Parasites and infectious diseases of prairie grouse: should managers be concerned?

by Markus J. Peterson

Abstract Historically, interest in the infectious agents of prairie grouse (*Tympanuchus* spp.) (PG) mirrored trends in how North American wildlife scientists perceived host–parasite interactions. Increased ecological interest in host–parasite interactions since the 1980s led to increased awareness of PG–parasite interactions beginning in the 1990s. Prairie grouse are hosts to parasitic arthropods (e.g., lice, mites, ticks) and helminths (e.g., nematodes, cestodes, trematodes), as well as microparasites such as protozoa, bacteria, fungi, and viruses. Although many of these infectious agents cause disease in individual PG, few data address their potential influence on host population dynamics. Based on existing data on the parasites of PG, studies of other grouse species, and theoretical perspectives, the macroparasites *Dispharynx nasuta* and *Trichostrongylus cramae;* the microparasites *Eimeria dispersa*, *E. angusta*, *Leucocytozoon bonasae*, and *Plasmodium pedioecetii;* and the infectious bronchitis and reticuloendotheliosis viruses exhibit characteristics that suggest they have the potential to regulate PG populations. Infectious agents such as *Histomonas meleagridis*, *Pasteurella multocida*, *E. dispersa, E. angusta,* and other microparasites that cause high mortality across a broad range of galliform hosts have the potential to extirpate small, isolated PG populations. Nonparasitic diseases caused by mycotoxins, pesticides, and other toxic compounds also have the potential to influence population dynamics. Because there appears to be a behavioral component to PG population extinction, the fact that parasites might influence breeding behavior also requires further evaluation. Although it is difficult to establish whether parasites regulate their host populations, research models such as that associated with *T. tenuis* in red grouse (*Lagopus lagopus scoticus*) are available for reference. These approaches could be used to determine whether relevant macro- and microparasites influence the dynamics of declining or at-risk PG populations. Natural-resource policy-makers must become aware that macro- and microparasites of PG are not something they can safely ignore and should fund research designed to determine whether parasites regulate or have the potential to extirpate PG populations while there is still time for management intervention.

Key Words behavior, infectious disease, parasite, population dynamics, population regulation, prairie grouse, regulation, *Tympanuchus*

Author's address: Department of Wildlife and Fisheries Sciences, Texas A&M University, TAMU-2258, College Station, TX 77843, USA; e-mail: mpeterson@tamu.edu.

Prairie grouse (*Tympanuchus* spp.) abundance has declined across much of their range. For pinnated grouse particularly, population and subspecies extinction is not only a threat but also a reality. Despite the efforts of biologists and other interested citizens, the heath hen (HH; *T. cupido cupido*) was extinct by 1932 (Gross 1928, 1932). Similarly, Lehmann (1939, 1941) argued that unless multiple, large refuges were quickly established, extinction of the Attwater's prairie-chicken (APC; *T. c. attwateri*) could be expected. These refuges never were realized, and the APC might well now be the most endangered avian in the United States (United States Fish and Wildlife Service [USFWS] 1993; Peterson and Silvy 1994, 1996; Peterson et al. 1998*a*; Silvy et al. 1999). Similar reductions in abundance of greater prairie-chickens (GPC: *T. c. pinnatus*) also occurred. By 1984 this species had been extirpated from Alberta, Arkansas, Indiana, Iowa, Kentucky, Manitoba, Ohio, Ontario, Saskatchewan, Tennessee, and Texas (Schroeder and Robb 1993). Greater prairie-chicken populations now are at risk of extinction in Iowa (reintroduced 1987; Moe 1999), Illinois, and North Dakota, while numbers in Oklahoma decreased substantially during recent decades, resulting in only about 1,500 individuals in 1997 (Svedarsky et al. 2000). Similarly modest, yet stable, populations exist in Minnesota, Wisconsin, and Missouri. Fortunately, relatively robust GPC populations still occur in Colorado, Kansas (declining), Nebraska, and South Dakota. Lesser prairie-chicken (LPC; *T. pallidicinctus*) abundance declined dramatically from about 1880–1980 (Crawford 1980, Taylor and Guthery 1980). Although numbers increased somewhat during the mid-1980s, the 1990s again were characterized by declining abundance (Giesen 1998). For this reason, the FWS ruled in 1998 that listing the LPC as threatened under the Endangered Species Act of 1973 was warranted but precluded by higher listing priorities (50 CFR 17). Although the sharp-tailed grouse (STG; *T. phasianellus*), as a species, is certainly at less risk of extinction than the pinnated grouse, abundance has declined substantially in the southern and eastern portions of its range (Connelly et al. 1998). This species was extirpated from California, Illinois, Iowa, Kansas, Nevada, New Mexico, Oklahoma, and Oregon by 1969 and remains at risk in British Columbia, Colorado, Idaho, Oregon, Utah, and Washington.

Natural-resource policy-makers must become aware that macro- and microparasites of wildlife are not something they can safely ignore.

Many PG populations are at risk of extinction or exhibit long-term declining abundance, so we must better understand factors that might account for this situation. Most biologists assume that habitat loss and conversion ultimately were responsible for these trends (e.g., Schroeder and Robb 1993, Connelly et al. 1998, Giesen 1998). However, infectious agents might contribute to declining abundance or even extinction of insular populations (Cleaveland et al. 2002, Tompkins et al. 2002). For example, after admitting that the role of infectious agents in APC populations was unclear, the authors of the recovery plan (USFWS 1993) argued that these agents must still be considered potential limiting factors. Similarly, Mote et al. (1999), in their range-wide conservation assessment for the LPC, could not determine whether infectious agents regulated LPC populations but suggested that those transmitted independently from host density could have "drastic effects." Although Tirhi (1995), in the Washington State plan for managing Columbian STG (*T. p. columbianus*), did not think infectious agents typically caused significant mortality, she argued that they could limit STG populations subjected to adverse weather conditions or energy limitations. Whereas wildlife managers view infectious agents as potential problems for PG populations, they are less clear regarding which infectious agents might be most important, the role such agents play in host population dynamics, or how one might mitigate for their effects.

Here I review what is known regarding infectious agents of *Tympanuchus* spp., and then discuss what this information might mean to wildlife policy-makers, managers, and researchers. Specifically, I 1) contextualize research addressing the infectious agents of PG within wildlife science's broader perspectives toward host–parasite interactions, 2) summarize the literature regarding the macro- and microparasites of PG, 3) delineate specific agents most likely to regulate PG populations or extirpate small, isolated populations, and 4) discuss research and management implications. During this process I identify gaps in our knowledge regarding PG–parasite interactions.

Historical perspective

Awakening interest

In the mammoth *The Grouse in Health and Disease* (Committee of Inquiry on Grouse Disease 1911), biologists argued that *Trichostrongylus tenuis* (=*T. pergracilis*) was the primary cause of "Grouse Disease" in red grouse (*Lagopus lagopus scoticus*) in the British Isles. There is

little doubt that this publication stimulated North American grouse researchers not only to attempt similar studies (e.g., Bump et al. 1947) but also to search for their own version of "the Grouse Disease" (Gross 1925*a*:424, Lack 1954:164). For example, a flurry of surveys addressing the infectious agents of ruffed grouse (*Bonasa umbellus*) soon was completed, with *Dispharynx* sp. and *Leucocytozoon bonasae* considered likely candidates (Gross 1925*a*, *b*; Allen and Gross 1926; Levine 1932; Clarke 1935*a*, *b*, 1936, 1938; Fisher 1939). Similarly, by the early 1930s, surveys of the infectious agents of PG also had been completed (Gross 1928, 1929, 1930 1931; Green and Shillinger 1932).

Leopold (1933:325), in his influential *Game Management*, probably increased interest in infectious agents of wildlife by arguing that "the role of disease in wild-life conservation has probably been radically underestimated." He also maintained that "density fluctuations, such as cycles and irruptions, are almost certainly due to fluctuations in the prevalence of, virulence of, or resistance to [infectious] diseases." Thus Leopold placed host–parasite interactions on par with other important interspecific relationships, such as predator–prey interactions. He did not, however, offer any empirical or experimental evidence to support his suppositions.

Several relatively comprehensive surveys of the parasitic helminths of GPCs and STG were completed during this period of keen interest in the infectious agents of wildlife (Gross 1930, Boughton 1937, Leigh 1940, Morgan and Hamerstrom 1941, Schwartz 1945). These studies serve as a useful point of comparison for more modern surveys. Unfortunately, only a few comprehensive studies addressed the microparasites of PG (Gross 1930), while most reports were of an anecdotal nature (e.g., Green and Shillinger 1932, Saunders 1935, Baumgartner 1939).

Parasites as a byproduct of habitat

By about 1950, North American wildlife scientists began to assume that infectious agents of free-roaming wildlife were ecologically unimportant, except as almost inanimate extensions of poor habitat conditions or as natural disasters (Trippensee 1948, Lack 1954, Taylor 1956). In his *Wildlife Management*, Gabrielson (1951) did not even mention wildlife diseases or parasitism, suggesting that he felt infectious agents of wildlife were inconsequential. Not surprisingly, only sporadic efforts were made to evaluate PG–parasite interactions from an ecological perspective until the 1990s. In a special issue of *The Journal of Wildlife Management* addressing grouse, Herman (1963) generally echoed the assumption that infectious agents influenced grouse population dynamics only when habitat conditions were substandard. He pointed out, however, that few studies had been conducted in such a manner that the population-level effects of infectious agents could be documented even if they occurred. Unfortunately, this criticism still holds (Peterson 1996, Tompkins et al. 2002). At any rate, perceiving bacterial or viral diseases as natural disasters where management could not reasonably be brought to bear (e.g., much like hurricanes or volcanic eruptions) led wildlife scientists to neglect these important interspecific relationships (Peterson 1991*a*).

