura petrowi*) and caecal worms (*Aulonocephalus pennula*; = *A. lindquisti*), with infection rates as high as 66% and 91%, respectively (Bruno, 2014).

Even though this was not the first time these parasites had been reported in bobwhite from the Rolling Plains (Jackson and Green, 1965; Jackson, 1969), OID marked the beginning of a determined investigation into the potential of parasitic infection to affect bobwhite in the area. Prior to OID, diseases, including parasites, were generally viewed as inconsequential in terms of bobwhite management (Stoddard, 1931; Rollins, 2002; Peterson, 2007), despite some researchers arguing for a greater consideration of their impacts (Robel, 1993; Brennan, 2002; Peterson, 2007). This perspective set the paradigm for bobwhite conservation, and to this day, habitat and land management practices are the predominant means of maintaining local bobwhite populations (Hernández and Guthery, 2012). However, even in areas where habitat is carefully and specifically managed for quail, such as the Rolling Plains Quail Research Ranch (RPQRR), bobwhite populations continue to follow the boom and bust cycles characteristic of the species (Thogmartin et al., 2002; Rollins, 2018; Texas Parks and Wildlife Department , 2019). While proper habitat management is a foundation for successful bobwhite conservation, the cause of bobwhite population fluctuations remains undetermined (Guthery, 2002; Hernández et al., 2002) and bobwhite continue to experience a range-wide decline (Sauer et al., 2013). This suggests that additional factors are influencing bobwhite abundance, and it is possible that parasites have a greater effect on bobwhite populations than previously purported.

4. Using the WOE framework to investigate the role of parasites in the bobwhite decline

Using a WOE approach that integrates data from field and laboratory studies, augmented by the observations and collaboration of local landowners and quail hunters, may yield a more comprehensive understanding of how parasites affect bobwhite population dynamics. This method is employed by the Wildlife Toxicology Laboratory (WTL) at Texas Tech University when investigating the implications of *O. petrowi* and *A. pennula* in bobwhite of the Rolling Plains. *Oxyspirura petrowi* and *A. pennula* are parasitic nematodes that infect the eyes and caeca, respectively, of their definitive hosts, and undergo an indirect life cycle requiring an insect intermediate host for transmission (Chandler, 1935; Addison and Anderson, 1969; Peterson, 2007). This indirect life cycle further exacerbates the already complex task of understanding the consequence of infection, making the WOE approach particularly valuable in this instance.

We compiled what was, to our knowledge, all available information regarding *A. pennula* and *O. petrowi* in bobwhite. We then subjected this information to the series of 7 questions discussed in section 2. In doing so, we can establish whether the research conducted thus far holds enough weight to warrant continued investigations into this issue and demonstrate the value of a WOE approach. We begin by addressing the first of the 7 fundamental questions, that of plausibility.

4.1. Question 1: Does the proposed impact of the disease(s) make sense logically and scientifically?

In 1979, Anderson and May provided the theoretical justification for the ability of parasites to suppress host abundance to the extent in which this results in cyclical fluctuations of host populations (Anderson and May 1979; May and Anderson, 1979). Since then, our understanding of host parasite interactions has advanced from this theoretical foundation, to one in which parasites are increasingly recognized for their potential to affect hosts at the population scale and higher, even when the effects are not immediately apparent (Tompkins et al., 2011). This increasing recognition of parasites as a mechanism affecting host population dynamics is mirrored with regards to the effects of parasites on bobwhite in the Rolling Plains ecoregion of West Texas.

While parasites have long been known to infect bobwhite of the Rolling Plains, their significance in terms of bobwhite conservation has remained largely obscure. However, contemporary investigations of parasites in the region have revealed epizootic events, a high prevalence, and the potential of two helminths, *A. pennula* and *O. petrowi*, contributing to the declines of local bobwhite populations (Bruno, 2014; Dunham et al., 2014a; Bruno et al., 2019a). Evidence exists of parasites like *Loa* and *Thelazia callipaeda*, which are closely related to *O. petrowi* (Xiang et al., 2013; Kalyanasundaram et al., 2018a), causing irritation and impaired vision in their hosts (Otranto et al., 2004; Barua et al., 2005; Nayak et al., 2016). Moreover, Kalyanasundaram et al. (2018a) determined *A. pennula* to have a 90% relation to the ascarids, specifically *Toxascaris leonine* which is common parasite of cats and dogs that is known to cause nutrient loss, weight loss, and death (Kalyanasundaram et al., 2017).

