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SALMON POISONING DISEASE: RESEARCH ON A POTENTIAL METHOD OF LETHAL CONTROL FOR COYOTES

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ABSTRACT: Salmon poisoning disease (SPD) was tested as a potential method of lethal control for coyotes (Canis latrans). Fresh fish containing the agents for SPD was fed to 72 captive adult coyotes. Coho (Oncorhunchus kisutch) and steel head salmon (Salmo gairdneri) from Oregon hatcheries were the principal species of fish used. Coyotes that ate the fish developed observable signs of SPD in a mean of 8 days. The overall rate of mortality was 50%, and death occurred in a mean of 20 days from consuming fish. Coyotes that died from SPD lost a mean of 32% of their body weight during the course of the disease. Other coyotes were fed preserved fish samples or administered oral or intraperitoneal treatments of lymph node matter from coyotes that died from SPD. In light of the relatively low rate of mortality observed, feeding coyotes fish to cause death from SPD appears to be a method of questionable value for controlling numbers of adult coyotes in areas of livestock production unless fish with a highly virulent strain of the SPD agent can be obtained.

INTRODUCTION

The difficulty of minimizing the persistent phenomenon of coyote depredation on livestock under some grazing situations and within various regions of North America has invited continued research on methods to reduce predation. A method that gains acceptance must do so by satisfying several criteria. It must be effective, and, ideally, it must also be selective, humane, economically practical, relatively simple--requiring a minimum of specialized equipment, and have minimal adverse impact on the environment. A recent paper (Foreyt et al. 1982) reported a high rate (>90%) of mortality in juvenile coyotes (Canis latrans) experimentally exposed to salmon poisoning disease (SPD).

SPD is a helminth-borne disease highly fatal in canids and is enzootic in the Pacific Northwest (Knapp and Millemann 1970, Millemann and Knapp 1970). The vector is the trematode Nanophuetus salmincola, and the etiologic agents are the rickettsiae Neorickettsia helminthoeca (Cordy and Gorham 1950, Philip et al. 1954) and Elokomin Fluke Fever agent (Farrell 1966). Three hosts are required for completion of the life cycle of the trematode: one species of snail, various salmonid and some non-salmonid fishes, and fish-eating mammals and birds. The distribution of the snail limits the distribution of the trematode and thus the distribution of the disease (Simms et al. 1931). Although many mammals including man may become infected and carry the trematode, only canids are susceptible to the rickettsial agents. Agents are transmitted through all stages of the trematode, and natural infection of canids occurs as they eat infected fish. Canids have been infected experimentally by injection of infected blood, spleen, and lymph node suspensions; injection of ground adult flukes; injection of helminth-infected snail livers; and injection of helminth eggs (Knapp and Millemann 1970).

SPD appears to be a potential candidate for biological control of coyotes since it is specific to canids and apparently causes a relatively high rate of mortality. Domestic dogs (C. familiaris) that contract the disease and are correctly diagnosed can be successfully treated with sulfonamides or various tetracycline drugs (Knapp and Millemann 1970) and thereafter, apparently, are refractory to further infection. There is little likelihood of the complex becoming established outside its present distribution because of the limited range of the intermediate snail host, thus minimizing the potential impact on the environment. We conducted studies with captive adult coyotes to determine the practicality of using SPD as a method of control

METHODS

Coyotes used in the study were part of a captive colony maintained at the U.S. Sheep Experiment Station near Dubois, Idaho. Most were born in captivity, and all originated outside the enzootic area of SPD. Each animal was at least 2 years of age, and those captured from the wild had been in captivity for at least 6 months. Both males and females were used. Most coyotes were housed in enclosed kennel facilities constructed of chain-link fencing with concrete floors. Several coyotes in early trials were kept in outdoor kennels with wire-over-dirt floors. Food (dry dog kibble) and water were provided ad libitum for all kennelled coyotes. Several coyotes were maintained individually within 65-ha enclosures where sheep carrion was the source of food. In most trials, coyotes were weighed before and after the study period.

