Pesticide, PCB, and Heavy Metal Residues in South Carolina Mink

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Abstract: Tissues from 61 mink (Mustela vison) harvested in two areas in South Carolina during the 1987-88 season were screened for pesticide, PCB, and heavy metal residues. Low levels of DDT and DDE were detected. Although 90% of samples contained measurable levels of DDE, the low concentrations found in all but 1 animal should not present any problems to these mink populations. Many (43%) samples contained PCBs, and all samples had detectible heavy metal residues. Levels did not approach those published for mink that suffered mortality in laboratory studies; however, sublethal effects on mink reproduction need to be considered.

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The mink is a commercially valuable furbearer harvested in South Carolina by trappers and hunters. Historical records indicate annual mink harvests reached a high of 11,408 animals in 1938–39 (Novak et al. 1987). Recently, mink harvests have been consistently low, averaging only 250 annually over the last 10 years (S. C. Wildlife and Marine Resour. Dep. 1989).

In a mail survey of sportsmen purchasing mink tags during the 1987–88 trapping season, >40% of the respondents perceived the mink population to be declining (S. C. Wildl. and Mar. Resour. Dep., unpubl. data). Experienced mink trappers have noted that the species is rare or absent from areas of former abundance, although the habitat is seemingly intact. Age structure analysis of the South Carolina mink population revealed noticeably fewer juveniles and yearlings than reported elsewhere, possibly indicating a lower recruitment rate (S. C. Wildl. and Mar. Resour. Dep., unpubl. data). This information, along with a declining harvest, spurred an investigation of factors possibly affecting the mink population in South Carolina.

Environmental pollution has long been recognized as a biological hazard to wildlife as well as to mankind. Industrial pollutants, pesticides, and some heavy metals are known to exhibit biomagnification as they pass up the food chain. The mink occupies a niche at or near the top of the food chain and therefore could be especially vulnerable to these environmental contaminants. The objective of this pilot study was to determine the prevalence of pesticide, PCB, and heavy metal residues in wild mink in South Carolina.

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Methods

Mink carcasses (N = 61) were collected from trappers in South Carolina during the 1987–88 fur harvest season; 58 were from the Piedmont Region of the state and 3 were from the Northern Lower Coastal Plain Region. All mink were sexed and weighed, and canine teeth were collected for aging by cementum analysis. Samples (1 g) of abdominal fat for pesticide/PCB analysis and liver for heavy metal analysis were removed from each carcass, wrapped in aluminum foil, and frozen in plastic bags.

Chemical analysis was performed under contract by the University of Georgia, Riverbend Research Center. Lipids were extracted by gel permeation chromatography (Johnson et al. 1976) and analyzed for residues of 21 pesticides (aldrin, BHC, carbophenothion, chlordane, DDD, DDE, DDT, diazinon, dieldrin, endrin, ethion, heptachlor, heptachlor epoxide, lindane, malathion, methoxychlor, methyl parathion, mirex, parathion, Rabon, and toxaphene) and PCBs by gas chromatography (Bush et al. 1977). Liver tissue was tested for mercury (Clay et al. 1978) and 9 other heavy metals (Mayack et al. 1981, U.S. Environ. Protection Agency 1982). All concentrations are reported on a wet-weight basis. Percent recoveries were 80%. Limits of detectibility ranged from 0.01 ppm to 0.20 ppm for pesticides, 0.05 ppm to 0.08 ppm for mercury, and 0.01 ppm to 0.58 ppm for other heavy metals.

Results and Discussion

Pesticides

No pesticide residues were detected except DDT and 1 of its metabolites, DDE (Table 1). Only 1 sample contained a detectible amount of DDT. However, 90% of mink fat samples contained DDE residues.