In birds, similar intestinal parasites have also been documental to cause inactivity, weight loss, growth reduction, and inflammation of the caecal mucosa in infected individuals (DeRosa and Shivaprasad, 1999; Vandegrift et al., 2008; Nagarajan et al., 2012). Field studies have demonstrated the capacity of parasites to exhibit effects on hosts at the population level as well, as in the case of the caecal worm, *Trichostrongylus tenuis*, which suppressed populations of another Galiforme, the red grouse (*Lagopus scoticus*), by reducing fecundity and increasing susceptibility to predators (Hudson, 1986; Hudson et al., 1992, 1998). As such, the potential of parasites to induce population decline in bobwhite quail is being increasingly recognized as a plausible threat which necessitates further investigation.

4.2. Question 2: Is there evidence that the population of interest is, or has been exposed to the disease(s)?

The first reported evidence of parasites in bobwhite from the Rolling Plains of Texas came in the 1940s, when Webster and Addis (1945) documented a number of parasites, including caecal worms (*A. lindquisti*). Further study into the parasite fauna of bobwhite in the Rolling Plains did not occur until the 1960s, when Jackson and Green (1965) conducted more rigorous assessments and found *A. pennula* and *O. petrowi* to be relatively common in the regions quail. With the exception of studies conducted by Rollins (1980) and Demarais et al. (1987) in the late 1970s and early 1980s, research into parasitic infection of bobwhite in Texas waned once again, until over 50 years later.

Villarreal et al. (2012) renewed the investigations into the *O. petrowi* that infect bobwhite, finding 57% of bobwhite to be infected from 2007 to 2011, and during sampling from February 2010–January 2011, 82% of bobwhite were infected with *A. pennula* (Villarreal et al., 2016). Additionally, OID sparked a proliferation of studies investigating the impacts of parasites on bobwhite of the Rolling Plains and South Texas, and these studies have documented *A. pennula* and *O. petrowi* to be ubiquitous in quail throughout the region (Dunham et al., 2016a; Olsen and Fedynich, 2016). In subsequent studies, Bruno et al. (2019b) found 40% of bobwhite sampled from 2011 to 2013 to be infected with *O. petrowi*, while 73% were infected with *A. pennula*. However, Dunham et al. (2014a) suspected previous surveys underreported eyeworm prevalence as those studies only examined the nictitating membrane and the surface of the eye. By examining eye-associated tissues as well, Dunham et al. (2014a) found 97% of bobwhite infected with *O. petrowi*. Today, the Rolling Plains is considered to be the epicenter of caecal worm and eyeworm infection in bobwhite (Kubečka et al., 2017), and surveys regularly yield infection rates approaching 100% with infection levels averaging >400 *A. pennula* and >30 *O. petrowi* (Henry et al., 2017; Brym et al., 2018b; RPQRF, 2019).

Oxyspirura petrowi have also been documented in wild turkey (*Meleagris gallopavo*; Kubečka et al., 2018), songbirds (Dunham and Kendall, 2014), lesser prairie-chickens (*Tympanuchus pallidicinctus*; Dunham et al., 2014b), Gambel's (*Callipepla gambelii*), and scaled quail (*Callipepla squamata*) (Dunham and Kendall, 2017), while *A. pennula* have been found in scaled quail (Dunham et al., 2017a) and wild turkey (Hon et al., 1975). The wide range of hosts for *O. petrowi* and *A. pennula* highlights the possibility that these parasites may be more widely distributed that previously thought, and if bobwhite populations recover the parasites may remain in reservoir hosts and be capable of infecting bobwhite in the future.

4.3. Question 3: Is there evidence that the disease(s) is associated with adverse effects in the population in either time or space?

Because wild bobwhite populations in the Rolling Plains are under constant and simultaneous exposure to a variety of dynamic and interacting stressors, determining a causative link between disease and its effect(s) on bobwhite populations may be extremely difficult. Consequently, correlative associations may provide a more pragmatic alternative into potential interactions as these may be the only available evidence in these circumstances. Correlative associations are typically supported by models of parasite induced host mortality (PIHM), which predict lower parasite burdens in surviving hosts due to the concurrence of host mortality and infection intensity (Wilber et al., 2016). However, Wilber et al. (2016) cautioned that models alone do not provide conclusive evidence of PIHM but should instead be used as a supplement to experimentation and a comprehensive understanding of parasite host interactions. Thus, we must also consider the cumulative effect of these parasites in order to obtain a clearer picture of the "parasitic pressure" a host may be facing (Bordes and Morand, 2009).