Conversely, since the early part of the twentieth century, those interested in parasite systematics continued to study their favorite taxa in wild hosts, including PG. These efforts tended to emphasize host lists and parasite descriptions. For example, several surveys addressing the hematozoa of PG were published during the 1970s (e.g., Stabler et al. 1974, Stabler and Kitzmiller 1976, Stabler 1978, White and Bennett 1979). Such reports often were catalogued under key words related to specific, often now obsolete, parasite taxonomic names, rather than terms transparent to wildlife scientists (e.g., "disease" or "parasite"). This renders comprehensive literature searches difficult for many wildlife scientists. European grouse researchers, in contrast to their North American colleagues, were not so quick to abandon ecological studies of the infectious agents in grouse. Their reports, while not always published in English, are useful resources.

An ecological perspective

Anderson and May (1978) and May and Anderson (1978), in a 2-part article, provided a theoretical framework for evaluating host–parasite interactions from an ecological perspective. They demonstrated that parasites could, under certain circumstances, not only affect the health of individual animals but also regulate host populations. Probably in part because of May's stature as a leading theoretical ecologist of this period, studying host–parasite ecology in wild populations suddenly was again orthodox, and numerous theoretical and a few applied publications grounded in these ideas soon followed. These included the well-known studies demonstrating that *T. tenuis* can regulate red grouse populations under certain circumstances (e.g., Hudson 1986; Dobson and Hudson 1992; Hudson et al. 1992*a*, *b*, 1998). This renewed interest in the relationship between parasites and wild hosts eventually spread to North America, resulting in studies of the infectious agents of PG.

From an ecological perspective, parasites are organisms that meet the following 3 conditions: use of their hosts as habitat, nutritional dependence on the host, and causing "harm" to the host during some point in their life

cycle (Anderson and May 1978). Anderson and May (1979) also offered an ecologically based categorization of parasites that is directly relevant to those concerned with wildlife conservation. Macroparasites (parasitic arthropods and helminths) tend to have longer generation times than microparasites, direct multiplication in or on the host is either absent or occurs at a low rate, and the immune response elicited by these metazoans depends on the number present and typically is of short duration. For these reasons, macroparasites generally occur as endemic host infections that are more likely to cause morbidity than mortality. Conversely, microparasites (e.g., protozoans, fungi, bacteria, viruses) are characterized by small size, short generation times, high rates of direct reproduction within the host, and a tendency to induce long-lasting immunity to reinfection. Microparasitic infection typically is short relative to the expected lifespan of the host. For this reason, microparasitic diseases often occur as epidemics where the pathogen apparently disappears as susceptive hosts die or become immune, only to reappear when sufficient densities of susceptible hosts are again available in the population. Parasites can complete their life cycles either 1) directly by contact between hosts, inhalation, ingestion, or skin penetration or 2) indirectly via biting vectors, penetration by free-living larva produced in an intermediate host, or by the host ingesting an intermediate host. One of the primary approaches to controlling infectious diseases in wildlife is to interrupt parasite life cycles, so understanding how these agents are transmitted is important to wildlife managers.

Host population regulation. For a parasite to be regulatory, it must cause the abundance of hosts in the population to decrease when it is above a particular level, but allow it to increase when it is below that level. Such parasites could have serious consequences for small, isolated populations (Scott 1988, Peterson 1996). For example, if pathogens suppress the size or resilience of an endangered population, they concomitantly increase the probability of extinction due to other biotic or abiotic factors (Holmes 1996, Peterson et al. 1998*b*, Cleaveland et al. 2002).

Theoretically, macroparasites can regulate host populations when the per-capita production of infectious stages is greater than the weighted growth rate of the host population (Tompkins et al. 2002). Regulation is more likely when a large proportion of the macroparasite population is aggregated in a small proportion of a host population. Because most macroparasitic species exhibit aggregated distributions (Anderson and May 1978, Shaw and Dobson 1995, Shaw et al. 1998), it is reasonable to expect that certain of these parasites might regulate their host populations, particularly when the parasite limits host fecundity (May and Anderson 1978, Tompkins et al. 2002). Macroparasites typically are more likely to regulate host populations than microparasites because the regulatory effects of reduced host fecundity tend to be greater than those of increased mortality (Tompkins et al. 2002). Macroparasites also are predicted to induce host population cycles when parasite-induced reductions in fecundity are large relative to parasite-induced mortality (Dobson and Hudson 1992).

Directly transmitted viral or bacterial microparasites have the potential to regulate their host populations if the per-capita influence of parasite-induced mortality or reduced fecundity exceeds the intrinsic growth rate of the host population, weighted by the period that a host remains infectious or immune (May 1983, Tompkins et al. 2002). Acquired immunity, by decreasing the period of infectivity, renders host population regulation less likely, while long latency periods or decreased host fecundity can lead to periodic cycles in host populations (Anderson et al. 1981, Tompkins et al. 2002). If other biotic or abiotic factors decrease the growth rate of the host population, the parasite still could regulate the host population even if only a few individuals were killed.

Although several models of indirectly transmitted microparasites have been developed (e.g., May and Anderson 1979, Hudson et al. 1995, Randolph et al. 2002), most modeling efforts concentrated on how these agents persist, seasonal dynamics, reservoir hosts, and sinks rather than whether they regulate host populations. It is probable that microparasites transmitted by ubiquitous insects have a potential for regulating host populations similar to that of directly transmitted agents. Longer-lived intermediate hosts, such as ticks or helminthic endoparasites, complicate the picture. This is particularly true for vectors with cosmopolitan host ranges. Regardless of whether these indirectly transmitted microparasites regulate host populations, they certainly have the potential to periodically constrain host abundance. One of the difficulties associated with studying regulation of wild host populations by parasites is that population regulation in general is much easier to explain theoretically than to demonstrate experimentally.

Host population extirpation. Parasites, regardless of whether they regulate their host populations, still might influence host population dynamics. For example, because certain microparasitic epizootics are characterized by high host mortality, thus markedly reducing the number of birds in local or regional areas, they by definition alter host population dynamics. Well-documented examples include avian cholera in waterfowl and other waterbirds (Moore and Simpson 1981, Botzler 1991, Friend 1999*a*, Friend et al. 2001), conjunctivitis in house

finches (*Carpodacus mecicanus*; Nolan et al. 1998, Hochachka and Dhondt 2000), and Newcastle disease in double-crested cormorants (*Phalacrocorax auritus*; Wobeser et al. 1993, Meteyer et al. 1997, Docherty and Friend 1999). While such epizootics undoubtedly can decimate local abundance, in ecologically robust host populations numbers soon rebound due to reproduction and immigration. Conversely, many small, isolated host populations already have difficulty maintaining numbers, and immigration is not possible. In these situations microparasitic epizootics, particularly, could lead directly to local extinction (Thorne and Williams 1988, Peterson et al. 1998*b*, Cleaveland 2002). Clearly, epizootic diseases characterized by high mortality and broad host ranges pose a significant threat to small, isolated populations of PG.

Infectious agents of prairie grouse

My primary objective for this section is to review what is known regarding the infectious agents or PG. Where possible and appropriate, I address this goal by briefly discussing parasite range extent, prevalence, intensity, seasonality, pathogenicity, and whether previous research suggests the parasite might influence host population dynamics. It is beyond the scope of this essay to detail pathogenesis, clinical signs, lesions, and diagnostic techniques for every agent discussed. I provide numerous citations, however, that detail this information for interested readers. Additionally, although most reference books addressing the infectious diseases of wild and domestic birds cover tetraonids only superficially (e.g., Calnek et al. 1997, Davidson and Nettles 1997, Friend and Franson 1999*a*), they do include useful general treatments of most infectious agents discussed below.

Unfortunately, during the century biologists have studied these parasites, the specific if not the generic names of many agents have changed at least once. Further, there is no single source of currently approved names for all parasite taxa. This renders the binomial nomenclature associated with parasites murky at best. I used names agreed upon by current consensus where they could be verified and include the synonyms used in cited sources in parentheses. Otherwise, I followed the nomenclature used by the cited authority.

Macroparasites

Parasitic arthropods. Mallophaga of 5 genera and ≥7 species have been described for PG (Table 1). *Goniodes* spp. were most frequently observed, found over the largest geographic extent, and associated with all races of PG. It appears that *G. cupido* is restricted to pinnated grouse, while *G. nebraskensis* is a parasite of STG (Table 1). Dick (1981), studying STG in Manitoba, found high prevalences of *Amyrsidea* sp., *G. nebraskensis*, and *Lagopoecus gibsoni* for birds collected April–October, 1974–1976 (Table 1). Intensities of these infestations varied seasonally, with *Amyrsidea* sp. and *L. gibsoni* peaks in May (April–October $\bar{x}$=4.4 and 1.7; range=1–8 and 1–22 per bird, respectively), and peak *G. nebraskensis* intensity in June (peak $\bar{x}$>60 per grouse, April–October $\bar{x}$=17.7; range=1–135). Although most authors did not list parasite intensity, relatively high prevalence of lice was found (Table 1). Similarly, while Gross (1930:37) did not tabulate prevalence, he noted, "bird lice were found on a large percent[age]...of the prairie chickens and sharp-tailed grouse examined." Apparently, one should expect to find Mallophaga of ≥1 species associated with PG populations.

