Management and Conservation Article

Deer Carcass Decomposition and Potential Scavenger Exposure to Chronic Wasting Disease

CHRISTOPHER S. JENNELLE,[1] *Department of Forest and Wildlife Ecology, University of Wisconsin, 1630 Linden Drive, Madison, WI 53706, USA*

MICHAEL D. SAMUEL, *United States Geological Survey, Wisconsin Cooperative Wildlife Research Unit, University of Wisconsin, 1630 Linden Drive, Madison, WI 53706, USA*

CHERRIE A. NOLDEN, *Department of Forest and Wildlife Ecology, University of Wisconsin, 1630 Linden Drive, Madison, WI 53706, USA*

ELIZABETH A. BERKLEY, *Department of Forest and Wildlife Ecology, University of Wisconsin, 1630 Linden Drive, Madison, WI 53706, USA*

ABSTRACT Chronic wasting disease (CWD) is a transmissible spongiform encephalopathy afflicting the Cervidae family in North America, causing neurodegeneration and ultimately death. Although there are no reports of natural cross-species transmission of CWD to noncervids, infected deer carcasses pose a potential risk of CWD exposure for other animals. We placed 40 disease-free white-tailed deer (*Odocoileus virginianus*) carcasses and 10 gut piles in the CWD-affected area of Wisconsin (USA) from September to April in 2003 through 2005. We used photos from remotely operated cameras to characterize scavenger visitation and relative activity. To evaluate factors driving the rate of carcass removal (decomposition), we used Kaplan–Meier survival analysis and a generalized linear mixed model. We recorded 14 species of scavenging mammals (6 visiting species) and 14 species of scavenging birds (8 visiting species). Prominent scavengers included American crows (*Corvus brachyrhynchos*), raccoons (*Procyon lotor*), and Virginia opossums (*Didelphis virginiana*). We found no evidence that deer consumed conspecific remains, although they visited gut piles more often than carcasses relative to temporal availability in the environment. Domestic dogs, cats, and cows either scavenged or visited carcass sites, which could lead to human exposure to CWD. Deer carcasses persisted for 18 days to 101 days depending on the season and year, whereas gut piles lasted for 3 days. Habitat did not influence carcass decomposition, but mammalian and avian scavenger activity and higher temperatures were positively associated with faster removal. Infected deer carcasses or gut piles can serve as potential sources of CWD prions to a variety of scavengers. In areas where surveillance for CWD exposure is practical, management agencies should consider strategies for testing primary scavengers of deer carcass material. (JOURNAL OF WILDLIFE MANAGEMENT 73(5):655–662; 2009)

DOI: 10.2193/2008-282

KEY WORDS camera trap, carcass decomposition, chronic wasting disease, *Odocoileus virginianus*, prions, scavengers, transmissible spongiform encephalopathy, white-tailed deer, Wisconsin.

Chronic wasting disease (CWD) is a transmissible spongiform encephalopathy (TSE) affecting mule deer (*Odocoileus hemionus*), white-tailed deer (*O. virginianus*), Rocky Mountain elk (*Cervus elaphus nelsoni*), and moose (*Alces alces shirasi*), which has been found in ≥13 states and 2 Canadian provinces (Williams and Miller 2002, Baeten et al. 2007, Sigurdson and Aguzzi 2007). Disease is characterized by progressive accumulation of misfolded prion proteins (PrP^{CWD}) in the central nervous system and brain, ultimately leading to death. Other TSE diseases include scrapie in sheep (*Ovis* spp.), bovine spongiform encephalopathy in cattle, and variant Creutzfeldt–Jakob disease in humans; the latter 2 are food-chain diseases transmitted by consumption of infected animal tissues. Oral ingestion of infectious materials from conspecific animals or from environmental sources is a known route of CWD and scrapie infection (Williams and Miller 2003, Miller et al. 2004, Beekes and McBride 2007). Potential transmission of CWD via the food chain has not been extensively evaluated, but human consumption of CWD-infected tissue should be avoided (World Health Organization 2000). The brain, spinal cord, and lymphoid tissues of the gut and head harbor the highest concentrations of PrP^{CWD} (Spraker et al. 2002), but prion deposition also occurs in the pituitary glands, adrenal glands, and pancreas, as well as striated muscle tissues of infected cervids (Sigurdson et al. 2001, Jewell et al. 2006). Body fluids including blood and saliva can also contain infectious prions (Mathiason et al. 2006). Although many tissues from infected animals may contain PrP^{CWD}, it is still unclear which source(s) pose the highest risk to susceptible hosts.

Carcasses infected with CWD are an important source of infectious prions to susceptible cervids and may expose vertebrate scavengers to PrP^{CWD} (Miller et al. 2004, 2006). Agents that cause TSEs can remain viable in the environment for many years and prions bound with soil particles of montmorillonite increase oral infectivity (Miller et al. 2004, Georgsson et al. 2006, Johnson et al. 2007, Seidel et al. 2007). During decomposition, ungulate carcasses release nutrients into surrounding soils, stimulate subsequent biomass production that attracts herbivores, and serve as a potential source of infectious material (Towne 2000, Miller et al. 2004). Carcasses also provide a ready source of protein for vertebrate consumers. Although species barriers to TSE infection are generally robust (Chesebro 2003), transmission to a resistant species was achieved in the laboratory by passage of infectious material through an intermediate species (Bartz et al. 1998). Although this experiment demonstrates the possibility of cross-species transmission, the probability of such events in natural systems is unknown. Of additional concern is that acidic conditions found in the mammalian gastrointestinal system can enhance conversion of normal cervid prions to PrP^{CWD} in a variety of species (Li et al. 2007). Together, these findings highlight the

[1] *E-mail: jennelle@wisc.edu*

importance of understanding the potential exposure of vertebrate scavengers to CWD-infected deer carcasses.