Fish were obtained from several hatcheries in Oregon and Washington within the enzootic area of SPD. Since fluke metacercariae are most easily observed in fish kidney, microscopic examination of kidney tissue confirmed the presence of metacercariae in all fish samples used in the study. Initially, a small number (N = 1-5) of animals was used in preliminary feeding trials to determine whether fish from a particular hatchery contained the agents to cause SPD. In most preliminary trials, only the kidneys of

fish were offered to coyotes. Each coyote received approximately 0.2 kg of kidney. In later trials coyotes received approximately 1 kg of fish including flesh and kidney but excluding viscera and heads.

To prepare the doses of fish tissue, all fish (N = 10-20) were cut into approximately 10 pieces. Doses were composed of from three to five pieces of flesh selected randomly, each from different fish until the appropriate weight (1 kg) was obtained. Fish tissues were placed on the floor in the kennel of each coyote. Initially the regular ration was left in the kennel, but in later trials it was removed the day prior to offering the fish and was replaced when the coyote consumed the fish. In the enclosures, fish tissues were placed at established feeding sites.

Coyotes in the kennel were observed daily after fish tissues were offered and the following noted: 1) when the fish tissues were consumed and 2) the general condition of the coyote (activity, food con-sumption, frequency and appearance of scats). A rating scale from 0 to 3 was used to grade the condition of coyotes: 0 - no signs; 1 - mild signs, 1-3 days of diarrhea; 2 - moderate signs, anorexia and 4-7 days of diarrhea; and 3 - severe signs, anorexia with >8 days diarrhea and >4 days hemorrhagic diarrhea. Scat was collected from most coyotes several weeks after the fish was consumed to determine the presence or absence of fluke ova. Coyotes that succumbed were necropsied, and fresh tissue samples were collected for analysis. Feeding sites in the enclosures were observed daily until the fish was consumed by the coyotes. Coyotes were observed thereafter daily to determine the day of mortality. Time of onset of signs and general condition of the animal could not accurately be determined for coyotes in enclosures.

Several techniques were used in an attempt to preserve fish, thereby prolonging the period during which it would cause SPD in coyotes. Samples of fish known to contain the causative agents for SPD were treated as follows: 1) frozen at -10ºC. 2) frozen at -58ºC, 3) refrigerated at 4ºC, 4) stored in a glycerine bath at 4ºC, 5) stored in a propylene glycol bath at 4ºC, and 6) pickled in a brine solution for 7 hours followed by hickory smoking for 24 hours at 50ºC. A sample of each type of treated fish was fed to individual coyotes at 3 weeks following preservation. Preserved samples were fed to individual coyotes thereafter as follows: at 6 weeks, one sample of refrigerated fish; at 8 weeks, two samples of refrigerated fish and one sample of each of the other types of preserved fish; and at 13 weeks, two samples of refrigerated fish.

Mesenteric lymph nodes were collected from several coyotes that died from SPD. Nodes were mixed with normal saline (2 cc/g tissue) and ground in a blender. The resultant suspension was used to treat coyotes, either orally or by intraperitoneal (IP) injection. For oral administration, 25 to 35 cc of the suspension was given by oral gavage followed by 30 cc tap water. For IP administration of the lymph-node suspension, 25 to 35 cc of the suspension were injected with a syringe and 16-gauge needle. Intact mesenteric lymph nodes were fed to other coyotes by wrapping the nodes in a small piece of raw meat. Coyotes treated with lymph material were observed in the same manner as coyotes fed fresh or pre-served fish.

Coyotes used as controls were either fed no fish or, in the case of lymph treatments, were given lymph tissue (orally or IP) from normal coyotes.

RESULTS

Ninety-five coyotes were used in the study (this total excludes coyotes that were used as nontreated controls). The majority (75) received only one treatment while the remainder were used twice, either because they did not eat the fish in their first trial or because they were refed to determine whether they were refractory to SPD. Only 4 of 72 coyotes did not eat fresh fish when it was offered. Usually all of the fish was consumed within 24 hours of feeding.