Only 3 ectoparasite surveys of PG found mites, with low prevalences reported for the 2 studies where such data were presented (Table 1). Because of the methods used by many ectoparasite surveys, however, mites might not have been detected even if they were present. It is reasonable to assume that feather mites probably occur at some level in most PG populations. Ticks of 2 species have been found on PG (Table 1) and were common during the warmer months (Gross 1930, Dick 1981). Dick (1981) found that intensity varied by season, with peaks in July and August for *Haemaphysalis chordeilis* and *H. leporispalustris* (=*H. leporis-palustris*), respectively (peak $\bar{x}$>50 per grouse; April–October $\bar{x}$=18.8 and 18.7; range=4–180 and 9–225, respectively). He also found the hippoboscid fly *Ornithoyia anchineuria* on Manitoba STG (Table 1), but intensities were low (April–October range=1–2).

During the first half of the twentieth century, biologists studying PG often maintained that ectoparasites could be detrimental to hosts, particularly chicks, incubating hens, or anytime parasite intensity was high (e.g., Gross 1930, Leigh 1940). Later it was more typical to assume that parasitic arthropods were important to PG only when habitat conditions led to nutritional or other "stresses" for hosts (e.g., Hillman and Jackson 1973:28–30, Tirhi 1995). Dick (1981:235), however, in his analysis of the ectoparasites of a STG population, argued that "the role of ectoparasitism on mortality and population fluctuations...is far from clear." More recently, ecologists discovered that ectoparasites could be important mediators of host behavior, thus influencing host populations (Dobson 1988). For example, in other arena species, females differentially selected males with fewer ectoparasites (Borgia and Collis 1990, Boyce 1990, Johnson and Boyce 1991). Conversely, in their study of

Table 1. Mallophaga, mites, ticks, and hippoboscid flies found on heath hens (HH), sharp-tailed grouse (STG), and Attwater's (APC), greater (GPC), and lesser prairie chickens (LPC) by state or province. Prevalence (%; *n* positive/*n* examined) was included where data were available.

Order or superfamily *Genus species*	Prairie grouse	State or province	Prevalence	Reference
Mallophaga				
Amyrsidea sp.	STG	Manitoba	21.1 (46/218)	Dick 1981[a]
	STG	Wisconsin		Emerson 1951
A. perdicis	STG	South Dakota	23.3 (14/60)	Boddicker and Hugghins 1965[b]
Chapinia sp.	GPC	Wisconsin		Gross 1930
Goniodes sp.	GPC	North Dakota		Aldous 1943
	STG	Ontario		Tsuji et al. 2001
	STG	Wisconsin		Gross 1930
G. cupido	GPC	Unknown		Osborn 1896, Kellogg 1899
	GPC	Nebraska		Emerson 1951[c]
	GPC	Oklahoma		Emerson 1951
	HH	Massachusetts		Giebel 1866, 1874
	HH	Massachusetts	100 (9/9)	Gross 1928
	LPC	Oklahoma		Emerson 1951
G. nebraskensis	STG	Manitoba		Emerson 1951
	STG	Manitoba	94.0 (201/218)	Dick 1981[a]
	STG	Montana		Emerson 1951
	STG	Nebraska		Emerson 1951
	STG	North Dakota		Emerson 1951
	STG	Ontario		Emerson 1951
	STG	South Dakota	55.0 (33/60)	Boddicker and Hugghins 1965[b]
Lagopoecus sp.	GPC	Oklahoma		Emerson 1951
	LPC	Oklahoma		Emerson 1951
L. gibsoni	STG	Manitoba	56.0 (122/218)	Dick 1981[a]
L. perplexus	GPC	Missouri	31.8 (7/22)	Schwartz 1945
	STG	Ontario		Emerson 1951
	STG	South Dakota	3.3 (2/60)	Boddicker and Hugghins 1965[b]
	STG	Washington		Kellogg 1899, Emerson 1951
Menopon sp.	GPC	Missouri	9.1 (2/22)	Schwartz 1945
M. monostaechum	GPC	Illinois	14.3 (4/28)	Leigh 1940
Mites				
Ornithonyssus sylviarum	STG	Manitoba	6.9 (15/218)	Dick 1981[a]
Tetraolichus cupido	APC	Texas		Atyeo and Gaud 1992
Unidentified	GPC	Illinois	7.1 (2/28)	Leigh 1940
	STG	South Dakota	1.7 (1/60)	Boddicker and Hugghins 1965[b]
Ticks				
Haemaphysalis sp.	STG	Minnesota		Green and Shillinger 1932
H. chordeilis	STG	Manitoba	95.0 (207/218)	Dick 1981[a]
	STG	South Dakota	3.3 (2/60)	Boddicker and Hugghins 1965[b]
H. leporispalustris	GPC	Wisconsin		Gross 1930
	STG	Manitoba	95.9 (209/218)	Dick 1981[a]
	STG	Michigan		Baumgartner 1939
	STG	South Dakota	5.0 (3/60)	Boddicker and Hugghins 1965[b]
	STG	Wisconsin		Gross 1930
Hippoboscid fly				
Ornithoyia anchineuria	STG	Manitoba	16.1 (35/218)	Dick 1981[a]

[a] Collected April–October. [b] Collected October–June. [c] Also listed for GPC from unspecified Canadian Province(s).

STG, Tsuji et al. (2001) found that males possessing central territories, and presumably doing most of the breeding, actually had more lice than those on the periphery. They also argued that because most males were only lightly infested, discriminating females would garner little benefit as far as exposure to ectoparasites was concerned. At any rate, although ectoparasites are commonly found on PG, their population-level significance requires further clarification.

Nematodes. Two nematode species, *Cyrenia colini* (=*Seurocyrnea colini*) and *Heterakis gallinarum* (=*H. gallinae*, *H. pedioecetes*; Inglis 1957), were found by most studies of the endoparasites of PG, typically at high prevalences (Table 2). High *Trichostrongylus cramae* and *Tetrameras* sp. prevalence was observed in Attwater's and LPC populations, respectively, the only PG where these parasites were described. The proventriculus and cecum were more likely to be parasitized than other internal organs, with proventricular and cecal nematodes found for 10 of 12 and 11 of 12 study locations, respectively (Table 2).

Ascaridia galli (=*A. lineata*), *Capillaria contorta*, *H. gallinarum*, *H. isolonche*, and *T. cramae* all have direct life cycles. The intestinal nematode *A. galli* and the crop worm *C. contorta* were found in both GPCs and STG taken in the northern midwestern states (Table 2). Although it typically is assumed that *A. galli* is of low pathogenicity except in chicks (e.g., Gross 1930, Boughton 1937, Morgan and Hamerstrom 1941), large numbers of *C. contorta* can cause severe illness, particularly in captivity (Ruff and Norton 1997). Cecal threadworms, either *H. gallinarum* or *H. isolonche*, were found during most helminthic surveys of PG (Table 2; Mawson 1956, Hillman and Jackson 1973). Most authors assumed the

Table 2. Prevalence of enteric nematodes found for Attwater's (APC), greater (GPC), and lesser prairie chickens (LPC) and sharp-tailed grouse (STG) by state, study, and number examined (*n* ex.) in North America.

Prairie grouse	State	*n* ex.	*Ascaridia Galli*		*Capillaria contorta*		*Cheilospirura spinosa*		*Cyrenia colini*		*Dispharynx nasuta*		Heterakis *gallinarum*		*Subulura strongylina*		Other		Reference[a]
			n	%	*n*	%	*n*	%	*n*	%	*n*	%	*n*	%	*n*	%	*n*	%	
APC	Tex.	3, 9[b]									1	33.3					8[c]	88.9	**9**
GPC	Ill.	28							14	50.0			11	39.3					**3**
GPC	Kans.	106							82	77.4	14	13.2	26	24.5					**7**
GPC	Mo.	11							5	45.5			4	36.4					**5**
GPC	Wis.	34	[d]						3	8.8			17	50.0					**1**
GPC	Wis.	39	2	5.1	6	22.5[e]			13	33.3 [f]			8	20.5					**4**
LPC	Kans.	91, 88[g]													54[h]	59.3	81[i]	92.0	**10**
LPC	Tex.	41											21[j]	51.2					**8**
STG	Minn.	53	5	9.4			2	3.8											**2**
STG	S.D.	6					1	16.7	2	33.3			1	16.6	3	50.0			**2**
STG	S.D.	60							37	61.7	3	5.0			31	51.7			**6**
STG	Wis.	62	12	19.4	12	9.5[e]	3	4.8	39	62.9[f]			19	30.6	3	4.8			**4**
Total		531	19	3.6	18	3.0	6	1.1	195	36.7	18	3.4	107	24.2	91	17.0	89	16.5	

[a] **1**, Gross 1930; **2**, Boughton 1937; **3**, Leigh 1940; **4**, Morgan and Hamerstrom 1941; **5**, Schwartz 1945, **6**, Boddicker and Hugghins 1965; **7**, Harper et al. 1967; **8**, Pence and Sell 1979, Pence et al. 1983; **9**, Peterson et al. 1998*b*; **10**, Robel et al. 2003.

[b] Samples collected opportunistically; 3 could be evaluated for *D. nasuta* and most other helminths and 9 for *Trichostrongylus cramae* (reflected in totals).

[c] *Trichostrongylus cramae*.

[d] Listed as the "commonest" parasite found (no numbers were given, so not reflected in totals).