Little is known about the rate of scavenger consumption and decomposition of deer remains, the types and numbers of wildlife species that might be exposed to PrP^{CWD} agent by consuming infected deer tissues, the potential role of these species in CWD dynamics, or the implication of PrP^{CWD} ingestion by scavenger species. A host of mammals including canids, felids, raccoons (*Procyon lotor*), opossums (*Didelphis virginiana*), and striped skunks (*Mephitis mephitis*) are likely scavengers on deer carcasses. It has even been reported that deer may consume animal tissues, including flesh and bone of dead conspecifics (V. Geist, University of Calgary, personal communication; Cook et al. 2004). Avian scavengers also are consumers of ungulate carrion (Wilmers et al. 2003, Cook et al. 2004), but birds are not susceptible to mammalian TSEs (Wopfner et al. 1999). It may be possible, however, for avian and mammalian scavengers to consume TSE-infected materials and spread prions, or other infectious agents, through deposition of feces in the environment (Bullock 1956, Houston and Cooper 1975, European Commission 2002) or by transport of infectious carrion during food-caching or young-provisioning. Therefore, infected deer carcasses should be considered a source of infectious prions that could infect free-living deer, contribute to CWD spread, and facilitate interspecies transmission. Our goals were to 1) identify mammalian and avian scavengers potentially exposed to PrP^{CWD} from consumption of experimentally placed deer carcasses, 2) determine the length of time that deer carcasses and gut piles persisted in the environment, and 3) determine biotic and abiotic factors that affect rates of carcass consumption and decomposition in the environment.

STUDY AREA

Our study area of approximately 544 km^2 encompassed the core area of highest disease prevalence within the disease eradication zone (DEZ) of Dane and Iowa counties in south-central Wisconsin, USA (Joly et al. 2003). The landscape in this area was characterized by rolling hills and small stream valleys with a mixture of dairy farms, oak–hickory woodlots, and agricultural fields, almost exclusively in private ownership. Prior to CWD discovery (posthunt 2001), deer density in the core study area was estimated at 13.5–15.5 deer/km^2 (Grear et al. 2006). The study area was approximately bordered by the Wisconsin River between Sauk and Iowa counties to the North, Ridgeway to the southwest, and Mount Horeb to the southeast.

METHODS

Experimental Methods

Most deer carcasses we used were fawns (approx. 6–9 months of age) because they were unlikely to be infected with CWD (Grear et al. 2006). We also used 2 adults obtained within the DEZ, which were CWD-negative, to prevent environmental contamination and exposure to scavengers during our study. We qualitatively assessed each carcass we chose for our study to be approximately similar in size (approx. 39 kg).

We randomly placed 40 deer carcasses throughout the landscape and monitored them for 2–3 months between September and April of 2003–2004 and 2004–2005. We either placed fresh deer carcasses in the environment or temporarily froze them for later use. We used remotely activated cameras to quantify scavenger activity of animals scavenging and visiting carcasses. We visited carcasses approximately 2–3 times/week to download data from cameras and qualitatively assess carcass volume (% remaining); E. A. Berkley always assessed carcass volume. We monitored carcasses until all flesh was consumed or decomposed, at which point only pieces of hide and bones persisted, which can remain intact in the environment for years (Behrensmeyer 1978).

We used CamTrakker™ 35-mm film cameras (CamTrakker, Watkinsville, GA) during the first year of our study (18 carcasses) and Reconyx™ Silent Image digital cameras (Silent Image Professional, Holmen, WI) during the second year (22 carcasses). We calibrated CamTrakker cameras to capture images of moving animals (at least the size of small rodents) in the proximity of a carcass, with a 3-minute delay before movement in the camera zone could initiate another image. We used Reconyx cameras in the second year because CamTrakker cameras used a white flash, experienced a slow wake-up period (i.e., delay before image taken), and made mechanical noise prior to image capture. Reconyx cameras used an infrared flash invisible to scavengers and produced negligible noise during operation. We calibrated Reconyx cameras to collect images of moving animals in the proximity of a carcass; at initiation, 4 images were taken at 1-second intervals, followed by a 30-second delay. Given these differences in photographic equipment, calibration, and potential behavioral effects of CamTrakker cameras on scavenger visits, a direct comparison of carcass consumption and scavenger activity between years was not appropriate, so we analyzed each year separately.

We placed deer carcasses on private property during autumn (carcass deposition in Oct), winter (Nov through Jan), and winter–spring (Feb or Mar) in 4 habitat types (i.e., wooded, field, edge, stream). Wooded habitats were second-growth oak (*Quercus* spp.) and hickory (*Carya* spp.) forest, and we placed carcasses in the forest interior $\geq$300 m from an edge. Field habitats were warm- and cool-season grasses, and we placed carcasses $\geq$100 m from the closest edge. Edge habitat was characterized by lands bordering fields and forests, and we placed carcasses within 5 m of a habitat edge. Streams were located within forests, open grassland, or fields, where we placed carcasses within 5 m of a riparian area.

In each photograph, we identified animals to species and counted the number of distinct individuals in the field of view. Because animals were not marked, we could not determine whether the same individuals were present on subsequent photos. When we used CamTrakker cameras in the first year, there were few cases (<1%) when we could

not identify animals to species. We further improved species identification in the second year by taking an initial sequence of 4 successive images using the Reconyx cameras. We calculated an index of weekly scavenger activity (SA) corrected for average species mass as

$$\mathrm{SA}_{ijk} = \sum_{j=1}^{x} \sum_{i=1}^{y} C_{ijk} \times m_i$$

where C_{ijk} is the count of distinct individuals of species i in photo j of week k, and m_i is average adult mass (kg) of species i (derived from Poole 2002 and Myers et al. 2006). We included mass to account for potential species difference in tissue consumption based on body size. This index provided a measure of scavenger activity at each carcass that accounted for variation between scavenger species and permitted us to summarize the relative activity levels of broader taxonomic groups (e.g., mammals vs. birds).

We randomly placed gut piles (i.e., visceral remains consisting of the lung, heart, spleen, liver, and gastrointestinal tract) from 10 deer on private property consisting of either wooded, old field, or wooded edge landscapes between 20 September and 5 November 2004. We monitored gut piles daily until complete removal occurred (presumably due to consumption). We recorded vertebrate scavenger activity with Reconyx digital cameras to determine species composition and relative activity at gut piles.