For instance, heavy precipitation during 2016 (RPQRF, 2016) led to favorable conditions for the arthropod intermediate hosts of *O. petrowi* and *A. pennula* (Branson, 2014; Kistler et al., 2016a; Almas et al., 2018; Henry et al., 2018, 2020), which coupled with high bobwhite populations (TPWD, 2019), created an environment rich in both intermediate and definitive hosts, an ideal situation for the proliferation of parasites (Sures and Streit, 2001; Liccioli et al., 2014). This may have facilitated the transmission of parasites leading to the increased infection levels of both *O. petrowi* and *A. pennula* during the spring of 2017 (Henry et al., 2017). The increased intensity of the parasites was concomitant with greater difficulty trapping bobwhite, and then a subsequent die-off of bobwhite that was speculated to be due to PIHM.

Following the hunting season of 2017–2018, Brym et al. (2018c) also reported difficulty in trapping bobwhite, reinforcing previous reports suggesting a scarcity of birds that may have resulted from the consistently elevated parasite burdens documented throughout the region. Commons et al. (2019) likewise documented difficulty trapping amidst high parasite burdens compared with previous years. This scarcity of birds was found throughout the region when TPWD (2019) reported the third lowest amount of bobwhite seen since 1978 in 2018, leading to concern for localized extinctions of bobwhite. Ultimately, there appears to be a correlation between high parasite burdens and reduced bobwhite abundance, and this link needs to be investigated further.

4.4. Question 4: Does the disease(s) appear to have a biologically meaningful threshold beyond which there is an observable/quantifiable response in the host?

While the widespread incidence of *O. petrowi* in the Rolling Plains may have exposed a large proportion of bobwhite to infection, it is also important to consider the intensity of these infections, as even highly pathogenic organisms may have negligible effects on their hosts if present only in low numbers (Fredensborg et al., 2004). Consequently, Dunham et al. (2017a) developed infection level thresholds to provide a systematic way of gauging the intensity of parasitic infection in bobwhite; during which, a large proportion of bobwhite (48%) were found to have lower eyeworm infections (<20 *O. petrowi*), while relatively few (15%) were heavily parasitized (>40 *O. petrowi*). Dunham et al. (2017a) hypothesized that this was due to highly infected individuals suffering reduced fitness, which ultimately led to mortality, and this is consistent with models of populations experiencing PIHM discussed in section 4.3.

The hypothesis of Dunham et al. (2017a) is further supported as throughout 2017 heavy parasite burdens were documented in bobwhite that landowners observed flying into obstacles, being taken by predators, and two specimens that were hand captured (Brym et al., 2018a). These anecdotal accounts of parasitized bobwhite exhibiting signs of potential visual impairment parallel reports by Jackson (1969), who was the first to report such behavior in parasitized bobwhite. There was also a bobwhite that was hand captured during the 2017–2018 hunting season was severely emaciated and possessed an extreme caecal worm infection (n = 1722) (Brym et al., 2018c). Collection of a bobwhite with such a high parasite load was unusual, as Dunham et al. (2017a) found 50% of bobwhite with <100 *A. pennula* and only 19% with >200, and the high infection may have contribute to the birds condition given that pathological changes were noted in the caeca of scaled quail infected with >100 caecal worms (Rollins, 1980).

Hunters from the Rolling Plains continued to donate parasitized bobwhite during the 2017–2018 hunting season, in which the highest average intensities of both *O. petrowi* (n = 44) and *A. pennula* (n = 599) were recorded (Brym et al., 2018b). This sample also exhibited an increased proportion of birds in the strong and extreme infection level range when compared to earlier surveys of parasites. Towards the end of the hunting season, hunters began reporting fewer coveys and more feather piles indicating predation that coincided with consistently elevated parasites burdens (Brym et al., 2018c). Moreover, Kalyanasundaram et al. (2018b) documented an increase in *Physaloptera* sp. In bobwhite infected with *A. pennula* and *O. petrowi*, leading them to

postulate that bobwhite with high levels of these parasites may be more susceptible to co-infection with other helminths. These reports suggest that at strong and extreme levels of infection a threshold is reached in which bobwhite survival may be reduced and/or bobwhite become immunocompromised.

4.5. Question 5: Does the individual response elicited by the disease(s) extend to a quantifiable impact on the host population?