[e] A total of 52 greater prairie chickens and 126 sharp-tailed grouse were examined for *C. contorta* (reflected in totals).

[f] The authors noted that the total number examined was somewhat less than that listed, so the percentage positive might be elevated.

[g] 91 and 88 individuals could be evaluated for *Subulura* sp. (and other cecal nematodes) and *Tetrameras* sp. and most other helminths, respectively (reflected in totals).

[h] Not keyed to species. Most closely resembled *S. suctoria*.

[i] *Tetrameras* sp. Because only female parasites were recovered, these parasites could not be keyed to species. Probably *T. pattersoni* or *T. americana*.

[j] *H. isolonche*.

primary significance of *H. gallinarum* was that it can transmit the protozoan *Histomonas meleagridis*, the etiologic agent of histomoniasis or blackhead disease (see Other protozoa, below). Although *H. isolonche* causes significant disease in ring-necked pheasants (*Phasianus colchicus*), few pathologic changes were noted in grouse even with high parasite intensities (Ruff and Norton 1997). Pence et al. (1983) found no significant differences, spring to fall, in *H. isolonche* prevalence (10 of 15 and 11 of 26, respectively) or intensity ($\bar{x}$=17.5 and 66.5; range=1–15 and 1–271, respectively).

Durette-Desset et al. (1993) and Freehling and Moore (1993) revised the name of the commonly observed cecal worm (*Trichostrongylus* sp.) of northern bobwhites (*Colinus virginianus*) from *T. tenuis* to *T. cramae*. After comparing the species of *Trichostrongylus* found in APCs with *Trichostrongylus* spp. from a variety of wild hosts, Peterson et al. (1998*b*) determined it to be *T. cramae* as well. Freehling and Moore (1993) thought *T. cramae* was not particularly pathogenic for northern bobwhites, while Davidson et al. (1982) found no evidence that it limited or regulated bobwhite populations. Conversely, in red grouse, *T. tenuis* causes disease in individuals and can regulate host populations (Shipley 1911; Wilson and Leslie 1911; Hudson and Dobson 1991; Hudson et al. 1992*a*, *b*, 1998). *Trichostrongylus cramae* intensity in APCs ($\bar{x}$=1,019.3; Range=3–1,906; *N*=3) appears more similar to that seen for *T. tenuis* in red grouse in northern England and Scotland (Hudson 1986, Shaw and Moss 1989, Hudson et al. 1992*a*), than to *T. cramae* in northern bobwhites in the southeastern United States (Forrester et al. 1984, Moore et al. 1986, Davidson et al. 1991, Purvis et al. 1998). Because cecal length of *Tympanuchus* spp. and *Lagopus* spp. (Tetraoninae) is similar (Leopold 1953), and much greater than that of the more distantly related northern bobwhite, comparisons of *Trichostrongylus* spp. intensities between red grouse and APCs are not unreasonable (Peterson et al. 1998*b*).

The life cycles of *Cheilospirura spinosa, C. colini*, *Tetrameras* sp., *Subulura strongylina*, *Dispharynx nasuta* (=*D. spiralis*), and *Gongylonema phasianella* are indirect (Table 2, Wehr 1938). The intermediate hosts for the gizzard nematode *C. spinosa* and proventricular *C. colini* and *Tetrameras* sp. are grasshoppers (*Melanoplus* sp.)

and cockroaches (*Blattella germanica*; Cram 1931, 1933). Robel et al. (2003) could not determine the species of *Tetrameras* recovered from the LPCs they examined from southwestern Kansas because only female parasites were recovered, but narrowed their identification to either *T. americana* or *T. pattersoni*. They found no differences in prevalence by host age or sex (Table 2), although slightly higher prevalences were observed during spring and fall as compared to winter (12 of 12, 11 of 11, and 54 of 61, respectively). Mean intensity for all individuals was 21 (range=1–66), with juveniles ($\bar{x}$=24, range = 3–66) harboring slightly more parasites than adults ($\bar{x}$=17, range=1–59). The exact life cycle of the cecal nematode *S. strongylina* has yet to be worked out (Ruff and Norton 1997). Robel et al. (2003) maintained that the *Subulura* species they found in LPCs most closely resembled *S. suctoria*, but could also be a new species. They found no differences in prevalence (Table 1) or intensity ($\bar{x}$=28, range=1–319) by host age or sex, but prevalence was lower during spring than fall or winter (2 of 11, 8 of 11, and 41 of 65, respectively). Typically, *C. spinosa*, *C. colini*, and *S. strongylina* are thought to cause few pathologic changes in hosts (Leigh 1940, Harper et al. 1967, Ruff and Norton 1997), although *C. spinosa* can cause disease and mortality when intensities are high for extended periods (Cram 1930, 1931; Ruff and Norton 1997). The pathogenicity of *T. americana* or *T. pattersoni* in PG is unknown, but Robel et al. (2003) found that *Tetrameras* sp. intensity was not related to LPC movements, reproductive productivity, or survival. While *T. americana* causes little pathologic change in northern bobwhites, it can cause severe disease in domestic chickens under certain husbandry regimes (Cram et al. 1931, Ruff and Norton 1997). Conversely, *T. pattersoni* is somewhat more pathogenic for northern bobwhites than is *T. americana* (Ruff and Norton 1997).

The proventricular nematode *D. nasuta* uses the sowbug (*Porcellio scaber*) or pillbug (*Armadillidium vulgare*) as intermediate hosts (Cram 1931). Severe damage to the proventriculus and death occur in ruffed grouse, leading many to conclude that *D. nasuta* is "the chief cause of 'grouse disease' in the northeastern United States" (Ruff and Norton 1997:821–822). The pathogenicity of *D. nasuta* in PG is less clear. Gross (1928) maintained that *Dispharynx* sp. might have been a factor in the demise of the HH, while Harper et al. (1967) did not consider *D. nasuta* detrimental to GPCs collected in November. However, because *D. nasuta* primarily causes disease only in young birds, one would not expect to see significant pathologic changes in otherwise healthy prairie chickens collected during November even if this parasite was as pathogenic for PG as it is for ruffed grouse.

Although I found only one report of *G. phasianella* identified from the crop of STG from Nebraska (Wehr 1938), Barre (1980) still considered this species valid in his key to the avian *Gongylonema*. This parasite probably has an arthropod intermediate host. Whether *G. phasianella* is pathogenic for individual PG, or has population-level significance, is unknown.

Saunders (1935) and Cram (1937) first reported finding the eye worm (*Oxyspirura petrowi*; =*O. lumsdeni*) under the nictitating membrane of 42 of 129 GPCs and STG in Michigan. Boddicker and Hugghins (1965) found this parasite in 19 of 60 STG from South Dakota, while Addison and Anderson (1969) identified it for STG, GPCs, LPCs, and STG-GPC hybrids collected from various portions of their range in both Canada and the United States. Pence and Sell (1979) and Pence et al. (1983) found *O. petrowi* in 25 of 41 LPCs from the Texas panhandle, with no significant differences in spring to fall prevalence (8 of 15 and 17 of 26, respectively) or intensity ($\bar{x}$=3.8 and 5.4; range=1–19 and 1–12, respectively). Robel et al. (2003) found *O. petrowi* in 53 of 56 LPCs from southwestern Kansas, with no differences in spring, fall, or winter prevalences (12 of 13, 10 of 10, and 30 of 32, respectively). They did, however, find slightly higher prevalence in adult as compared to juvenile hosts (100 and 91%; n=30 and 23, respectively), yet higher intensities for juveniles than adults ($\bar{x}$=21 and 9; range=1–81 and 2–32, respectively). No differences were found in prevalence or intensity by host sex. Because this parasite often is overlooked during routine examination (Cram 1937, Pence 1972), it might well occur in most PG populations. Although the life history of *O. petrowi* has yet to be detailed, it is assumed to have an arthropod, probably insect, intermediate host (Pence 1972). Saunders (1935:343) observed ocular irritation in several parasitized STG and GPCs, leading him to conclude that "serious consequences," such as decreased foraging efficiency and increased predation, were associated with high *O. petrowi* intensities. Conversely, Pence (1972) found that even intensities as high as 30 worms per eye in an array of avian hosts caused no gross or histopathologic changes. Similarly, Robel et al. (2003) found that *O. petrowi* intensity was not related with LPC movements, productivity, or survival. Definitive experiments designed to determine the significance of *O. petrowi* to PG populations have yet to be completed.

Filarial nematodes have been less commonly reported in PG. Gibson (1967) found *Splendidofilaria pectoralis* under the pectoral skin of 2 of 7 and 7 of 8 STG collected in British Columbia and southeastern Alaska, respectively. Possible dipterid vectors include black flies (*Simulium* spp.) and biting midges (*Culicoides* spp.).

Although these filarioids caused considerable localized tissue damage, Gibson (1967:1143) found no evidence they were "seriously detrimental" to hosts. Boughton (1937) and Boddicker (Hillman and Jackson 1973) found *Physaloptera* sp. larvae near the surface of the breast, leg, and wing musculature of STG from Minnesota and South Dakota, respectively. Pence et al. (1983) found *Physaloptera* sp. larvae in the crop or proventriculus of 16 of 41 LPCs collected in Texas, with higher parasite prevalence and intensity during spring than fall. Prairie grouse presumably acquire larval *Physaloptera* sp. by ingesting infected arthropods (Pence et al. 1983). Lastly, microfilaria were observed on 3 of 41 and 1 of 8 blood films taken from STG sampled in Wisconsin (Flakas 1952) and Colorado (Stabler et al. 1974), respectively. The population-level significance, if any, of these parasites is unknown.