We identified nearly all vertebrate animals in photographs to species, and made a distinction between scavengers and visitors based on animal behavior in each picture. We unambiguously defined as scavengers species in which we observed individuals feeding at a carcass or gut pile. Some individuals, however, showed behavior consistent with strict inspection (visual and olfactory) of a carcass or gut pile, suggesting a high likelihood of direct contact with remains. We quantified relative scavenger activity (RSA) of each species by their proportional activity:

$$\mathrm{RSA}_i = \sum_{j=1}^{x} C_{ij} / \sum_{j=1}^{x} \sum_{i=1}^{y} C_{ij}$$

where C_{ij} is the count of distinct individuals of species i in photo j. We summarized this activity index separately for each deer carcass and gut pile for each year of our study to estimate the relative (proportional) contact that a given species had with carcass material.

Statistical Analysis

We conducted analyses using SAS/STAT™ software version 9.1 (SAS Institute, Cary, NC). To characterize persistence time of carcasses (only flesh components) and gut piles in the environment, we used PROC LIFETEST, which estimates a survival function with the Kaplan–Meier method. We used this method to estimate median number of days each carcass persisted and, therefore, the primary period of potential CWD exposure for scavengers. Given the small sample size of gut piles, we could not test for seasonal and habitat effects on persistence; thus, we simply estimated median number of days gut piles were present in the environment.

We performed a repeated-measures analysis, using week as the temporal unit of the repeated measure, with deer carcasses as experimental units. We determined how the between-carcass factors of season and habitat and the within-carcass factors of average temperature and SA index for mammalian and avian scavengers affected the weekly proportion of deer carcass consumed and decomposed during each year of our study. Because we measured avian and mammalian activity on the same scale (in the SA index), we compared model parameter estimates to assess the relative importance of these factors with respect to carcass removal. We used PROC GLMMIX with a Beta distribution for the response variable of weekly proportion of carcass consumed and decomposed; this procedure numerically estimates model parameters by maximizing the residual log pseudo-likelihood. To ensure convergence of parameter estimates we used the Nelder–Mead Simplex Optimization. We modeled the autocorrelation between repeated measures on deer carcasses with an autoregressive structure, which assumes highest covariance between successive weeks and is more parsimonious than either unstructured or compound symmetry.

RESULTS

Scavenger Communities

Although we used better camera equipment in the second year (2004–2005) with a shorter photograph interval, scavenger composition and activity between years generally agreed. We identified 14 species of scavenging mammals and 14 species of scavenging birds, as well as 6 mammalian and 8 avian visitors (Table 1). Each species we identified as a visitor accounted for only a fraction of a percent of total animal activity at carcass remains (except deer at gut piles). American crows (*Corvus brachyrhynchos*), raccoons, and opossums were consistently the top 3 active scavengers (Fig. 1). Although mammalian scavenger activity predominated, prominent avian scavengers included turkey vultures (*Cathartes aura*) and red-tailed hawks (*Buteo jamaicensis*), and we identified 6 passerine and 2 galliform bird species as visitors to carcass remains (Table 1).

We found white-tailed deer were infrequent visitors at carcasses and gut piles. Deer visits represented only a fraction (<1%) of total animal activity at carcasses in both years and accounted for 4.5% of all activity at gut piles. During the second year of our study the daily rate of deer visits to carcasses ($\bar{x} = 0.24$, SE = 0.09, $n = 19$) was lower (Mann–Whitney U-test; $Z = 1.73$, $P = 0.042$) than to gut piles ($\bar{x} = 1.3$, SE = 0.44, $n = 10$). Despite longer persistence of carcasses in the environment, distributions of the absolute number of deer visits were not different (Mann–Whitney U-test; $Z = -1.27$, $P = 0.206$) between carcasses ($\bar{x} = 13.5$, SE = 4.1, $n = 19$) and gut piles ($\bar{x} = 5.8$, SE = 1.7, $n = 10$).

We documented deer-carcass scavenging or visitation by several animal species that have direct contact with humans or human food supplies, including domestic dogs and cats,

Table 1. Vertebrate species encountered at white-tailed deer carcasses and gut piles between September 2003 and April 2005 in Dane and Iowa counties, Wisconsin, USA. We consistently observed some species captured in photographs around the periphery of a carcass or gut pile, and we thereby tentatively classified them as visitors to a site (whereas we classified species clearly consuming materials from carcasses or gut piles as scavengers).

Mammals	Birds
American mink (*Neovison vison*)	American robin (*Turdus migratorius*)[a]
Common raccoon (*Procyon lotor*)	American crow (*Corvus brachyrhynchos*)
Cow[a]	Bald eagle (*Haliaeetus leucocephalus*)
Domestic cat	Barred owl (*Strix varia*)
Domestic dog	Black-capped chickadee (*Poecile atricapillus*)[a]
Eastern cottontail (*Sylvilagus floridanus*)[a]	Blue jay (*Cyanocitta cristata*)
Eastern coyote (*Canis latrans*)	Dark-eyed junco (*Junco hyemalis*)[a]
Eastern gray squirrel (*Sciurus carolinensis*)[a]	Downy woodpecker (*Picoides pubescens*)
Ermine (*Mustela erminea*)	European starling (*Sturnus vulgaris*)[a]
Gray fox (*Urocyon cinereoargenteus*)	Hairy woodpecker (*Picoides villosus*)
Red fox (*Vulpes vulpes*)	Golden eagle (*Aquila chrysaetos*)
Northern river otter (*Lontra canadensis*)[a]	Great-horned owl (*Bubo virginianus*)
Northern short-tailed shrew (*Blarina brevicauda*)	Northern flicker (*Colaptes auratus*)
Southern flying squirrel (*Glaucomys volans*)	Red-bellied woodpecker (*Melanerpes carolinus*)
Striped skunk (*Mephitis mephitis*)	Red-tailed hawk (*Buteo jamaicensis*)
Long-tailed weasel (*Mustela frenata*)	Ring-necked pheasant (*Phasianus colchicus*)[a]
White-footed mouse (*Peromyscus leucopus*)	Rough-legged hawk (*Buteo lagopus*)
White-tailed deer (*Odocoileus virginianus*)[a]	Tree swallow (*Tachycineta bicolor*)[a]
Virginia opossum (*Didelphis virginiana*)	Tufted titmouse (*Baeolophus bicolor*)[a]
Woodchuck (*Marmota monax*)[a]	Turkey vulture (*Cathartes aura*)
	White-breasted nuthatch (*Sitta carolinensis*)
	Wild turkey (*Meleagris gallopavo*)[a]

[a] Classified as a visitor to carcass or gut-pile sites.

and visitation of one carcass by cows. Of these species, dogs were the most active at carcasses, accounting for as high as 6% of total animal activity during year 1, whereas cats and cows accounted for <1% of total animal activity.