Anecdotal accounts of eyeworm infected bobwhite from the Rolling Plains exhibiting erratic behavior (Jackson, 1969) spurred concerns that these parasites may be causing visual impairment by damaging structures within the eyes of infected individuals. Later, researchers conducted pathological assessments of the eyes of infected bobwhite and confirmed inflammation and damage to the eye tissues and cornea of bobwhite hosts, as well as hemorrhaging of nasolacrimal ducts (Bruno et al., 2015; Dunham et al., 2015, 2016b; Hunter, 2016). Because bobwhite are highly dependent on their sense of vision when foraging, navigating their environment, and evading predators, the potential effects of impaired vision may be substantial. However, currently it is unknown precisely how eyeworm infection impacts the visual acuity of infected bobwhite and how this may impact their interaction with and ability to survive in the environment. Although pathological evidence suggests that damage is occurring and is supported by anecdotal accounts of impaired vision in quail infected with *O. petrowi* (Brym et al., 2018a, 2018c), additional research is necessary in order to evaluate the effect of *O. petrowi* on bobwhite vision and overall impact it could have on a population.

Caecal worm infections have also been correlated with negative impacts on bobwhite. For example, Dunham et al. (2017b) demonstrated that birds with *A. pennula* are often found with only minimal amounts of digesta in the caeca, while Lehmann (1953, 1984) associated high burdens with lower levels of vitamin A and drought. These observations led the researchers to postulate that caecal worm infection could reduce feed intake, impede digestion, and exacerbate periods of stress, which are commonly associated with intestinal parasites (Petkevičius, 2007). Due to the important functions of the avian caecum, such as nutrient and water absorption, antibody production, and cellular digestion (Clench and Mathias, 1995), disruption to its function may indeed exacerbate periods of stress for bobwhite, such as drought and food scarcity. In addition to being coincident with periods of low precipitation, caecal worm infections are also known to peak in winter (Lehmann, 1953; Rollins, 1980; Blanchard et al., 2019), and both of these periods typically result in high mortality for bobwhite (Hernández et al., 2005; Hernández and Peterson, 2007). Furthermore, the effects of *A. pennula* may not only be limited to survivability, but these worms could also impede the breeding potential of bobwhite by reducing the availability of vitamin A, a key nutrient for reproduction and survival (Nestler, 1946), as well as diverting resources from reproduction. As such, *A. pennula* may have the potential to impact bobwhite populations through reduced survival and fecundity, although additional research is needed to evaluate this potential impact at the population level.

4.6. Future work to address questions 6 and 7

While further studies are needed to determine if parasitic infection elicits other specific biological effects in bobwhite and whether they have a measurable impact at the population level, the WOE framework accounts for the well-known difficulty in determining these questions in a wild system. This allows us to move to questions 6 and 7 in the framework. Question 6 evaluates observed or specific effects that have been documented in the laboratory. Currently, laboratory experiments are underway, and the infection of bobwhite with *O. petrowi* in the laboratory has been completely worked out, from intermediate host to definitive host (Kalyanasundaram et al., 2019a). Challenge experiments have also been conducted, which determined that eyeworm and caecal worm glycoproteins elicit an immune response in bobwhite (Kalyanasundaram et al., 2019b), and *O. petrowi* can cause oxidative stress and mount an immune response (Hunter, 2016). Multiple studies are in development to better understand how parasites affect bobwhite including: studies to replicate the life cycle of *A. pennula* within the laboratory; studies to investigate the biological responses that *O. petrowi* and *A. pennula* may elicit in bobwhite; and experiments to assess impacts on the health and fitness of infected individuals.

Finally, in regards to question 7, which entails assessing the response of the bobwhite populations to the removal of parasites, field studies as those conducted by Hudson et al. (1998) on red grouse provide a model for future work. However, the empirical studies by Hudson (1986) and Hudson et al. (1992, 1998) investigating the potential of parasites to affect population dynamics of red grouse took over 20 years to complete and utilized a medicated grit. Studies such as these are currently hindered as there are currently no anthelmintics registered for use in wild bobwhite in the United States (Needham et al., 2007). Therefore, experimental manipulation studies must await registration of a treatment for wild bobwhite.

Nevertheless, a great deal of progress has been made to pave the way for future work investigating the interactions between bobwhite and parasites. Molecular techniques have been developed in order to non-lethally assess parasitic infection in bobwhite via a cloacal swab or feces sample that is evaluated by quantitative PCR (qPCR) and can detect the DNA for as little as one egg (Kistler et al., 2016b; Kalyanasundaram et al., 2018c). These methods were then adapted for use at a regional scale (Blanchard et al., 2018), as they are considered an effective form of parasite monitoring (Gray et al., 2012) that provides a valuable supplement to traditional methods (Archie et al., 2009). This will be beneficial for long term studies to non-lethally evaluate the effects of these parasites at the population level. Molecular techniques may also be useful in understanding the transmission dynamics of parasites, which are often influenced by climate (Harvell et al., 2002; Benton et al., 2015). For example, Blanchard et al. (2019) used qPCR and climatic variables to determine that temperature and precipitation could influence eyeworm and caecal worm egg shedding in bobwhite.