Cestodes. Cestodes commonly were encountered during surveys of the parasitic helminths of PG (Table 3); prevalences typically were low. *Choanotaenia infundibulum*, *Rhabdometra nullicollis*, and *Raillietina variabilis* (=*R. variabila*) were identified for both pinnated and STG across much of their ranges (Table 3; Ransom 1909, Hillman and Jackson 1973). *Rhabdometra odiosa* was identified for STG in Quebec (Swales 1934) and LPCs in Texas (Pence and Sell 1979, Pence et al. 1983), while *Raillietina centrocerci* was recorded only for STG captured in South Dakota (Hillman and Jackson 1973). Because avian tapeworms use arthropod or isopod intermediate hosts (Pence et al. 1983, Reid and McDougald 1997), prevalence and intensity might be expected to vary by season. Pence et al. (1983) found higher *R. odiosa* prevalence (14 of 26) and intensity ($\bar{x}$=9.4, range=1–29) in LPCs captured during the fall than spring (1 of 15; 1 scolex). Arthropods (e.g., grasshoppers, crickets, beetles) made up 8, 27, 60, and 65% of the diet of LPCs on their study area during winter, spring, summer and fall, respectively, thus offering a reasonable explanation for differential *R. odiosa* prevalence and intensity observed by season. Further, because arthropods and isopods constitute a larger proportion of the diet in young birds, they tend to have higher cestode prevalence and intensity than adults (Leigh 1940, Yeatter 1943, 1963; Harper et al. 1967).

Most authors who described cestodes in PG did not observe gross pathologic changes attributable to these parasites, but some maintained that certain cestodes could be pathogenic, particularly in young birds. For example, Harper et al. (1967) noted inflammation where tapeworm scoleces were attached. Leigh (1940, 1941) found that sufficiently intense *R. variabilis* infections in young birds occluded the intestinal lumen; he maintained that this could reduce host vitality and render these young birds more susceptive to predation or microparasitic infection. Prairie grouse biologists periodically echoed this supposition (e.g., Yeatter 1943, 1963; Harper et al. 1967). At any rate, possible direct and indirect effects of cestodes on PG populations have yet to be explored.

Table 3. Prevalence of cestodes found for Attwater's (APC), greater (GPC), and lesser prairie chickens (LPC) and sharp-tailed grouse (STG) by state, study, and number examined (*n* ex.) in North America.

Prairie grouse	State	*n* ex.	*Choanotaenia infundibulum*		*Raillietina centrocerci*		*Raillietina variabilis*		*Rhabdometra nullicollis*		*Rhabdometra odiosa*		Other		References
			n	%	*n*	%	*n*	%	*n*	%	*n*	%	*n*	%	
GPC	Ill.	28	2[a]	7.1			10	35.7							Leigh 1940, 1941
GPC	Kans.	106											3[b]	2.8	Harper et al. 1967
GPC	Mo.	11											1	9.1	Schwartz 1945
GPC	Wis.	34					1	2.9							Gross 1930, Leigh 1941
GPC	Wis.	39	2	5.1					5	12.8					Morgan and Hamerstrom 1941
LPC	Tex.	41									15	36.6			Pence et al. 1983
STG	Minn.	53											14[c]	26.4	Boughton 1937
STG	N.D.	34					1[d]	2.9	3	8.8					Aldous 1943
STG	S.D.	60			37	61.7			6	10.0					Boddicker and Hugghins 1965
STG	Wis.	28					1[e]	3.6	1	3.6					Gross 1930
STG	Wis.	62	11	17.7					9	14.5					Morgan and Hamerstrom 1941
Total		496	15	3.0	37	7.5	13	2.6	24	4.8	15	3.0	18	3.6	

[a] Leigh (1940) keyed to genus only. Almost certainly *C. infundibulum*.
[b] Either *Choanotaenia* sp. or *Raillietina* sp.
[c] Predominately *C. infundibulum*; *R. nullicollis* was the only other cestode found.
[d] Keyed to genus only; almost certainly *R. variabilis*.
[e] Found ≥1 individual keyed to genus only; almost certainly *R. variabilis*.

Other parasitic helminths. The following helminths were infrequently observed in PG: Leigh (1940) reported thorny-headed worms (*Mediorhynchus papillosus*) in 2 of 28 Illinois GPCs. He viewed their occurrence as "undoubtedly accidental" (Leigh 1940:187). Trematodes, which use gastropods or bivalves as intermediate hosts, were infrequently identified in STG. Boughton (1937) found encysted *Agamodistomum* sp. adolescaria (Strigeidae) in the breast muscle and associated subcutaneous tissue of 1 of 62 birds from Minnesota. McIntosh (1937), Babero (1953), and Boddicker (Hillman and Jackson 1973) reported *Athesmia wehri*, *Brachylaima fuscatum* (=*B. fuscata*), and *Echinostoma revolutum* in STG from Montana, Alaska, and South Dakota, respectively. The significance of these parasites to host populations, if any, is unknown.

Microparasites

Hematozoa. Several species of hematozoa have been demonstrated in PG from throughout their range. *Leucocytozoon* sp. were found in STG in Michigan (Saunders 1935, Baumgartner 1939). Flakas (1952) reported *L. bonasae* from blood films from 15 of 41 STG and 1 of 60 GPCs sampled in Wisconsin, while Cowan and Peterle (1957) found this parasite in blood films from 67 of 126 STG captured in Michigan. Wetmore (1939) observed *Plasmodium* sp. on blood smears taken from clinically ill STG in North Dakota. This hematozoan, later designated as *P. pedioecetii* (Stabler et al. 1973, Stabler and Kitzmiller 1976), also was identified in blood films from 53 of 130 and 4 of 8 STG captured in North Dakota (Shillinger 1942) and Colorado, respectively (Stabler et al. 1974, Stabler and Kitzmiller 1976). Stabler (1978) identified *P. pedioecetii* on blood films from 2 of 29 and 2 of 8 LPCs from New Mexico and Texas, respectively. Stabler and Miller (1984) identified *P. pedioecetii* on blood films from 6 of 25 GPCs from Colorado and Smith et al. (2003) identified it on blood films from 4 of 32 LPCs captured in New Mexico. The flagellated protozoan *Trypanosoma avium* was found on blood smears taken from 2 of 8 STG in Colorado (Stabler et al. 1966, 1974). Lastly, White and Bennett (1979) identified *Haemoproteus mansoni* from blood films taken from STG from an unrecorded location.

Arthropod vectors are required to transmit hematozoa. The hematozoa of PG are thought to be transmitted a follows: *L. bonasae* by black flies and midges, *T. avium* by black flies and *Culex* mosquitoes, *P. pedioecetii* by *Culex* and *Aedes* mosquitoes, and *H. mansoni* by midges and hippoboscid flies (*Ornithomyia* spp.; Springer 1997). For this reason, these parasites tend to occur seasonally. A number of writers have argued that hematozoa might be a serious problem for PG, particularly young birds (e.g., Saunders 1935, Shillinger 1942, Cowan and Peterle 1957). Although parasite intensities sometimes were high, and disease observed (Flakas 1952, Cowan and Peterle 1957), studies addressing the population-level significance of these agents have yet to be completed.

Other protozoa. It is reasonable to assume that *Eimeria* spp. occur at some level in many, if not most, PG populations. Few surveys of intestinal coccidia, however, have been completed. Clarke (1936) found that intestinal coccidiosis killed an STG in western Ontario. Boughton (1937) identified *E. dispersa* and *E. angusta*, respectively, in 1 of 30 and 7 of 39 samples taken from Minnesota STG. In Wisconsin, Morgan, and Hamerstrom (1941) identified *E. dispersa* from 3 of 39 samples taken from GPCs and *E. angusta* in 3 of 62 STG samples. Smith et al. (2003) described *E. tympanuchi* for 5 of 64 LPCs captured in New Mexico. The life cycle of *Eimeria* spp. is direct, but oocysts shed in the feces of an infected grouse must sporulate to become infective to a susceptive host (life cycle about 7–14 days; McDougald and Reid 1997). While *Eimeria* spp. are highly pathogenic to many avian species under certain conditions, such as during captive propagation (Shillinger and Morley 1937), the significance of intestinal coccidiosis in free-living PG has yet to be evaluated.

Prairie grouse researchers have long assumed that the flagellated protozoan *Histomonas meleagridis* negatively influences PG populations (Gross 1928, 1930). There probably are 2 reasons for this supposition: the ubiquitous nature of the required vector and the severity of histomoniasis (blackhead disease) in PG. *Histomonas meleagridis* uses the commonly occurring cecal worm *H. gallinarum* (Table 2) as a vector (McDougald 1997). Conversely, *H. isolonche* does not appear to be a good vector for *H. meleagridis* (Davidson et al. 1978). Infected birds shed *H. meleagridis* oocysts within *H. gallinarum* eggs in the feces. Resulting larvae then are ingested directly by susceptible grouse, or indirectly when these birds consume earthworms or other soil invertebrates that have ingested the larvae (McDougald 1997). Gross (1928) documented *H. meleagridis*-induced mortality of HHs. Similarly, Gross (1930), Leigh (1940), and Schwartz (1945) described GPCs killed by histomoniasis in Wisconsin, Illinois, and Missouri, respectively. Although *H. meleagridis* definitely causes mortality of free-living PG, its population-level influence remains uncertain.