Carcass and Gut-Pile Persistence and Decomposition

We estimated that deer gut piles persisted a median of 3 days (95% CI = 2.8–6.0) during September and October 2004. Consumption and decomposition of all the flesh on deer carcasses ranged from 3 weeks to 15 weeks. In year 1 (2003–2004), deer carcasses persisted for a median of 55 days (95% CI = 43–55) in autumn, 101 days (95% CI = 85–106) in winter, and 65 days (not enough data for 95% CI) in winter–spring (Fig. 2A). Distribution of persistence times did not differ by habitat ($\chi^2_3 = 3.71$, $P = 0.29$). However, persistence differed between seasons ($\chi^2_2 = 6.10$, $P = 0.05$), with a shorter persistence in autumn compared to winter ($Z = 2.38$, $P = 0.03$) but no differences between autumn and winter–spring ($Z = 1.12$, $P = 0.39$) or winter and winter–spring ($Z = 1.28$, $P = 0.30$).

In year 2 (2004–2005), deer carcasses persisted for a median of 18 days (95% CI = 11–25) in autumn, 55 days (95% CI = 49–59) in winter, and 36 days (95% CI = 28–58) in winter–spring (Fig. 2B). The effect of habitat on differences in carcass persistence was negligible ($\chi^2_3 = 3.03$, $P = 0.39$). As in year 1, persistence differed between seasons ($\chi^2_2 = 20.10$, $P < 0.001$) and was shorter in autumn compared to winter ($Z = 3.61$, $P < 0.001$) or winter–spring ($Z = 2.18$, $P = 0.04$), with no difference between winter and winter–spring ($Z = 1.21$, $P = 0.34$). Carcass persistence was shorter in year 2 when compared with corresponding seasons in year 1 for autumn ($\chi^2_1 = 8.01$, $P = 0.005$), winter ($\chi^2_1 = 9.60$, $P = 0.002$), and winter–spring ($\chi^2_1 = 3.96$, $P = 0.05$).

For the first year (2003–2004), the generalized chi-square divided by degrees of freedom (0.99) indicated the data were not overdispersed and parameter variances were unbiased. The repeated-measures mixed model revealed that neither season ($F_{2,12} = 0.11$, $P = 0.90$) nor habitat ($F_{3,12} = 1.02$, $P = 0.42$) described variation in the weekly proportion of deer carcass removed. Likewise, the weekly SA index for avian species did not influence ($F_{1,96} = 2.04$, $P = 0.16$) carcass decomposition. There was a marginal effect of temperature ($F_{1,96} = 2.82$, $P = 0.09$) and a strong effect of mammalian activity ($F_{1,96} = 23.91$, $P < 0.001$). Model parameters were positive, indicating that greater mammalian activity and higher temperatures were associated with higher weekly carcass decomposition.

For the second year (2004–2005), the generalized chi-square divided by degrees of freedom was 1.00, indicating no overdispersion in the data. As in the first year, neither season ($F_{2,16} = 0.35$, $P = 0.71$) nor habitat ($F_{3,16} = 0.16$, $P = 0.92$) explained variation in the weekly proportion of deer carcass removed. However, we found that temperature ($F_{1,133} = 5.97$, $P = 0.016$), avian activity ($F_{1,133} = 23.63$, $P < 0.001$), and mammalian activity ($F_{1,133} = 24.6$, $P < 0.001$) were positively associated with weekly carcass decomposition. Parameter estimates for effects of avian and mammalian activity indicated a 1-unit increase in avian SA index had 2.5 times the impact on carcass decomposition as did a 1-unit increase in mammalian SA index. Despite a greater effect of birds per unit body mass on carcass removal, mammals had a higher absolute impact because on average they had approximately 10 times greater mass than birds.