Few studies have addressed environmental contaminants in wild mink in the southeastern United States. Analysis of river otters (*Lutra canadensis*) from Georgia in the late 1970s and early 1980s also found high frequencies of DDE residues, but at much greater levels than we found in mink (Clark et al. 1981, Halbrook et al. 1981). Clark et al. (1981) noted a decreasing trend in residues of DDT and its metabolites over time, presumably because of the 1972 ban on DDT use in the United States. Hill and Lovett (1975) found residues of several pesticides including DDT and related compounds in river otter and beaver (*Castor canadensis*) from Alabama. Both species appeared to be abundant and no population effects from the contaminants were apparent. Louisiana river otters were examined to determine

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Tissue and residue	N	%ª	x	Min.	Max.
Fat tissue					
DDT	61	1.7	0.11°		
DDE	61	90.0	0.105	0.01	6.78
PCB	61	43.3	0.420	0.07	2.32
Liver tissue					
Mercury	61	100.0	0.404	0.05	7.55
Silver	55	100.0	0.207	0.04	4.96
Arsenic	55	100.0	0.259	0.06	2.07
Barium	55	100.0	0.053	0.01	22.87
Cadmium	55	100.0	0.044	0.01	0.24
Chromium	55	100.0	0.156	0.01	5.08
Lead	55	100.0	0.242	0.02	4.89
Selenium	55	100.0	0.050	0.01	1.87
Tin	35	100.0	0.029	0.01	0.12
Nickel	55	100.0	0.023	0.01	0.31

Table 1. Residues (ppm, wet weight) of pesticides and PCBs in fat tissue and heavy metals in liver tissue of wild mink harvested in South Carolina, 1987–88.

*Percentage of mink sampled with detectible residues.

^bMeans are geometric; samples with no detectible residues were not included in mean calculations. ^cOnly 1 sample contained a detectible DDT residue.

possible effects of environmental contaminants on reproduction (Fleming et al. 1985). Low levels of DDE and PCBs were found, but there was no significant correlation between contaminants and otter reproductive histories. Low concentrations of DDT and DDE detected in mink in Maryland were not considered a threat to those populations (O'Shea et al. 1981) and DDE residues detected in mink liver tissue in Oregon also were judged to be below harmful levels (Henny et al. 1981). Fat samples from Iowa mink exhibited 6 times higher mean residues of DDT and its metabolites than those we found, but also were not considered high enough to affect reproduction (Franson et al. 1974).

Ranch-raised mink, given food containing 0.42–0.58 ppm DDE under laboratory conditions, exhibited decreased reproductive capability (Gilbert 1969); the fat tissue levels of DDE in these animals ranged from 3.68 to 12.30 ppm. Only 1 South Carolina mink, collected from the Foothills - Mountain Region, exhibited residues in that range (6.78 ppm). With the exception of this animal, mink in the areas sampled in our study did not have levels of DDE and other pesticide residues which might have a limiting effect on mink populations such as that described by Gilbert (1969). However, mink have been shown to be less tolerant of physiological stresses such as extreme cold weather when exposed to pesticide residues (Aulerich et al. 1968), and the combined effects of low levels of several different compounds are unknown (Franson et al. 1974).

PCBs

Many mink fat samples contained detectible residues of PCBs (Table 1). PCB residues reported for river otters in Georgia were generally higher (Clark et al. 1981,

Halbrook et al. 1981). It is interesting that Clark et al. (1981) noted in otters a decreasing trend in pesticide residues and an increasing trend in PCBs. The Louisiana otter study concluded that PCB residues of ≤ 0.65 ppm in liver were not a problem in terms of reproduction (Fleming et al. 1985). However, laboratory studies have shown the European ferret (*Mustela putorius*) to be much less sensitive to PCBs than the mink (Blevines et al. 1980). Harmful residues determined for otters may therefore not be applicable to mink. Mink analyzed from several areas of Oregon had a 22% prevalence of PCB residues (Henny et al. 1981), with several individuals containing levels as high as mink that experienced total reproductive failure in laboratory studies (Platonow and Karstad 1973). Likewise, PCB levels in mink in Maryland were also judged high enough to affect reproduction, even though large-scale PCB contamination was not believed to have occurred in that area (O'Shea et al. 1981).