I found documentation for only one tissue-inhabiting protozoan in PG. Drouin and Mahrt (1979) observed *Sarcocystis* sp. in 1 of 76 STG examined in Alberta. Two vertebrate hosts are required for *Sarcocystis* spp. to com-

plete their life cycles: typically a prey species (intermediate host) and predator (definitive host) (Springer 1997). Pathogenicity for individuals and influence on PG population dynamics are unknown.

Bacteria and similar organisms. I found few clearly documented cases of disease caused by bacteria in free-living PG. Green and Shillinger (1932) cultured *Francisella tularensis* (=*Pasteurella tularensis*), the etiologic agent of tularemia, from the liver of an apparently healthy STG harvested from Minnesota. The tick *H. leporispalustris* acts as a vector for this agent. Harper et al. (1968) failed to culture *Salmonella* sp. or other pathogenic bacteria from the spleens and livers of 71 GPCs collected by Kansas hunters during November. *Mycoplasma* sp. and *Salmonella* sp. (group B) were isolated from the trachea and kidney, respectively, of apparently healthy LPCs captured in Oklahoma and Kansas, 2001–2002 (D. H. Wolfe, Sutton Avian Research Center; C. A. Hagen, Kansas State University, respectively, unpublished data). Captive STG have been diagnosed with ulcerative enteritis (Morley and Wetmore 1936, Shillinger and Morley 1937), a serious epizootic disease caused by *Clostridium colinum* (Berkhoff et al. 1974, Berkhoff 1985). Unless a major epizootic was underway, however, finding fresh, intact PG that recently succumbed to bacterial infection in the wild would be rare indeed. Moreover, case reports documenting bacterial diseases in individual animals are not encouraged by most refereed journals, so only the biologists who worked up such cases are likely to be aware of them. For example, researchers in Kansas isolated *Pasteurella multocida* from lung, liver, and spleen of 2 LPCs with clinical signs of avian cholera during 2001 (C. A. Hagen, Kansas State University, unpublished data).

During the last few years, researchers increasingly have used serologic techniques to determine whether apparently healthy PG had developed antibodies specific to various bacterial diseases of galliforms. Peterson et al. (1998*b*) tested sera from 19 APCs captured on 3 study areas in coastal Texas for specific antibody against *Salmonella typhimurium*, *S. pullorum*, *Mycoplasma gallisepticum*, *M. synoviae*, and *Chlamydophila psittici* (=*Chlamydia psittici*); all were negative. They did, however, find 4 of 27 samples positive for antibody against *P. multocida*. Numerous waterfowl had died of avian cholera in pastures where 3 of the 4 birds were captured. Similarly, Peterson et al. (2002) tested sera collected from 24 LPCs captured in northeastern Texas. All were negative for antibody against *S. typhimurium*, *S. pullorum*, *M. gallisepticum*, *M. synoviae*, and *C. psittici*. Hagen et al. (2002), however, found 8, 8, and 5 of 162 LPCs captured in Kansas seropositive for *M. gallisepticum*, *M. synoviae*, and *M. meleagridis*, respectively, using a serum plate antigen (SPA) test. Because these SPA-positive samples were not confirmed by hemagglutination inhibition testing, and no cultures were attempted (Hagen et al. 2002), the authors could not be certain which *Mycoplasma* sp. elicited the positive SPA responses (Peterson et al. 2002).

It is probable that PG would succumb to most typical galliform diseases caused by bacteria, *Mycoplasma* spp., or *C. psittici*. The exact influence these agents might have on PG population dynamics, however, has yet to be explored.

Fungi. Little has been published regarding the fungal diseases of free-living PG. Swales (1934) reported that a number of STG in Quebec died of blastomycosis shortly after being placed in captivity. Clarke (1936) found that mycotic pneumonia killed a STG in Manitoba. Similarly, Kubena (1969) reported that 5 captive APCs died of aspergillosis. *Aspergillus* spp. also can produce a potent mycotoxin. There are several other mycotic parasites of birds that almost certainly infect PG. Boddicker (Hillman and Jackson 1973), for example, identified ringworm (*Trichophyton* sp.) on STG captured in South Dakota. Further, the host range for most agents causing thrush is rather large and includes other grouse species (Hubbard et al. 1985, Chute 1997), so PG probably are susceptible as well. Although I found no records of PG mortality caused by mycotoxins, there is no reason to think they are not susceptible. While at least pulmonary aspergillosis and mycotoxins undoubtedly can cause serious disease, the importance of fungal agents to PG populations has yet to be evaluated.

Viruses. Beginning in the mid-1990s, interest in the viral diseases of PG increased. Peterson et al. (1998*b*) found 19 APCs captured in coastal Texas negative for specific antibody against the Newcastle disease, infectious bronchitis, and avian influenza viruses. Drew et al. (1998) isolated the reticuloendotheliosis virus (REV) from 7 and 1 clinically ill GPCs and APCs, respectively, held in captivity. Asymptomatic GPCs and APCs at this facility also were found positive for REV proviral DNA by polymerase chain reaction (PCR), REV-specific antibody by virus neutralization testing, and the REV by isolation. Additionally, Drew et al. (1998) found REV-specific antibody in sera from 2 of 25 free-living APCs. Lastly, Wiedenfeld et al. (2002) found only 2 GPCs from Oklahoma positive for REV proviral DNA by PCR out of 354 birds tested from 7 states.

Peterson et al. (2002) found blood samples from 0 of 24 LPCs captured in northeastern Texas positive for REV proviral DNA by PCR. Similarly, Wiedenfeld et al. (2002) found 0 of 184 LPCs trapped in Kansas, Oklahoma, and New Mexico positive for REV proviral

DNA. Peterson et al. (2002) also failed to detect antibody against the avian influenza (n=24) or Newcastle disease (n=23) viruses in samples taken from LPCs in Texas. Two of 18, and 8 of 17 individuals, however, were seropositive for the Massachusetts and Arkansas serotypes of the infectious bronchitis virus on microhemagglutination-inhibition testing. Five of the 8 positive individuals were juveniles, 2 of which were serologically positive for both serotypes.

Without doubt, the Newcastle disease, infectious bronchitis, avian influenza, and reticuloendotheliosis viruses could cause serious disease in individual PG and have the potential to influence population dynamics. It also is reasonable to assume that other viruses that cause disease in a broad array of galliforms might be pathogenic to PG as well. No data are available regarding how PG populations react to epizootics caused by these viruses.

Parasites and prairie grouse populations

The previous section should convince readers that while numerous parasites have been documented for PG, little is known regarding the consequences of specific infectious agents for PG populations. Here, I explore the macro- and microparasites known to occur in PG that, based on available data, are most likely to 1) regulate host populations or 2) extirpate small, isolated populations. I caution, however, that other parasites of *Tympanuchus* spp. might be just as important as those discussed below; there simply are too few data for adequate evaluation. Similarly, infectious agents not identified for PG, but known to cause disease in other galliforms, also could be equally important to PG populations.

Regulation

Macroparasites. If parasites suppress the size or resilience of small, isolated populations, they simultaneously increase the risk of extinction caused by other factors. Because the macroparasites of PG typically exhibit aggregated distributions (Dick 1981, Pence et al. 1983, Peterson et al. 1998*b*), they theoretically could regulate PG populations. Before addressing which macroparasites might be most likely regulatory candidates, it is important to recall that not all the helminthic endoparasites listed in Tables 2–3, for example, would be expected in all PG populations. There is ample evidence that climate largely can explain the helminthic endoparasite communities of PG across their range (Boughton 1937, Pence and Sell 1979, Peterson 1996). Thus a specific nematode might regulate a PG population in one region of North America, yet not even occur in another. For this reason, one must be cautious regarding generalizations.

Although most macroparasites found in PG have the potential to regulate populations, 2 stand out as likely candidates. Gross (1928) implicated *D. nasuta* in the demise of the HH. This parasite also caused significant disease and mortality in ruffed grouse, particularly in young birds (Gross 1925*a*, *b,* 1931; Allen and Gross 1926; Levine and Goble 1947; Ruff and Norton 1997). Similarly, Bendell (1955) found that *D. nasuta* was an important cause of mortality in blue grouse (*Dendragapus obscurus*) chicks, and argued that it regulated the blue grouse population he studied on Vancouver Island, Canada. If *D. nasuta* is as pathogenic for young prairie chickens as it appears to be for ruffed and blue grouse chicks, it could, for example, contribute to the comparatively low number of juvenile Attwater's versus GPCs surviving per brood prior to brood breakup (Peterson and Silvy 1996, Peterson et al. 1998*a*). Although Harper et al. (1967) did not consider *D. nasuta* detrimental to the GPC populations they studied, the viscera they examined were collected at hunter check stations in November, so no young birds were in the sample. For this reason, they could not address the potential importance of a parasite that is pathogenic primarily to chicks. At any rate, *D. nasuta* has the requisite characteristics to regulate PG populations where it occurs (Table 2).