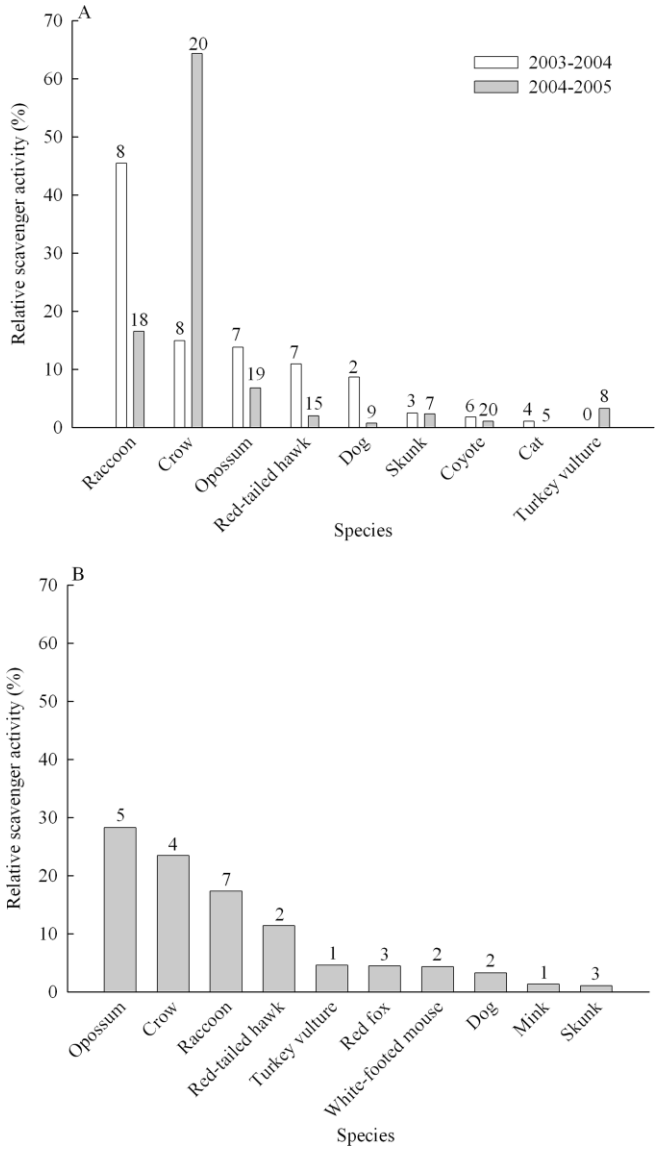

Figure 1. Distribution of relative activity by each scavenger species at white-tailed deer carcass and gut-pile sites in Dane and Iowa counties, Wisconsin, USA, limited to species contributing >1% of total activity. We calculated relative scavenger activity (RSA) at carcasses over 14 sites in the first year (2003–2004), 20 sites in the second year (2004–2005; panel A), and 9 gut piles (panel B) in 2004. We used a restricted set of sites because not all deer carcasses were monitored until all flesh remains were consumed. The number of sites in which we identified a given species is listed above each RSA value.

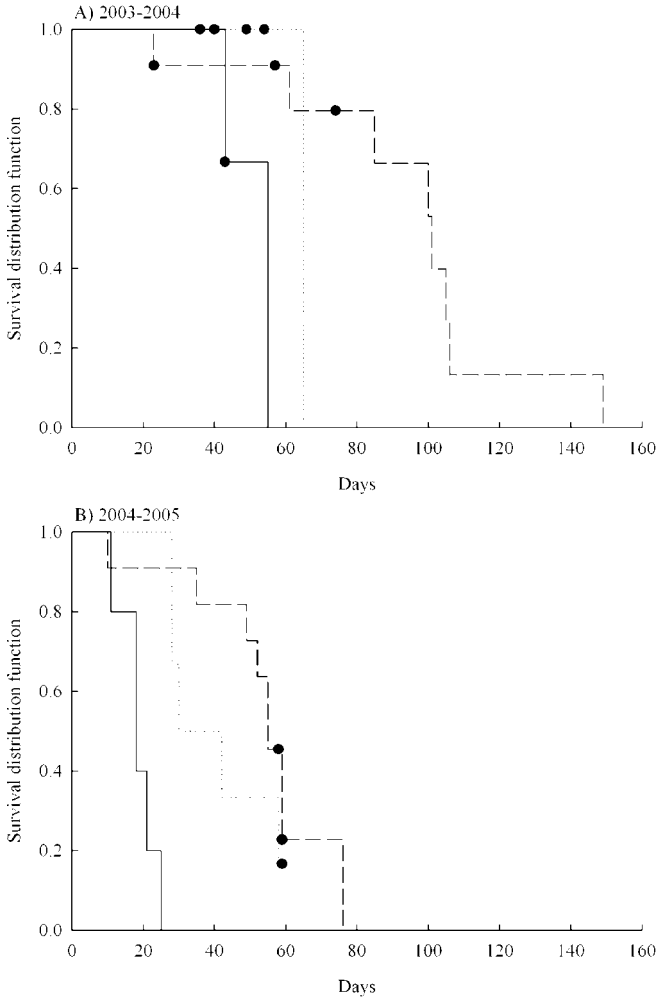

Figure 2. Persistence distributions of all flesh remains of white-tailed deer carcasses in A) 2003–2004 and B) 2004–2005, stratified by season: autumn (solid line), winter (dashed line), and winter–spring (dotted line) in Dane and Iowa counties, Wisconsin, USA. Solid circles represent right-censored data.

DISCUSSION

Miller et al. (2004) demonstrated that susceptible deer become infected when housed in paddocks with deer carcasses containing PrP^{CWD}, presumably through direct carcass contact or indirectly when decomposing carcasses contaminated the environment. By extension, deer carcasses harboring PrP^{CWD} (regardless of the proximate cause of death) may serve as a point-source of infectious material, which may persist in the environment for many years. Although our photographic evidence did not suggest that free-ranging deer consumed carcass materials, deer inspected carcasses, which increased probability of contact with infectious materials. Furthermore, the subsequent flush of vegetation surrounding a site of carcass decomposition may attract deer and other herbivores (Towne 2000, Carter et al. 2007). Consumption of this vegetation and infectious material in contaminated soil could facilitate indirect transmission of CWD (Miller et al. 2004, Johnson et al. 2006).

Longer persistence of infected deer carcasses may increase disease exposure for animals that inspect or consume carrion. Our study demonstrated the importance of mammalian and avian scavengers, as well as possible microbial and invertebrate action (inferred from positive temp effect), in removal of deer remains from the environment. We found annual and seasonal differences in the persistence of deer carcasses on the landscape, with >2 times longer persistence in the first year of our study, regardless of season. Within a given year, carcasses persisted >2 times longer in winter than autumn and >1.5 times longer in winter–spring than autumn. Thus, exposure of susceptible animals to infected deer carcasses was reduced in warmer seasons. We also expect that carcass persistence

during summer would be further reduced by the additive action of microbial decomposers, arthropods, and increased abundance of migratory avian scavengers such as turkey vultures.

Although we demonstrated that ambient temperature was correlated with the rate of carcass removal, there were minimal differences in seasonal temperatures between years. Thus, we believe longer carcass persistence in the first year occurred because the noise of operation and visible flash of CamTrakker cameras may have frightened some scavengers away from carcasses, thus increasing persistence time. This effect may have been particularly pronounced with crows, because there was clearly much lower proportional usage of carcasses by crows in the first year of our study. Furthermore, this potential disturbance may explain why the effect of the SA index for birds on weekly decomposition rate was negligible in the first year.