Laboratory studies have shown that mink are extremely sensitive to PCBs. Adverse effects are reflected in the form of adult mortality, embryo toxicity, or impairment of lactation and growth of the young (Ringer 1981). Platonow and Karstad (1973) conducted experiments in which mink survived long-term feeding of rations containing 0.64 ppm PCBs. However, only 1 of 12 females reproduced, and her kits died shortly after birth. Liver residues in these mink were as low as 0.39 ppm. Other lab studies have demonstrated increased stillbirths, low birth weights, and poor survival of young in mink receiving <1 ppm PCBs in the feed (Aulerich and Ringer 1977, Jenson et al. 1977, Hornshaw et al. 1983).

Whether or not mink in South Carolina are threatened by PCBs is uncertain. Certainly the highest residue we detected (2.32 ppm) does not approach the fat levels found in some mink that experienced reproductive failure in the lab. These values ranged from 13.4 ppm to 86 ppm (Jenson et al. 1977, Hornshaw et al. 1983). Biocontaminant tissue residue levels are difficult to interpret. PCB levels in fat and liver tissue will vary depending on physiological condition of the animal, with liver levels being high following exposure, then decreasing as PCBs are deposited in the fat. Unfortunately, we did not analyze liver tissues for PCBs.

Heavy Metals

All mink liver samples tested contained detectible residues of mercury (Table 1). Although mercury has no known metabolic function, it is a naturally occurring element and small amounts representing background levels are to be expected. Liver mercury residues reported for Louisiana and Georgia otters (Beck 1977, Halbrook 1978) were somewhat higher than those we found in mink. Clark et al. (1981) concluded that mercury levels in otters in Georgia were on the increase. Mink from Massachusetts and Connecticut exhibited mean liver mercury levels 2–3 times higher than those for South Carolina, and it was noted that the animals came from areas with no known mercury pollution (O'Conner and Nielsen 1981).

As with PCBs, the tissue levels of mercury that can be considered harmful are difficult to determine. Sheffy and St. Amant (1982) monitored various Wisconsin furbearers for mercury and considered 1–5 ppm in hair to be normal background

levels, and calculated ratios of fur to liver mercury of 2.5:1. "Normal" concentrations of liver mercury in mink could therefore be considered ≤ 2 ppm. Mink that died of mercury poisoning in lab studies exhibited liver residues on the order of 20–60 ppm (Aulerich et al. 1974, Wobesar et al. 1976, Wobesar and Swift 1976).

Three mink obtained from the Black River Swamp in the Coastal Plain of South Carolina contained liver mercury residues of 5.11, 5.37, and 7.55 ppm. These levels were substantially higher than any detected in other mink sampled, and must be considered greater than normal background levels. The source of mercury in these animals is unknown at this time. It is known that the sandy soils of the Coastal Plain Region allow contaminants to enter the nutrient cycle more readily than in clay soils of the Piedmont (Cumbie and Jenkins 1974, Clark et al. 1981).

Mink liver tissue samples screened for the other heavy metals all exhibited detectible residues (Table 1), most at low levels. However, several individual animals had substantial concentrations of 1 or more metals. Arsenic occurs commonly in air, soil, water, and all living tissues, and background concentrations in animal tissues are generally considered to be <1 ppm fresh weight (Eisler 1988). One sample, obtained from the Central Piedmont Region, had a liver arsenic residue of 2.07 ppm. The same animal also had chromium and lead concentrations of 3.07 ppm and 4.03 ppm, respectively. Eisler (1986) suggested that chromium tissue levels >4 ppm (dry weight) should be considered evidence of contamination. The range of lead residues present in our samples were comparable to those found for mink in Virginia, but our cadmium levels were much lower (Ogle et al. 1985). One South Carolina mink, collected from the Foothills-Mountain Region, contained a liver barium residue of 22.87 ppm. No other samples exceeded 0.20 ppm barium. Barium compounds are used as ingredients in certain rat poisons, and have many other uses in industry and medicine (Food Machinery and Chem. Corp. 1961). While analytical error is possible, we suspect this individual mink may have fed upon a recently poisoned rodent.

