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The importance of localized culling in stabilizing chronic wasting disease prevalence in white-tailed deer populations

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ABSTRACT

Strategies to contain the spread of disease often are developed with incomplete knowledge of the possible outcomes but are intended to minimize the risks associated with delaying control. Culling of game species by government agencies is one approach to control disease in wild populations but is unpopular with hunters and wildlife enthusiasts, politically unpalatable, and erodes public support for agencies responsible for wildlife management. We addressed the functional differences between hunting and government culling programs for managing chronic wasting disease (CWD) in white-tailed deer by comparing prevalence over a 10-year period in Illinois and Wisconsin. When both Illinois and Wisconsin were actively culling from 2003 – 2007, there were no statistical differences between state CWD prevalence estimates. Wisconsin government culling concluded in 2007 and average prevalence over the next five years was 3.09 ± 1.13% with an average annual increase of 0.63%. During that same time period, Illinois continued government culling and there was no change in prevalence throughout Illinois. Despite its unpopularity among hunters, localized culling is a disease management strategy that can maintain low disease prevalence while minimizing impacts on recreational deer harvest.

1. Introduction

North American cervids [mule deer (*Odocoileus hemionus*), elk (*Cervus elaphus*), moose (*Alces alces*), and white-tailed deer (*Odocoileus virginianus*)] are popular game animals making them economically and recreationally valuable species. Free-living cervids are susceptible to chronic wasting disease (CWD) (Miller et al., 2000; Spraker et al., 1997), a contagious and fatal prion disease with no cure or treatment (Williams et al., 2002). To date, CWD has been identified in free-ranging cervid populations in 17 states and two Canadian provinces (http://www.nwhc.usgs.gov/disease_information/chronic_wasting_disease/index.jsp). CWD is spread in free-living animals through contact with bodily secretions or infectious agents persisting in contaminated environments (Mathiason et al., 2009, 2006; Walter et al., 2011; Williams et al., 2002). Such transmissibility results in a self-sustaining CWD epizootic with prevalence increasing slowly over time (Miller et al., 2000; Miller and Conner, 2005; Saunders et al., 2012; Williams et al., 2002). Furthermore, the environmental load of infectious prions increases with the number of infectious animals making CWD exceedingly difficult to eliminate from free-ranging populations once established (Almberg et al., 2011; Gross and Miller, 2001). CWD models suggest substantial

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declines in cervid populations with high prevalence and highlight the importance of long-term, sustained management programs in controlling CWD (Gross and Miller, 2001; Mateus-Pinilla et al., 2013; Wasserberg et al., 2009).

Information on CWD transmission dynamics in wild populations is very limited. A lack of data has resulted in uncertainty about management actions (Peterson, 1991; Wasserberg et al., 2009). The large number of stakeholders (including hunters, deer biologists, environmental interest groups, and the general public) increase the complexity of decision-making when dealing with threats to economically important agriculture commodities (Carstensen et al., 2011), human health (Daszak et al., 2000), environmental health and the conservation of native plant and animal species. State wildlife agencies are faced with the challenging task of managing deer herds for multiple objectives such as maintaining hunter opportunities, controlling disease spread, limiting negative deer–human interactions and conserving natural resources. Such conflicting objectives make best management practices for wild deer populations highly situational (Carstensen et al., 2011). Because neither practical vaccines nor treatments are available for CWD, reducing deer densities through culling is a common yet controversial disease management approach to minimize contact between infected and susceptible hosts (Carstensen et al., 2011; Potapov et al., 2012; Schmitt et al., 2002; Wasserberg et al., 2009). This practice has been important in successfully eliminating bovine tuberculosis from free-ranging deer in Minnesota (Carstensen et al., 2011) but at this time it is unclear to what extent culling controls CWD and to what extent culling affects hunter opportunity in CWD infected areas (Wasserberg et al., 2009).