In preliminary trials to find a source of fish that would cause SPD, 25 coyotes ate fish from eight hatcheries. Ten of the 25 (40%) died. Histological analysis confirmed the presence of rickettsiae in lymph tissue (a finding consistent with SPD-related death) of 5 of the 10 coyotes. Analysis of tissues from four coyotes did not confirm that death resulted from SPD; however, observation of fluke ova in the feces and the appearance of gross signs of SPD (lethargy; anorexia; diarrhea, often hemorrhagic) and death) provided a presumptive diagnosis for SPD (Knapp and Millemann 1970). In addition, mesenteric lymph nodes from canids that die from SPD are usually enlarged (Cordy and Gorham 1950). Tissues from the tenth coyote were not analyzed, but it also displayed signs characteristic of SPD prior to death and had enlarged mesenteric lymph nodes.

Based on the preliminary studies, coho salmon (Oncorhunchus kisutch) from the Siletz Hatchery near Blodgett, Oregon, and steelhead (Salmo gairdneri) from Cedar Creek Hatchery near Hebo, Oregon, were used as the primary treatment for the remainder of the studies. Table 1 lists the species of fish used, sources of the fish, and whether the fish caused SPD when fed to coyotes.

Table 1. Species and source of fish used to produce salmon-poisoning disease (SPD) in captive coyotes.

Species	Hatchery	Caused Mortality
Chinook salmon (Oncorhynchus tshawytscha)	Elokomin Hatchery Cathlamet, Washington	Yes
	Trask Hatchery Tillamook, Oregon	Yes
Chum salmon (Oncorhynchus keta)	John's Creek Hatchery Olympia, Washington	Yes*
Coho salmon (Oncorhynchus kisutch)	Cedar Creek Hatchery Hebo, Oregon	Yes
	Fall Creek Hatchery Alsea, Oregon	Yes*
	Klaskanine Hatchery Astoria, Oregon	No
	Salmon River Hatchery Lincoln City, Oregon	Yes*
	Siletz Hatchery Blodgett, Oregon	Yes
	Trask Hatchery Tillamook, Oregon	Fish not consumed
Steelhead salmon (Salmo gairdneri)	Cedar Creek Hatchery Hebo, Oregon	Yes
Cutthroat trout (Salmo clarki)	Cedar Creek Hatchery Hebo, Oregon	Yes

*Necropsy and pathology did not confirm SPD, but coyote died and displayed signs typical of SPD.

Forty-one coyotes were offered and consumed fresh fish from Siletz and Cedar Creek Hatcheries following completion of the preliminary feeding experiments. Twenty-three of the 41 (56%) died with SPD either confirmed by histological analysis (N = 9) or by observation of signs characteristic of SPD.

Refrigeration at 4°C was the only method of preservation that maintained the properties enabling fish to cause SPD in coyotes. Coyotes that ate fish refrigerated for 3 and 6 weeks died from SPD. Coyotes that ate fish refrigerated for 8 weeks displayed signs characteristic of SPD but did not die. Histological analysis did not confirm the presence of rickettsial bodies in lymph tissue although feces from one of the coyotes contained fluke ova. Fish that were refrigerated for 13 weeks and consumed by coyotes caused no signs of SPD, and fluke ova were not observed in the coyotes' feces. Signs of SPD were not observed in any of the coyotes that consumed frozen or otherwise preserved fish, and fluke ova were not found in their feces.

No signs of SPD were observed in coyotes that received oral doses of SPD-infected or normal lymph material. One of the four coyotes that received SPD lymph suspension IP died, and histology confirmed SPD. Two of the coyotes displayed signs of SPD (one with moderate and one with severe signs) but did not die. The fourth coyote displayed no observable signs of SPD. Histology of two of the three survivors (one with moderate and one with no signs) did not confirm SPD. None of the coyotes given normal lymph material IP displayed signs.

Twelve coyotes that ate fish, displayed signs of SPD, and recovered, were fed fresh fish again from 64 to 360 days later (Table 2). None of the coyotes died, and only four showed mild signs of SPD.

Table 2. Results of feeding infected fish to coyotes after the coyotes had recovered from salmon poisoning disease.

Coyote	Signs after first feeding	Days between first and second feeding	Signs after second feeding
316	moderate*	64	mild
466	mild	64	none
545	mild	64	none
557	moderate	64	mild
572	moderate	64	mild
581	severe	64	none
438	severe	125	none
520	severe	125	none
584	severe	131	mild
458	moderate	189	none
504	severe	189	none
463	severe	360	none

*Mild - 1-3 days diarrhea; moderate - anorexia, 4-7 days diarrhea; severe - anorexia, >8 days diarrhea, >4 days hemorrhagic diarrhea.