Contaminant Combinations

Fish in most rivers and lakes throughout the United States are now contaminated with PCBs (Veith et al. 1979, Jacknow et al. 1986), and dietary levels as low as 0.64 ppm have been shown to seriously impair reproduction in mink (Platonow and Karstad 1973). Likewise, mercury is widespread in the environment, and 1 ppm mercury in the mink diet for as little as 2 months is known to be lethal (Kirk 1971). Smaller doses would likely have sublethal effects on reproduction and behavior.

The current Environmental Protection Agency (EPA) limits for fish deemed safe for consumption is 2 ppm for PCBs and 1 ppm for mercury. Wild mink eating fish containing PCBs and mercury at these levels, or even less, are at risk. Recent lab studies with mink have shown reduced survival in kits born to mink that received both PCBs and mercury simultaneously (Wren et al. 1987b). Over 40% of mink we sampled contained residues of both PCBs and mercury. If other conditions such as food shortages, extreme climatic conditions such as cold weather or drought, or parasite burdens are added, tolerance levels for these contaminants may be very low for a species like the mink (Wren and Stokes 1986, Wren et al. 1987a).

Conclusion

Residues of DDE, PCBs, and heavy metals were detected in mink tissue samples from South Carolina. Residue levels were generally lower than those reported for other wild mustelid populations, and did not approach levels published for mink that suffered mortality in laboratory studies. However, effects of low levels of these contaminants on reproduction and survivability in wild mink is still questionable. Ringer (1981) stated that reproduction in mink would normally not be impaired by chlorinated hydrocarbon pesticides such as DDT at levels typically encountered in the environment. Pesticide residues in general do not seem to be a problem for mink in South Carolina, as 19 of the 21 compounds tested for were not present at detectible levels. Small doses of PCBs and mercury (or other heavy metals), singly or in combination are of concern.

We recommend that future studies on mink in South Carolina focus on obtaining samples of female mink in an effort to determine how environmental contaminants may be affecting reproduction. Only 8 of the 61 mink we collected were females. Also, samples of mink from areas of South Carolina not sampled in this study should be tested, and fish and other aquatic organisms should be collected to determine concentrations of environmental contaminants, especially PCBs and mercury, in mink prey.

Literature Cited

- Aulerich, R. J., R. K. Ringer, R. E. Bostrum, P. J. Scaible and G. R. Hartsough. 1968. Heavy doses of pesticides can kill mink. Prog. Rep. Mink Farmers Res. Found., Milwaukee, Wis. 8pp.
 - —, R. K. Ringer and S. Iwamoto. 1974. Effects of dietary mercury on mink. Arch. Environ. Contam. Toxicol. 2:43–51.
 - ——— and R. K. Ringer. 1977. Current status of PCB toxicity to mink and effect on their reproduction. Arch. Environ. Contam. Toxicol. 6:279–292.
- Beck, D. L. 1977. Pesticide and heavy metal residues in Louisiana river otter. M.S. Thesis. Texas A&M Univ., College Station. 95pp.
- Bleavins, M. R., R. J. Aulerich and R. K. Ringer. 1980. PCBs (Aroclors 1016 and 1242): Effects on survival and reproduction in mink and ferrets. Arch. Environ. Contam. Toxicol. 9:627-635.
- Bush, P. B., J. T. Kiker, R. K. Page, N. H. Booth and O. J. Fletcher. 1977. Effects of graded levels of toxaphene on poultry residue accumulation.
- Clark, J. D., J. H. Jenkins, P. B. Bush and E. B. Moser. 1981. Pollution trends in river otter in Georgia. Proc. Annu. Conf. Southeast. Assoc. Fish and Wildl. Agencies 35:71-79.
- Clay, D. L., I. L. Brisbin, Jr., P. B. Bush and E. E. Prevost. 1978. Patterns of mercury contamination in a wintering waterfowl community. Proc. Annu. Conf. Southeast. Assoc. Fish and Wildl. Agencies 32:309–317.