5. Conclusions

There is still much research needed to determine the full consequences of *O. petrowi* and *A. pennula* on bobwhite, particularly since the interactions of multiple parasites is understudied and not well understood (Pedersen and Fenton, 2007; Bordes and Morand, 2011). However, this WOE based approach reveals that parasites should be investigated further as they likely play a larger role in regulating bobwhite populations than previously thought. By using WOE, the pieces can begin to be assimilated and potential interactions may become evident (Fig. 3). This would be invaluable to studies of wildlife disease where it is impossible to control all factors and where disease effects can be abstruse (May, 1988; Woolf et al., 1993).

The proliferation and impacts of disease amidst widespread and rapid global change involve overarching, interrelated, and complex processes, which may test the bounds of traditional methods of inquiry (Plowright et al., 2008). In this ever-changing world, conventional strategies for quail management, as well as the management of other species, may be insufficient (Deem et al., 2001; Palmer et al., 2012), and crisis management is not suitable for conserving a species either (Plowright, 1988; Friend, 2014; Grant et al., 2017). Instead, multidisciplinary approaches are necessary and should be used when addressing disease and conservation of a species (Daszak et al., 2000; Deem et al., 2001; Plowright et al., 2008; Hoverman and Searle, 2016).

Thus, we suggest that WOE is a valuable tool for identifying potential instances of significant disease impacts. Although the WOE approach cannot answer all the questions of how a disease impacts a wild population, “disease management requires acting with imperfect information” (Grant et al., 2017), and WOE provides an effective means of

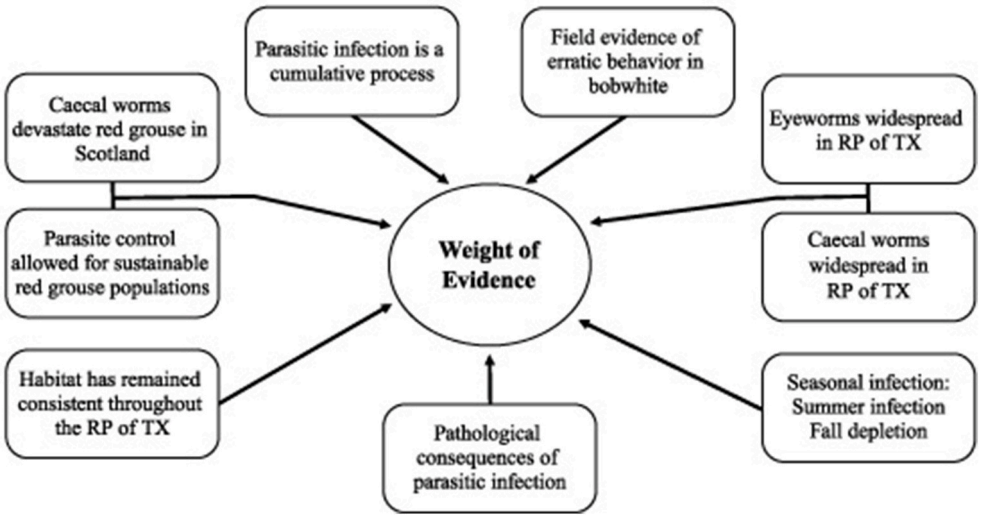

Fig. 3. Factors contributing to the weight of evidence that parasites negatively affect bobwhite.

investigating multiple lines of evidence in a systematic manner. However, WOE is not a replacement for empirical studies and should be used to identify whether it is worthwhile to move forward with empirical studies that are often logistically difficult in wild populations. Following identification of a problem using WOE, empirical study and an active adaptive management strategy could be adopted for more complete insight into the complex nature of wildlife systems. This will help achieve immediate management needs while gaining knowledge that is beneficial to developing robust long-term strategies (McDonald-Madden et al., 2010).

Declaration of competing interest

The authors declare no conflict of interest.

Acknowledgements

We are grateful to the Park Cities Quail Coalition, USA (24A125), Rolling Plains Quail Research Foundation, USA (23A582), and landowners for the continued support and funding that make this research possible. We also thank Wildlife Toxicology Laboratory members for their contribution to this work.

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