Warmer environmental temperatures increase fungal and bacterial growth, as well as arthropod consumption and use of carcasses (Putman 1978). Although active competition occurs between animal scavengers and carrion decomposers (Janzen 1977, Burkepile et al. 2006), scavengers may be the primary carrion consumers (Putman 1983). Although many studies have been conducted using bird and rodent carrion to monitor scavenger activity (DeVault et al. 2003, 2004), only a few studies have utilized large vertebrate animals or gut piles (Wilmers et al. 2003, Roen and Yahner 2005, Selva et al. 2005) and none have incorporated cameras into their design. The primary scavenger species that we identified at deer remains (raccoons, opossums, and crows) or their close taxonomic relatives were typically cited in the aforementioned studies. Because we incorporated cameras to record scavenger activity, our study is likely more representative of the scavenger communities (and visitors) of deer carcasses and gut piles in our study area. Furthermore, use of cameras incorporating infrared technology likely minimized disturbance to scavengers.

Although our study was primarily concerned with the potential exposure of scavenger communities to CWD, animal carcasses can also serve as reservoirs that mediate transmission and spread of a broad array of pathogens or contaminants to wild animals, highlighting the potential importance of scavengers in the study of disease dynamics. Evidence suggests that the causal agent of bovine tuberculosis (*Mycobacterium bovis*) is a major threat to livestock (and in some cases humans) around the world. Magnifying the problem is the ease with which scavengers of tuberculosis-infected carcasses are infected (Bruning-Fann et al. 2001, Delahay et al. 2002, Michel 2002, Renwick et al. 2007). Furthermore, it has been shown that livestock carrion can be a source of pathogenic bacteria such as Salmonella for avian scavengers including red kites (*Milvus milvus*; Blanco et al. 2006), a species experiencing historical declines. Even domestic cats can become infected with West Nile virus after consuming infectious rodent carcasses (Austgen et al. 2004), yet it is not clear whether cats can serve as infectious reservoirs for mosquito vectors. Although considered non-infectious agents, contaminants and poisons can be passed along to scavengers of tainted carcasses. For example, deer carcasses containing lead from bullet fragments can function as a source of lead poisoning to scavengers (Hunt et al. 2006), and livestock carcasses containing the drug diclofenac were responsible for precipitous declines of the Oriental white-backed vulture (*Gyps bengalensis*; Oaks et al. 2004). Taken together, such evidence supports the need for further research regarding scavenger ecology within the context of wildlife and human health, wildlife management, and biological conservation.

Our study provides evidence that deer remains are routinely scavenged or visited by a broad range of mammalian and avian scavengers and decomposed by microbes. In particular, we identified cats, raccoons, and 4 mustelid species at deer remains, which are all susceptible to TSE infection (Eckroade et al. 1973, Marsh and Hadlow 1992, Pearson et al. 1992, Hamir et al. 2003). Despite the short persistence of deer gut piles (median of 3 days), a variety of scavengers used this ephemeral resource (Table 1). Furthermore, the daily rate of deer contacts with gut piles was greater than with whole carcasses, suggesting a higher daily risk of potential CWD exposure for susceptible deer. However, the total risk of exposure will also depend on the persistence and abundance of gut piles versus carcasses in the environment.

In the context of disease transmission mediated by contact with carrion, it is important to measure the activity level of scavengers to infer relative exposure to disease agents and potential for spread. Ideally, studies on disease exposure should distinguish between scavengers and visitors, and identify specific infectious tissues that scavengers consume; a limitation in our study. Scavenging of CWD-infected deer remains poses a confounding duality between the removal of infectious materials by consumption and potential dispersion of infectious material in the environment. Scavengers are directly exposed to infectious material by consuming contaminated deer tissues, creating the potential for cross-species transmission (Bartz et al. 1998), with subsequent fecal deposition of infectious CWD material on the landscape. On the other hand, scavengers are effectively removing infectious tissue, decreasing the load of PrP^{CWD} from a carcass site and potentially reducing exposure to susceptible deer. Little is known about the importance of these processes and how they might contribute to CWD dynamics. Yet it stands to reason that scavengers may serve as alternative hosts in multispecies disease systems, buffers that effectively reduce the load of infectious material from the environment, or asymptomatic disease spreaders.

MANAGEMENT IMPLICATIONS

We support research to determine whether primary scavengers of deer remains might be useful bio-indicators of environmental contamination with PrP^{CWD} and provide insights on the possible cascade of infectious prions through food webs. Future investigations of CWD exposure to noncervids should focus on the primary scavengers of deer

remains including opossums, raccoons, crows, red-tailed hawks, and turkey vultures, as well as susceptible species of felids and mustelids. Experimental research should determine susceptibility of these mammalian species to infection and the likelihood of PrP^{CWD} passage through the gastrointestinal tract of all these animal species. Because CWD-infected deer could be differentially susceptible to hunting mortality (Conner et al. 2000, but see Grear et al. 2006) and there is a temporal concentration of gut piles produced during hunting seasons (during the 2005 and 2006 hunting seasons, we estimated 4–5 gut piles/km^2 were produced in our study area), deer carcasses and gut piles should be considered from a disease management perspective. When selective harvesting or sharp-shooting of deer is warranted in high-risk CWD regions, we suggest management agencies consider removal of harvested animals (including viscera) to limit potential PrP^{CWD} deposition near a kill site. When public hunting is permitted in CWD regions, it may be useful to consider practical solutions for hunters to bury (or otherwise remove) visceral remains from cervids with a higher likelihood of infection, to reduce exposure of susceptible animals to potentially infectious material. Further research to assess the risk of cervid infection from CWD-infected carcasses or viscera in free-ranging cervid populations would be beneficial.

ACKNOWLEDGMENTS

We thank M. Mead and C. Niebur for field assistance, private landowners for use of their properties, and R. Rolley for deer harvest data in Wisconsin. We thank D. Drake, C. Jacques, K. VerCauteren, and 2 anonymous reviewers for comments on earlier drafts. We thank the United States Geological Survey National Wildlife Health Center for funding and the Wisconsin Department of Natural Resources for logistic support and assistance in obtaining deer remains. Mention of trade names or commercial products does not imply endorsement by the United States government.

LITERATURE CITED

Austgen, L. E., R. A. Bowen, M. L. Bunning, B. S. Davis, C. J. Mitchell, and G. J. Chang. 2004. Experimental infection of cats and dogs with West Nile virus. Emerging Infectious Diseases 10:82–86.