There was no observable difference in the severity of signs, the time to death, or in other characteristics between coyotes that received fresh or refrigerated fish or infected lymph material. Therefore, in the remainder of the discussion, the term "treated coyotes" will refer to all coyotes that showed signs of SPD.

Only three of the coyotes (N = 62) that ate fresh fish and could be observed daily failed to display observable signs of disease. The remainder, and coyotes that became sick with SPD from other treatments, displayed signs after a mean of 8 days (SE = 0.3, range 3-14 days). The majority (71%) of the 36 coyotes that died from SPD showed severe signs. The remainder showed moderate signs; however, most of them died before their classification would have been changed to severe (>8 days diarrhea). Coyotes that became ill but survived had symptom rating from 0 to 3.

The mean number of days between treatment and death was 19.8 (SE = 1.4, range 11-47). Only four coyotes died after more than 25 days posttreatment, and 74% of the coyotes died in 21 days or less.

Coyotes that died lost a mean of 32% (SE = 2.2, range 17-58%, N = 25) of their body weight from treatment to death. Most coyotes that were clinically affected but survived lost weight also but to a lesser degree ($\bar{x}$ = 10%, SE = 2, range 2-33%, N = 16). Several coyotes that showed signs and recovered, gained weight ($\bar{x}$ = 8%, SE = 2, range 6-11%, N = 3). Coyotes that were controls or that showed no signs of SPD following treatment either gained weight ($\bar{x}$ = 5%, SE = 1.1, range 2-12%, N = 8) or lost weight ($\bar{x}$ = 3%, SE = 0.6, range 1-6%, N = 8).

A typical progression of SPD in coyotes was as follows; day 8, signs began; days 8 to 20, anorexia, diarrhea (hemorrhagic diarrhea in approximately 50% of the coyotes), lethargy; day 20, death or, for those that recovered, resumption of eating and gradual decrease of other signs.

DISCUSSION

We encountered several difficulties in conducting this research. Probably the most problematic was having confidence that the fish we used were infected with virulent SPD agents. At present the only way to confirm that a fish is appropriately infected is to feed it to a canid and observe the results. If signs of SPD and death occur and if histological analysis reveals the presence of rickettsial bodies, it is logical to conclude that the fish were appropriately infected. However, as was observed in this study, many coyotes survived even though they ate the fish from an infected group. Despite attempts to minimize potential variation among doses of fish tissue (i.e., using pieces of several different fish to make up each dose), there is no way to ensure that all fish were infected.

Another question is how to determine the amount of the rickettsial agents the coyote receives while consuming a portion of the fish. In other words, what is an appropriate dosage? Merely the quantity of

fish or metacercariae consumed may have little bearing on the outcome. We fed a coyote one heart (approximately 13 g) from a coho salmon that was observed to contain metacercariae. The coyote died from SPD in 17 days. In a different study, a coyote ate an entire salmon (approximately 7 kg) that presumably contained the agents for SPD and showed no signs.

The only method that successfully preserved the infective properties of fish was refrigeration. Although a report indicated a domestic dog contracted SPD after eating kippered salmon (Farrell et al. 1968), we were unable to cause SPD in coyotes by similar treatment.

We were interested in determining whether it would be possible to infect a coyote with SPD by feeding it the etiologic agents without the trematode vector. We were unable to find in the literature any discussion of oral administration of rickettsiae without the fluke. Bosnian et al. (1970) documented SPD infection in dogs by exposing them by mechanical aerosolization or rectal administration to infected lymph-node suspensions or rectal mucosa homogenates. Nyberg et al. (1967) produced SPD in dogs using IP injections of ground fluke eggs, and several researchers achieved similar results with IP injection of SPD-infected lymph-node suspensions (Cordy and Gorham 1950, Philip et al. 1954). The suspension apparently contained rickettsiae but not components of the fluke vector. We were able to transmit SPD to one of four coyotes by IP injection, but we were unable to cause SPD in coyotes given an oral dose of intact or ground SPD-infected lymph material. Based on our observations, unless a virulent agent can be injected, it appears that the trematode vector is necessary to provide the rickettsiae access to the circulatory and/or lymphatic system of the recipient animal.