CWD was first detected in Illinois and Wisconsin in 2002. Both states banned translocation and baiting of deer in CWD areas but responded with independent disease management strategies. The Illinois Department of Natural Resources (IDNR) implemented a disease management program to bring about small scale population reductions in known CWD infected areas by incorporating additional hunting seasons and government culling (Barlow, 1996). Culling was selective, only occurring in specific 64 km^2 sections [based on the Public Land Survey System (United States Department of the Interior, 2011)] where CWD had been detected by testing hunter-harvest deer. This approach focused culling on localized areas containing deer at greatest risk of current infection and future transmission to additional individuals while limiting the overall number of deer killed.

The Wisconsin Department of Natural Resources' (WDNR) CWD management program aimed at eradicating CWD from the state by establishing a disease management zone consisting of a 1064.5 km^2 area of complete deer eradication surrounded by herd reduction zones (Holsman et al., 2010; VerCauteren and Hygnstrom, 2011). Toward this goal, the WDNR began widespread government culling and attempted to increase hunter harvest opportunities despite declining hunter participation. In 2007, the WDNR culling program was greatly reduced because of public resistance and declining legislative support (Holsman et al., 2010; VerCauteren and Hygnstrom, 2011). Since then, Wisconsin has shifted from a government culling disease management strategy to controlling CWD primarily through public hunting (*Wisconsin's Chronic Wasting Disease Response Plan: 2010–2025*, 2010), while Illinois has consistently used localized government culling to control CWD for the past 10 years.

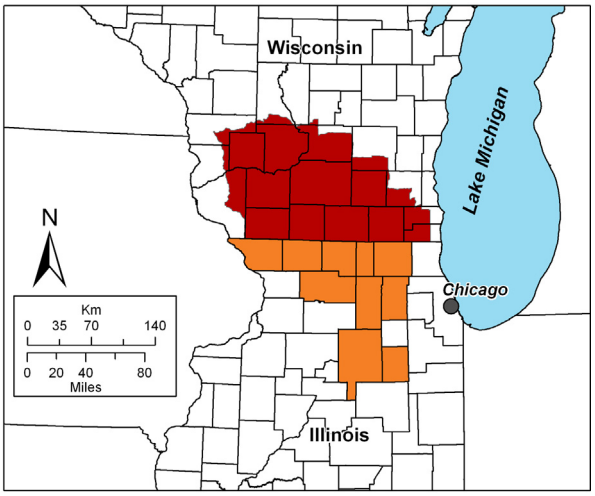

Fig. 1. Areas in Illinois and Wisconsin with chronic wasting disease detected over fiscal year 2003–2012. Orange areas are counties in Illinois and red areas are Wisconsin deer management units that were included in prevalence calculations.

Public opposition to culling as a disease management strategy necessitates an analysis of a sustained culling program that would help guide agencies in selection of CWD management options. Our objectives were to determine if CWD prevalence was affected by the shift in disease management strategies between IL and WI in 2007 and to determine if hunting opportunities in the state of Illinois, where the management has been consistent over 10 years, were affected by disease management strategies.

2. Methods

Samples tested for CWD originated from both public hunting and government culling. All IL samples were tested by the Illinois Animal Disease laboratories using the gold standard immunohistochemical (IHC) examination of retropharyngeal lymph nodes and obex tissue samples. WI samples were tested by the WI Veterinary Diagnostic Laboratory using IHC, or an ELISA based screening test where positive samples were confirmed by IHC. The location of harvest was known for all tested deer samples at either section level or deer management units in IL and WI respectively. Both spatial resolutions met the geographical needs of our selected study area.

To determine whether CWD prevalence was affected by differing disease management strategies between both states, we evaluated 10 years of CWD test results from Illinois and Wisconsin. We calculated CWD prevalence as the number of positive deer divided by the number of total deer tested annually. We confined our prevalence calculations to those areas where CWD positives have been detected from 2002 to 2012 (Fig. 1). For