Baeten, L. A., B. E. Powers, J. E. Jewell, T. R. Spraker, and M. W. Miller. 2007. A natural case of chronic wasting disease in a free-ranging moose (*Alces alces shirasi*). Journal of Wildlife Diseases 43:309–314.

Bartz, J. C., R. F. Marsh, D. I. McKenzie, and J. M. Aiken. 1998. The host range of chronic wasting disease is altered on passage in ferrets. Virology 251:297–301.

Beekes, M., and P. A. McBride. 2007. The spread of prions through the body in naturally acquired transmissible spongiform encephalopathies. Federation of European Biochemical Societies Journal 274:588–605.

Behrensmeyer, A. K. 1978. Taphonomic and ecologic information from bone weathering. Paleobiology 4:150–162.

Blanco, G., J. A. Lemus, and J. Grande. 2006. Faecal bacteria associated with different diets of wintering red kites: influence of livestock carcass dumps in microflora alteration and pathogen acquisition. Journal of Applied Ecology 43:990–998.

Bruning-Fann, C. S., S. M. Schmitt, S. D. Fitzgerald, J. S. Fierke, P. D. Friedrich, J. B. Kaneene, K. A. Clarke, K. L. Butler, J. B. Payeur, D. L. Whipple, T. M. Cooley, J. M. Miller, and D. P. Muzo. 2001. Bovine tuberculosis in free-ranging carnivores from Michigan. Journal of Wildlife Diseases 37:58–64.

Bullock, D. S. 1956. Vultures as disseminators of anthrax. Auk 73:283–284.

Burkepile, D. E., J. D. Parker, C. B. Woodson, H. J. Mills, J. Kubanek, P. A. Sobecky, and M. E. Hay. 2006. Chemically mediated competition between microbes and animals: microbes as consumers in food webs. Ecology 87:2821–2831.

Carter, D. O., D. Yellowlees, and M. Tibbett. 2007. Cadaver decomposition in terrestrial ecosystems. Naturwissenschaften 94:12–24.

Chesebro, B. 2003. Introduction to the transmissible spongiform encephalopathies or prion diseases. British Medical Bulletin 66:1–20.

Conner, M. M., C. W. McCarty, and M. W. Miller. 2000. Detection of bias in harvest-based estimates of chronic wasting disease prevalence in mule deer. Journal of Wildlife Diseases 36:691–699.

Cook, W. E., E. S. Williams, and S. A. Dubay. 2004. Disappearance of bovine fetuses in northwestern Wyoming. Wildlife Society Bulletin 32: 254–259.

Delahay, R. J., A. N. S. DeLeeuw, A. M. Barlow, R. S. Clifton-Hadley, and C. L. Cheesman. 2002. The status of *Mycobacterium bovis* infection in UK wild mammals: a review. The Veterinary Journal 164:90–105.

DeVault, T. L., I. L. Brisbin, Jr., and O. E. Rhodes, Jr. 2004. Factors influencing the acquisition of rodent carrion by vertebrate scavengers and decomposers. Canadian Journal of Zoology 82:502–509.

DeVault, T. L., O. E. Rhodes, Jr., and J. A. Shivik. 2003. Scavenging by vertebrates: behavioral, ecological, and evolutionary perspectives on an important energy transfer pathway in terrestrial systems. Oikos 102:225–234.

Eckroade, R. J., G. M. ZuRhein, and R. P. Hanson. 1973. Transmissible mink encephalopathy in carnivores: clinical, light and electron microscopic studies in raccoons, skunks, and ferrets. Journal of Wildlife Diseases 9:229–240.

European Commission. 2002. Opinion on: necrophagous birds as possible transmitters of TSE/BSE. <http://ec.europa.eu/food/fs/sc/ssc/out295_en.pdf>. Accessed 20 Aug 2008.

Georgsson, G., S. Sigurdarson, and P. Brown. 2006. Infectious agent of sheep scrapie may persist in the environment for at least 16 years. Journal of General Virology 87:3737–3740.

Grear, D. A., M. D. Samuel, J. A. Langenberg, and D. Keane. 2006. Demographic patterns and harvest vulnerability of chronic wasting disease infected white-tailed deer in Wisconsin. Journal of Wildlife Management 70:546–553.

Hamir, A. N., J. M. Miller, R. C. Cutlip, M. J. Stack, M. J. Chaplin, A. L. Jenny, and E. S. Williams. 2003. Experimental inoculation of scrapie and chronic wasting disease agents in raccoons (*Procyon lotor*). Veterinary Record 153:121–123.

Houston, D. C., and J. E. Cooper. 1975. The digestive tract of the white-backed griffon vulture and its role in disease transmission among wild ungulates. Journal of Wildlife Diseases 11:306–313.

Hunt, W. G., W. Burnham, C. N. Parish, K. K. Burnham, B. Mutch, and J. L. Oaks. 2006. Bullet fragments in deer remains: implications for lead exposure in avian scavengers. Wilson Society Bulletin 34:167–170.

Janzen, D. H. 1977. Why fruits rot, seeds mold, and meat spoils. American Naturalist 111:691–713.

Jewell, J. E., J. Brown, T. Kreeger, and E. S. Williams. 2006. Prion protein in cardiac muscle of elk (*Cervus elaphus nelsoni*) and white-tailed deer (*Odocoileus virginianus*) infected with chronic wasting disease. Journal of General Virology 87:3443–3450.

Johnson, C. J., J. A. Pedersen, R. J. Chappell, D. McKenzie, and J. A. Aiken. 2007. Oral transmissibility of prion disease is enhanced by binding to soil particles. PLoS Pathogens 3:e93. doi:10.1371/journal.ppat.0030093. Public Library of Science, San Francisco, California, USA.

Johnson, C. J., K. E. Phillips, P. T. Schramm, D. McKenzie, J. M. Aiken, and J. A. Pedersen. 2006. Prions adhere to soil minerals and remain infectious. PLoS Pathogens 2:e32. Public Library of Science, San Francisco, California, USA.