The second macroparasite of PG that might regulate host populations is *T. cramae*. As detailed above, there are good reasons to deduce that the interaction between *T. cramae* and APCs is more similar to that of *T. tenuis* and red grouse than *T. cramae* and northern bobwhites (Peterson et al. 1998*b*). In red grouse, cecal lesions and inflammation caused by *T. tenuis* have long been recognized (Shipley 1911, Wilson and Leslie 1911, Watson et. al. 1987). High *T. tenuis* intensities were associated with reduced host fecundity and poor survival (Wilson and Leslie 1911, Potts et al. 1984, Hudson 1986, Hudson et al. 1992*b*). Experimental reductions in parasite intensities demonstrated that decreased *T. tenuis* intensities were associated with increased body weight, adult survival, clutch size, egg hatchability, nesting success, and brood-rearing success in red grouse (Hudson 1986, Shaw 1990, Hudson et al. 1992*a*, *b*). Moreover, Tompkins et al. (2002) maintained that the interaction between *T. tenuis* and red grouse in northern England was the only host–parasite relationship in which experimental field research has unambiguously demonstrated a parasite regulating a wildlife population (Hudson et al. 1998). If the ecological relationship between *T. cramae* and PG is similar to that of red grouse and *T. tenuis*, this parasite must

be considered a potential regulator of PG populations. Considering the precarious status of the remaining APC populations (Silvy et al. 1999), the possibility that *T. cramae* suppresses the size or resilience of these populations, rendering them more susceptible to stochastic extinction by other factors, deserves more serious consideration (Peterson et al. 1998*b*).

Delineation of *D. nasuta* and *T. cramae* as potential regulators of PG populations should not be construed as ruling out other macroparasites associated with these host species (Tables 1–3). For example, earlier authors found *R. variabilis* pathogenic for young PG (Leigh 1940, 1941; Harper et al. 1967). They also argued that host population dynamics would be altered because fewer young birds would be recruited into the breeding population. Further, if certain parasitic arthropods or helminths alter PG behavior, as they do in other galliforms (see detailed treatment, below), such interactions also might regulate host populations. Further research is needed to explore these possibilities.

Microparasites. As detailed earlier, endemic microparasitic diseases that reduce fecundity or recruitment of young into the breeding population in a density-dependent fashion could regulate host populations. The coccidia associated with PG (*E. dispersa*, *E. angusta*), for example, typically cause decreased growth and significant mortality in young birds (McDougald and Reid 1997, Friend and Franson 1999*b*), thus potentially limiting recruitment. Agents causing avian malaria (e.g., *L. bonasae* and *P. pedioecetii*) also might regulate PG populations because they can cause severe anemia, weight loss, and mortality, particularly in young birds within the first few weeks of hatching (Atkinson 1999).

If the infectious bronchitis virus is as pathogenic for PG as it is for domestic chickens, it could regulate numbers by greatly curtailing the number of chicks surviving to broad breakup (Peterson et al. 2002). Another microparasite that might regulate host populations is REV. This retrovirus causes immunosuppression and an overall disease syndrome in prairie chickens (Drew et al. 1998) that is not unlike that caused by the human immunodeficiency virus (HIV), also a retrovirus. The fact that REV exhibits characteristics needed to regulate host populations, and evidence of exposure was documented in free-ranging APCs in Refugio County, Texas, just prior to population extinction (Drew et al. 1998), should concern those managing small, isolated populations of PG.

Extirpation

Unlike the situation with waterfowl (Friend et al. 2001), there is little evidence of large-scale epizootics characterized by high mortality that are limited to PG. Although it would be foolish to assume such epizootics could never occur, if a disease outbreak were to extirpate small, isolated PG populations, it seems more likely that it would result from spillover from an epizootic primarily involving other wild or domestic avian species. Such scenarios have long concerned wildlife managers. Several authors have commented on the risk of PG contracting infectious diseases from domestic poultry (e.g., Gross 1928, Leigh 1940, Schwartz 1945). More recently, wildlife scientists have become concerned about epizootics in waterfowl spilling into isolated PG populations, possibly leading to their extirpation (Peterson et al. 1998*b*). There are numerous infectious agents documented for PG that sometimes occur epizootically, are characterized by significant mortality, and could extirpate small, isolated populations of PG.

Microparasites. As detailed earlier, histomoniasis has long concerned those managing small and/or isolated PG populations (Gross 1928, 1930; Leigh 1940, Schwartz 1945). This parasite caused morbidity and mortality ≥75% in ruffed grouse populations, and 75 and 50% morbidity and mortality, respectively, in northern bobwhite populations (Davidson and Nettles 1997). In southeastern wild turkeys (*Meleagris gallopavo*), histomoniasis was the second most common infectious disease observed during a 12-year period (Davidson et al. 1985), with mortality >75% of infected wild turkeys common (Davidson and Nettles 1997). There seems little doubt that 75% PG mortality, during a single epizootic, could devastate small, isolated populations. Moreover, *H. meleagridis* and *H. gallinarum* most likely have been associated with ring-necked pheasants since the late Cenozoic era (Lund and Chute 1974). Because ring-necked pheasants serve as nearly ideal hosts for these parasites (Lund and Chute 1972), wildlife managers should question the wisdom of perpetuating this species in areas inhabited by at risk populations of PG.

Avian cholera is another potential threat to small, isolated PG populations that are in areas where epizootics commonly occur in waterfowl or other species. There are several areas in the United States where pinnated grouse occur in habitats where avian cholera epizootics occur frequently. For example, those managing the Attwater Prairie Chicken National Wildlife Refuge take this threat seriously; to prevent this disease in APCs, they routinely collect and incinerate carcasses of waterfowl that succumbed to avian cholera (Peterson et al. 1998*b*).

There are insufficient data to develop informed judgments regarding many other epizootic, microparasitic diseases of galliforms. For example, although avian influenza, Newcastle disease, and salmonellosis have not

been reported in PG, there is no reason to assume that these agents would not be detrimental to small, isolated populations of PG if epizootics occurred. Additionally, there is no clearly discernible line of demarcation between parasites that regulate, and those that might extirpate, tenuous host populations. By way of illustration, although I discussed coccidia under the population regulation heading, *Eimeria* sp. killed approximately 400 juvenile greater sage-grouse (*Centrocercus urophasianus*) in Wyoming out of a total population of about 2,000 by September (Simon 1940). Thus, coccidiosis in PG not only could be regulatory, it also might threaten a small, isolated population.

Nonparasitic diseases. Although this essay is focused on host–parasite interactions, I would be remiss if I failed to acknowledge that several nonparasitic diseases also could limit PG reproduction or cause high mortality. For example, mycotoxins associated with milo or corn used as bait for white-tailed deer (*Odocoileus virginianus*) or northern bobwhites probably could lead to immunosuppression and decreased reproductive success, growth, and survival of PG chicks, as these toxins do in other galliforms (Hoerr 1997, Quist et al. 2000). Even though regulations are in place limiting aflatoxin levels in wildlife feed, as long as baiting remains legal in PG habitat, agencies will find it difficult to control mycotoxin levels in wildlife feed after these products leave retailers' shelves. Pesticides and other toxic compounds also could put small, isolated PG populations at additional risk (Julian and Brown 1997). Lehmann and Mauermann (1963) described hundreds of APCs found dead near a cotton field dusted aerially with arsenic. Watkins (1969) found higher DDT concentrations in the organs and tissues of 10 APCs that died during capture than for 9 surviving birds. Blus et al. (1989) documented substantial greater sage-grouse morbidity and mortality associated with dimethoate application in southeastern Idaho. Conversely, Flickinger and Swineford (1983) found low levels of organochlorine pesticide, polychlorinated biphenyl, and heavy metal residues in APC tissues, whether birds originated from agricultural landscapes or rangelands. In certain landscapes inhabited by PG, more systematic efforts to monitor and prevent mycotoxicoses, organophosphorus intoxication, or other toxic diseases should be considered.

Parasites and host behavior

Field biologists have long recognized that isolated PG populations tend to disappear once the numbers drop below some critical threshold, assuming habitat is not acquired or improved (Toepfer et al. 1990, Westemeier and Gough 1999). Sometimes, even when relatively large tracts of apparently suitable habitat are available, as was the case for the APC in Refugio County, Texas, extinction still occurs (Silvy et al. 1999). Biologists studying APCs have observed that once the number of displaying males per ancestral lek decreases to about 5, male behavior becomes erratic (e.g., attend ≥3 leks in <1 hr) and these leks typically disappear (M. J. Peterson, unpublished data), suggesting there might be a significant behavioral component to extinction of PG populations.

There also is increasing evidence that parasites can alter reproductive behavior of gallinaceous birds. For example, Zuk et al. (1990) found that male red jungle fowl (*Gallus gallus*) chicks, experimentally infected with the nematode *A. galli*, developed less-impressive secondary sex characteristics. At maturity, females preferred to mate with unparasitized roosters by approximately 2:1. The social rank of experimentally infected females inoculated as chicks was lower than non-infected hens, but mate choice was not influenced (Zuk et al. 1998). Similarly, Hillgarth (1990) found that captive male ring-necked pheasants treated to reduce coccidia burdens displayed more vigorously and were chosen more frequently as mates.

Johnson and Boyce (1991) found that male greater sage-grouse infected with the hematozoan *P. pedioecetii* exhibited lower lek attendance and bred later in the breeding season than uninfected males. They also learned that breeding males were less likely than nonbreeding males to bear the mallophagans *L. gibsoni* and *Goniodes centrocerci* and the associated hematomas on their air sacs. Spurrier et al. (1991) experimentally demonstrated that female greater sage-grouse discriminated against lousy males based on visually obvious hematomas. Conversely, Gibson (1990) found that *Haemoproteus* sp. did not appear to influence male greater sage-grouse mating behavior or success. Similarly, Tsuji et al. (2001) found no evidence that light infestations of *Goniodes* sp. influenced mating behavior of male STG.

Insufficient data are available to draw any firm conclusions regarding how parasites might influence reproductive behavior of PG. If parasitic helminths, coccidia, ectoparasites, hematozoa, or other parasites alter PG mating behavior as they do in other closely related galliforms, this condition could have important consequences for genetic composition and viability of small, isolated populations.

