

it appeared in Minnesota wolves (based upon retrospective serologic evidence) live-trapped as early as 1977 (Mech *et al.* 1986). However, Minnesota wolves may have been exposed to the virus as early as 1973 (Mech and Goyal 1995). Serologic evidence of gray wolf exposure to CPV peaked at 95 percent of a group of Minnesota wolves live-trapped in 1989 (Mech and Goyal 1993). In a captive colony of Minnesota wolves, pup and yearling mortality from CPV was 92 percent of the animals that showed indications of active CPV infections in 1983 (Mech and Fritts 1987), demonstrating the substantial impacts this disease can have on young wolves. It is believed that the population impacts of CPV occur via diarrhea-induced dehydration leading to abnormally high pup mortality (WI DNR 1999a).

There is no evidence that CPV has caused a population decline or has had a significant impact on the recovery of the Minnesota gray wolf population. However, Mech and Goyal (1995) found that high CPV prevalence in the wolves of the Superior National Forest in Minnesota occurred during the same years in which wolf pup numbers were low. Because the wolf population did not decline during the study period, they concluded that CPV-caused pup mortality was compensatory, that is, it replaced deaths that would have occurred from other causes, especially starvation of pups. They theorized that CPV prevalence affects the amount of population increase, and that a wolf population will decline when 76 percent of the adult wolves consistently test positive for CPV exposure. Their data indicate that CPV prevalence in adult wolves in their study area increased by an annual average of 4 percent during 1979–93 and was at least 80 percent during the last 5 years of their study (Mech and Goyal 1995). Additional unpublished data gathered since 1995 indicate that CPV reduced wolf population growth in that area from 1979 to 1989, but not since that period (Mech *in litt.* 1999). These data provide strong justification for continuing population and disease monitoring.

The disease probably stalled wolf population growth in Wisconsin during the early and mid-1980s. During those years, the Wisconsin wolf population declined or was static, and 75 percent of 32 wolves tested by the same method were positive for CPV. During the following years (1988–96) of population increase, only 35 percent of the 63 wolves tested positive for CPV (WI DNR 1999a). CPV exposure rates were at 50 percent in live-captured Wisconsin

wolves in 1995–96 (WI DNR 1999a, but no necropsy evidence of CPV mortalities from Wisconsin wolves exists (Nancy Thomas, National Wildlife Health Laboratory, *in litt.* 1998). Of 13 Wisconsin wolves that died and were examined in 2000, none of the deaths were attributed to CPV (Wydeven *et al.* 2001a). Similarly, CPV is not a suspected cause of death for the 22 wolves with a suspected cause of death identified in 2001 (WI DNR unpublished data). However, the difficulty of discovering CPV-killed pups must be considered.

Canine parvovirus is considered to have been a major cause of the decline of the isolated Isle Royale, Michigan, population in the mid and late 1980s. The Isle Royale gray wolf population decreased from 23 and 24 wolves in 1983 and 1984, respectively, to 12 and 11 wolves in 1988 and 1989, respectively. The wolf population remained in the low to mid-teens through 1995. However, factors other than disease may be causing, or contributing to, a low level of reproductive success, including a low level of genetic diversity and a prey population composed of young healthy moose that may make it difficult to secure sufficient prey for pups. There are no data showing any CPV-caused population impacts to the larger gray wolf population on the Upper Peninsula of Michigan (Peterson *et al.* 1998, Hammill pers. comm. 2002).

Sarcoptic mange is caused by a mite infection of the skin. The irritation caused by the feeding and burrowing mites results in scratching and then severe fur loss, which in turn can lead to mortality from exposure during severe winter weather.

From 1991 to 1996, 27 percent of live-trapped Wisconsin wolves exhibited symptoms of mange. During the winter of 1992–93, 58 percent showed symptoms, and a concurrent decline in the Wisconsin wolf population was attributed to mange-induced mortality (WI DNR 1999a). Seven Wisconsin wolves died of mange from 1993 through October 15, 1998, and severe fur loss affected five other wolves that died from other causes. During that period, mange was the third largest cause of death in Wisconsin wolves, behind trauma (usually vehicle collisions) and shooting (Nancy Thomas *in litt.* 1998).

The prevalence of mange and its impacts on the wolf population have increased in Wisconsin. During the 12-month period from April 2000 through March of 2001, mange appeared to be the second-most common cause of mortality in 23 Wisconsin wolves that

were found dead; mange-induced hypothermia caused the death of 4 wolves and contributed to the death of a fifth wolf. (Motor vehicle collisions caused the death of 10 Wisconsin wolves during this same period, while 2 were shot and 2 were killed by other wolves.) Wolves nearing death from mange generally crawl into dense cover and are difficult to discover if they are not radio-tracked. During the winter of 2000–2001, approximately 14 percent of the radio-collared wolves being tracked by WI DNR died from mange. Other observations showed that some mangy wolves are able to survive the winter (Wydeven *et al.* 2000b, 2001a).

Pup survival during their first winter is believed to be strongly affected by mange. However, estimated survival of Wisconsin pups from 2000 until late winter 2000–2001 was 28 percent, only slightly lower than the previous year's 31 percent pup survival, yet the State's wolf population increased by 21 percent from 1999 to 2000 and only 4 percent from 2000 to 2001 (Wydeven *et al.* 2000a, 2001a). This indicates that mange mortality may not be the primary determinant of wolf population growth in the State, yet the impacts of mange in Wisconsin need to be closely monitored. So far, mange has not caused a sustained decline in the State's wolf population, and the wolf population increased by about 26 percent from late winter 2000–2001 to 2001–2002 despite the continued prevalence of mange in Wisconsin wolves (Wydeven *et al.* 2002).

In a long-term Alberta wolf study, higher wolf densities were correlated with increased incidence of mange, and pup survival decreased as the incidence of mange increased (Brand *et al.* 1995). At least 7 wild Michigan wolves died from mange during 1993–97, making it the most common disease of Michigan wolves. From 1999–2001, mange-induced hypothermia was the cause of death for all 7 Michigan wolves whose cause of death was attributed to disease (Hammill *in litt.* 2002). The Michigan Wolf Management Plan acknowledges that mange may be slowing wolf population growth and specifies that captured wolves be treated with Ivermectin to combat the mites (MI DNR 1997). MI DNR currently treats all captured wolves with Ivermectin if they show signs of mange. In addition, MI DNR vaccinates all captured wolves against CPV and canine distemper virus (CDV), and administers antibiotics to combat potential leptospirosis infections.

Wisconsin wolves similarly had been treated with Ivermectin and vaccinated for CPV and CDV when captured, but