Joly, D. O., C. A. Ribic, J. A. Langenberg, K. Beheler, C. A. Batha, B. J. Dhuey, R. E. Rolley, G. Bartelt, T. R. Van Deelen, and M. D. Samuel. 2003. Chronic wasting disease in free-ranging Wisconsin white-tailed deer. Emerging Infectious Diseases 9:599–601.

Li, L., M. B. Coulthart, A. Balachandran, A. Chakrabartty, and N. R. Cashman. 2007. Species barriers for chronic wasting disease by *in vitro*

conversion of prion protein. Biochemical and Biophysical Research Communications 364:796–800.
Marsh, R. F., and W. J. Hadlow. 1992. Transmissible mink encephalopathy. Scientific and Technical Review of the Office International des Epizooties 11:539–550.
Mathiason, C. K., J. G. Powers, S. J. Dahmes, D. A. Osborn, K. V. Miller, R. J. Warren, G. L. Mason, S. A. Hays, J. Hayes-Klug, D. M. Seelig, M. A. Wild, L. L. Wolfe, T. R. Spraker, M. W. Miller, C. J. Sigurdson, G. C. Telling, and E. A. Hoover. 2006. Infectious prions in the saliva and blood of deer with chronic wasting disease. Science 314:133–136.
Michel, A. L. 2002. Implications of tuberculosis in African wildlife and livestock. Annals of the New York Academy of Sciences 969:251–255.
Miller, M. W., N. T. Hobbs, and S. J. Tavener. 2006. Dynamics of prion disease transmission in mule deer. Ecological Applications 16:2208–2214.
Miller, M. W., E. S. Williams, N. T. Hobbs, and L. L. Wolfe. 2004. Environmental sources of prion transmission in mule deer. Emerging Infectious Diseases 10:1003–1006.
Myers, P., R. Espinosa, C. S. Parr, T. Jones, G. S. Hammond, and T. A. Dewey. 2006. The Animal Diversity Web (online). <http://animaldiversity.org>. Accessed 15 Mar 2008.
Oaks, J. L., M. Gilbert, M. Z. Virani, R. T. Watson, C. U. Meteyer, B. A. Rideout, H. L. Shivaprasad, S. Ahmed, M. J. I. Chaudhry, M. Arshad, S. Mahmood, A. Ali, and A. A. Khan. 2004. Diclofenac residues as the cause of vulture population decline in Pakistan. Nature 427:630–633.
Pearson, G. R., J. M. Wyatt, T. J. Gruffydd-Jones, J. Hope, A. Chong, R. J. Higgins, A. C. Scott, and G. A. Wells. 1992. Feline spongiform encephalopathy: fibril and PrP studies. Veterinary Record 131:307–310.
Poole, A., editor. 2002. Birds of North America online. Cornell Lab of Ornithology, Ithaca, New York, USA. <http://bna.birds.cornell.edu>. Accessed 15 Mar 2008.
Putman, R. J. 1978. Patterns of carbon dioxide evolution from decaying carrion: decomposition of small mammal carrion in temperate systems. Part I. Oikos 31:47–57.
Putman, R. J. 1983. Carrion and dung: the decomposition of animal wastes. Edward Arnold, London, United Kingdom.
Renwick, A. R., P. C. L. White, and R. G. Bengis. 2007. Bovine tuberculosis in African wildlife: a multi-species host–pathogen system. Epidemiology and Infection 135:529–540.
Roen, K. T., and R. H. Yahner. 2005. Behavioral responses of avian scavengers in different habitats. Northeastern Naturalist 12:103–112.
Seidel, B. A., A. Thomzig, M.H. Buschmann, R. Groschup, M. Beekes Peters, and K. Terytze. 2007. Scrapie agent (Strain 263K) can transmit disease via the oral route after persistence in soil over years. PLoS One 2: e435. Public Library of Science, San Francisco, California, USA.
Selva, N., B. Jedrzejewska, W. Jedrzejewski, and A. Wajrak. 2005. Factors affecting carcass use by a guild of scavengers in European temperate woodland. Canadian Journal of Zoology 83:1590–1601.
Sigurdson, C. J., and A. Aguzzi. 2007. Chronic wasting disease. Biochimica et Biophysica Acta 1772:610–618.
Sigurdson, C. J., T. R. Spraker, M. W. Miller, B. Oesch, and E. A. Hoover. 2001. PrP^{cwd} in the myenteric plexus, vagosympathetic trunk and endocrine glands of deer with chronic wasting disease. Journal of General Virology 82:2327–2334.
Spraker, T. R., R. R. Zink, B. A. Cummings, M. A. Wild, M. W. Miller, and K. I. O'Rourke. 2002. Comparison of histological lesions and immunohistochemical staining of proteinase-resistant prion protein in a naturally occurring spongiform encephalopathy of free-ranging mule deer (*Odocoileus hemionus*) with those of chronic wasting disease of captive mule deer. Veterinary Pathology 39:110–119.
Towne, E. G. 2000. Prairie vegetation and soil nutrient responses to ungulate carcasses. Oecologia 122:232–239.
Williams, E. S., and M. W. Miller. 2002. Chronic wasting disease in deer and elk in North America. Scientific and Technical Review of the Office International des Epizooties 21:305–316.
Williams, E. S., and M. W. Miller. 2003. Transmissible spongiform encephalopathies in non-domestic animals: origin, transmission, and risk factors. Scientific and Technical Review of the Office International des Epizooties 22:145–156.
Wilmers, C. C., D. R. Stahler, R. L. Crabtree, D. W. Smith, and W. M. Getz. 2003. Resource dispersion and consumer dominance: scavenging at wolf- and hunter-killed carcasses in greater Yellowstone, USA. Ecology Letters 6:996–1003.
Wopfner, F., G. Weidenhofer, R. Schneider, A. von Brunn, S. Gilch, T. F. Schwarz, T. Werner, and H. M. Schätzl. 1999. Analysis of 27 mammalian and 9 avian PrPs reveals high conservation of flexible regions of the prion protein. Journal of Molecular Biology 289:1163–1178.
World Health Organization. 2000. Infection control guidelines for transmissible spongiform encephalopathies. Report of a World Health Organization consultation. World Health Organization, Geneva, Switzerland.

Associate Editor: VerCauteren.