SPD has been described as a disease "almost always fatal" (Knapp and Millemann 1970, Millemann and Knapp 1970), "highly fatal" (Philip et al. 1954), or "usually fatal" (Brown et al. 1972) in canids. A perusal of the literature leads one to believe that dogs and foxes (Vulpes vulpes) rarely survive a bout with SPD. A similar rate of mortality also appeared to be true for coyotes. Donham and Simms (1927) confirmed that mortality in three of four coyotes fed trout containing "living cystic flukes" resulted from SPD. One of the two that were presumably adults and both 8-week-old pups died in 12 to 14 days. Foreyt et al. (1982) observed mortality from SPD in 11 of 12 8-week-old coyote pups given metacercariae from coho and steelhead salmon and cutthroat trout "(Salmo clarki). Death resulted within 15 days.

The highest rate of mortality observed in this study (56%) occurred when adult coyotes were fed fish thought to contain the appropriate agents to cause SPD. This rate is well below the 75% (Donham and Simms 1927) and 92% (Foreyt et al. 1982) rates of mortality observed in other studies. There are several possible reasons for the differences in mortality between studies. First, the coyotes we used may have been more resistant to death from SPD than those used in other studies. Our coyotes and those of Foreyt et al. (1982) originated outside the enzootic area for SPD. Although the origin of the coyotes used by Donham and Simms (1927) is not specified, they likely came from within the enzootic area of SPD somewhere in western Oregon since the study was conducted at Corvallis, Oregon. If a differential resistance to SPD exists, it seems logical that coyotes within the enzootic area would be more resistant than those from without. Based on the scanty literature available, there does not appear to be sufficient evidence to support the theory that coyotes from various locations display a differential sensitivity to SPD.

A second reason for the differential mortality may be that fish from different hatcheries and during different seasons vary in their content of metacercariae infected with SPD agents. We used fish from eight hatcheries in preliminary studies, and two hatcheries were the principal source of most fish used. These hatcheries are located in the heart of the enzootic area for SPD (S. Knapp, pers. comm.). In light of the difficulty in determining the presence or absence of the rickettsiae in fluke metacercariae, the ideal source for fish causing SPD remains in question. In addition, the agents for SPD may occur in different strains or concentrations, perhaps some being more virulent than others (Farrell 1966).

A third reason for the differential rate of SPD mortality observed in coyotes may be age of the coyote. Foreyt et al. (1982) used 8-week-old pups. In later tests Foreyt used older animals and failed to produce a comparably high rate of mortality. In the only other published account detailing the rate of SPD mortality in coyotes (Donham and Simms 1927), two animals, presumably adults, were exposed, and one died from SPD. Both 8-week-old juveniles that were exposed died. It is possible that juveniles, 8-week-old animals, are more susceptible to SPD than adults. Further studies are needed to confirm this hypothesis; however, it appears that adult coyotes are more resistant to SPD than juvenile coyotes and perhaps other canids.

As a method of controlling predators, SPD's most notable benefit is its selectivity and the fact that domestic dogs that become infected can be successfully treated, resulting in lasting immunity. Using SPD would likely have a negligible impact on the environment; and should the method be shown to be effective, it is likely that it could be used economically. Whether the relatively long period of illness (a mean of 12 days from onset of signs to death) is acceptable, is open for debate. Nevertheless, it is doubtful that the rate of SPD-induced mortality observed in this study is sufficient to justify using infected fish (as was used in this study) as a method to remove depredating coyotes from areas of livestock production unless a virulent source of rickettsiae is located. Coyotes that recover from SPD are refractory to subsequent infection (at least for 360 days) and could conceivably use the fish bait as an integral portion of their diet. Most coyotes in this study, including those that recovered from SPD, appeared to relish fish, although this observation may partially be due to the fact that their normal ration was dry dog food. Several coyotes maintained in enclosures appeared to eagerly consume the fish even though other carrion and live prey (rodents, leporids, and birds) were available.

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