Research needed

The most significant shortcoming of existing research directed toward PG–parasite interactions is that almost nothing is known regarding the population-level significance of these agents. Readers should not construe my

call for experimental studies designed to determine the population-level significance of these agents as a prohibition of further cataloging of the infectious agents of PG, however. Rather, I maintain that while we should welcome additional comprehensive parasite surveys of the sort typically published in *Avian Diseases*, the *Journal of Wildlife Diseases*, or traditional parasitology journals, wildlife managers desperately need ecologically based studies that address the potential significance of these agents to PG populations. This is particularly critical for those attempting to manage small, isolated populations.

How does one approach determining whether a parasite might regulate or otherwise influence PG populations? As Peterson et al. (1998*b*) contended, research addressing *T. tenuis* and red grouse populations in northern England and Scotland offers a useful point of departure. For example, it was first determined that *T. tenuis* can cause cecal lesions and inflammation in free-roaming red grouse (Shipley 1911, Wilson and Leslie 1911, Watson et. al. 1987). High *T. tenuis* intensities then were associated with reduced host fecundity and survival of wild red grouse (Wilson and Leslie 1911, Potts et al. 1984, Hudson 1986, Hudson et al. 1992*b*). Experimental reductions in parasite intensities in free-living red grouse demonstrated that increased *T. tenuis* intensities were associated with decreased body weight, adult survival, clutch size, egg hatchability, nesting success, and brood-rearing success in red grouse (Hudson 1986, Shaw 1990, Hudson et al. 1992*a,b*), including rendering laying and incubating hens and their nests far more vulnerable to predation (Hudson et al. 1992*b*). This research theme then led to a definitive experimental field study (Hudson et al. 1998). Using data collected earlier (Potts et al. 1984, Hudson et al. 1985, Hudson 1992), Hudson et al. (1998) found cyclic fluctuations in red grouse abundance, with a period of 4–8 years, for 77% of 175 grouse moors evaluated. They then used long-term data from 6 of these moorlands to predict the next 2 crashes in grouse numbers at these sites. Red grouse in 4 of the 6 populations were captured and treated with an oral anthelmintic prior to the first predicted crash, 2 of the 4 were treated again prior to the second predicted crash, and the remaining 2 populations were used as untreated controls throughout. Using this replicated field experiment, Hudson et al. (1998) demonstrated that anthelmintic application markedly reduced the tendency of all 6 treated populations to cycle, as compared to the control moors, thus unambiguously demonstrating that *T. tenuis* was the driving force behind cycles in these populations. The mechanism accounting for this observation was identified earlier, using modeling and empirical data, as primarily a density-dependent reduction in host fecundity (Dobson and Hudson 1992).

Moss et al. (1996) also were able to prevent a population cycle at their study area in Scotland by removing territorial cocks from the population. They maintained that in this case, changes in food availability, nitrogen metabolism, and *T. tenuis* intensity could not explain their results. This study illustrates another strength of the *T. tenuis*–red grouse model: one should not necessarily expect *T. tenuis* to regulate red grouse numbers everywhere within the range of this host and under all conditions. For this same reason we would not know for certain whether *T. tenuis* could regulate populations of the closely related willow ptarmigan (*Lagopus lagopus*) in North America without further research. In sum, one should expect climatic, edaphic, and vegetative factors to be involved not only with where specific parasitic helminths occur but also with how hosts and parasites interact.

It seems that a logical place to begin such analyses would be with the macro- and microparasites delineated earlier. Although it is not particularly difficult to determine whether parasites reduce fecundity and survival of wild hosts, it is a different matter entirely to demonstrate that a parasite regulates host populations. In addition to the example of *T. tenuis* and red grouse, Tompkins and Begon (1999) and Tompkins et al. (2002) outlined useful criteria for this purpose. Correlative studies such as those relating PG nesting success or juvenile to adult ratios to parasite burdens are insufficient to demonstrate population-level influences of a parasite. It unquestionably is difficult to isolate the influence of parasites on PG populations from other interspecies and habitat-based interactions. Therefore, complex, integrative research approaches combining laboratory studies, retrospective analyses of field data, and field experiments will be necessary to determine whether a specific parasite influences PG population dynamics, and to describe the mechanisms that account for these changes (Peterson 1991*b*, 1996; Peterson et al. 1998*b*, 2002).

Summary and implications

Several surveys of the macroparasites of PG were completed between the late 1920s and mid-1940s, but comparatively few such studies were completed from the 1950s through the mid-1990s. If nothing else, Anderson and May's models served notice that if it is reasonable to assume that predators can influence prey population dynamics, it is equally reasonable to suppose that macro- and microparasites also have this potential. Since then, numerous authors have used models to evaluate the potential of parasites to regulate or otherwise influence

host population dynamics. When one leaves the theoretical realm and enters the practical, however, only one field experiment, addressing *T. tenuis* in red grouse, unambiguously demonstrated a parasite regulating a free-ranging grouse population (Hudson et al. 1998, Tompkins et al. 2002). Although this host–parasite interaction might not behave identically throughout the range of red grouse (Moss et al. 1996), it is likely that the dearth of evidence for such relationships speaks more about trends in wildlife science than wildlife populations.

A wide array of parasites has been documented for PG. Although many of these infectious agents cause disease in individual PG, there are few data regarding their influence on host population dynamics. By relying on existing data regarding PG parasitism, studies conducted in other grouse species, and theoretical perspectives toward host–parasite interactions, we can obtain some idea of the parasites most likely to regulate or otherwise influence PG populations. Macroparasites having the potential to regulate PG populations include *D. nasuta*, and *T. cramae*. Among microparasites known to occur in PG, coccidia such as *E. dispersa* and *E. angusta*, the malarial agents *L. bonasae* and *P. pedioecetii*, infectious bronchitis virus, and REV exhibit characteristics that make them potential regulatory agents. Microparasites of PG that occur epizootically and are characterized by high mortality have the potential to extirpate small, isolated populations. Likely suspects include *H. meleagridis*, *P. multocida*, *Eimeria* spp., and other microparasites having broad galliform host ranges. Nonparasitic diseases caused by mycotoxins, pesticides, and other toxic compounds also could extirpate small, isolated populations. Because there appears to be a behavioral component to the extinction of PG populations, and parasites of various types have been shown to influence the behavior of other galliforms (including tetraonids), increased effort should be made to evaluate whether and how parasites influence the breeding behavior of at-risk PG populations.

Unfortunately, almost no effort has been expended to determine how parasites influence PG populations. Definitive experiments designed to establish whether parasites regulate their host populations undoubtedly are difficult to design, but guidelines for this purpose have been published (Tompkins and Begon 1999, Tompkins et al. 2002). Further, research addressing the *T. tenuis*–red grouse interaction in northern England and Scotland serves not only to illustrate how one might determine whether a parasite regulates a grouse population but also to demonstrate several ways to evaluate the effect of parasites on specific aspects of grouse life history. It is critical that we employ such methodologies to evaluate whether selected macro- and microparasites influence the dynamics of declining or otherwise at-risk PG populations while there is still time for management intervention should it be warranted.

Natural-resource policy-makers must become aware that macro- and microparasites of wildlife are not something they can safely ignore. For example, administrators of several North American wildlife agencies recently came face-to-face with the ecological and political morass associated with chronic wasting disease in free-living cervids (Williams et al. 2002). There is no reason to believe that avian species, including PG, are necessarily exempt from such ecological and political conundrums. When one considers the number of extinct PG populations, overall declining PG abundance, and the number of populations currently at risk of extinction, it seems obvious that infectious agents with the potential to either regulate host abundance or extirpate small, isolated populations must be taken into consideration during formation and implementation of wildlife management plans.

This task will not be easily accomplished. Unfortunately, wildlife scientists are not accustomed to addressing host–parasite interactions in the systematic way they do other interspecific relationships, such as predator–prey interactions (Peterson 1991*a*). Probably for this reason, North American wildlife scientists rarely have designed, let alone conducted, studies that directly tested hypotheses pertinent to those formulating and implementing wildlife disease management policies (Peterson 1991*b*). More typically, wildlife disease researchers evaluated wild species as potential reservoirs for diseases of humans or domestic animals, parasite taxa of interest to systematists, limiting factors for captive production, or other reasons. This situation must be rectified. Wildlife managers and administrators could go a long way toward this goal by funding research designed to clarify the influence of parasites on PG population dynamics. Similarly, parasitologists and traditional wildlife disease researchers might consider integrating studies that address how macro- or microparasites influence PG populations with their more customary investigations of parasite taxonomy, pathogenesis, vaccine development, or diagnostic and monitoring techniques. In the end, if we are to tease out the influence of parasites versus habit, weather, predators, and other factors on PG population dynamics, complex, integrative research approaches using expertise from multiple academic disciplines will be needed. Results of these studies, however, would be directly relevant to those managing PG populations.

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Markus J. Peterson is an associate professor in the Department of Wildlife and Fisheries Sciences at Texas A&M University. He received his B.S. from University of Idaho, D.V.M. from Washington State University, and M.S. and Ph.D. from Texas A&M University. Markus has been a member of The Wildlife Society since the 1980s. His research interests include the ecology and management of terrestrial vertebrate populations with an emphasis on factors influencing animal abundance, such as wildlife disease, predation, weather, habitat conditions, and human exploitation. He also has a keen interest in environmental policy formation and implementation.

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