- Cumbie, P. M. and J. H. Jenkins. 1974. Mercury accumulation in native mammals of the Southeast. Proc. Annu. Conf. Southeast. Assoc. Game and Fish Comm. 28:639–648.
- Eisler, R. 1986. Chromium hazards to fish, wildlife, and invertebrates: a synoptic review. U.S. Fish and Wildl. Serv. Biol. Rep. 85(1.6). 60pp.

1988. Arsenic hazards to fish, wildlife, and invertebrates: a synoptic review. U.S. Fish and Wildl. Serv. Biol. Rep. 85(1.12). 92pp.

- Fleming, J. F., C. M. Bunck, G. Linscombe, N. Kinler and C. J. Stafford. 1985. PCBs, organochlorine pesticides, and reproduction in river otters from Louisiana. Proc. Annu. Conf. Southeast. Assoc. Fish and Wildl. Agencies. 39:337–343.
- Food Machinery and Chemical Corporation. 1961. Barium bibliography: a comprehensive survey of the literature on the applications of barium chemicals. FMC Corp., New York. 433pp.
- Franson, J. C., P. A. Dahm and L. D. Wing. 1974. Chlorinated hydrocarbon residues in adipose, liver and brain samples from Iowa mink. Bul. Environ. Contam. Toxicol. 11:379–385.
- Gilbert, F. F. 1969. Physiological effects of natural DDT residues and metabolites on ranch mink. J. Wildl. Manage. 33:933–943.
- Halbrook, R. S. 1978. Environmental pollutants in the river otter of Georgia. M.S. Thesis. Univ. Ga., Athens. 82pp.
- Halbrook, R. S., J. H. Jenkins, P. B. Bush and N. D. Seabolt. 1981. Selected environmental contaminants in river otters of Georgia and their relationship to the possible decline of otters in North America. Pages 1,752–1,762 in J. A. Chapman and D. Pursley, eds. Vol III, Worldwide Furbearer Conf., Frostburg, Md.
- Henny, C. J., L. J. Blus, S. V. Gregory and C. J. Stafford. 1981. PCBs and organochlorine pesticides in wild mink and river otters from Oregon. Pages 1,763–1,780 in J. A. Chapman and D. Pursley, eds. Vol. III, Worldwide Furbearer Conf., Frostburg, Md.
- Hill, E. P. and J. W. Lovett. 1975. Pesticide residues in beaver and river otter from Alabama. Proc. Annu. Conf. Southeast. Assoc. Game and Fish Comm. 29:365–369.
- Hornshaw, T. C., R. J. Aulerich and H. E. Johnson. 1983. Feeding Great Lakes fish to mink: effects on mink and accumulation and elimination of PCBs by mink. J. Toxicol. Environ. Health. 11:933–946.
- Jacknow, J., J. L. Ludke and N. C. Coon. 1986. Monitoring fish and wildlife for environmental contaminants: The National Contaminant Biomonitoring Program. U.S. Fish and Wildl. Serv. Leaflet 4. 15pp.
- Jenson, S., J. E. Kihlstrom, M. Olsson, C. Lundberg and J. Orberg. 1977. Effects of PCB and DDT on mink during the reproductive season. Ambio 6:239.
- Johnson, L. D., R. H. Waltz, J. P. Ussary and E. Kaiser. 1976. Automated Gel Permeation Chromatographic cleanup of animal and plant extracts for pesticide residue determination. J. Assoc. Off. Anal. Chem. 59:174–187.
- Kirk, R. T. 1971. Fish meal, higher cereal levels perform well. U.S. Fur Rancher 50(10):4– 6.
- Mayack, L. A., P. B. Bush, O. J. Fletcher, R. K. Page and T. T. Fendley. 1981. Tissue residues of dietary cadmium in wood ducks. Arch. Environ. Contam. Toxicol. 637– 645.
- Novak, M., M. E. Obbard, J. G. Jones, R. Newman, A. Booth, A. J. Satterthwaite and G. Linscombe. 1987. Furbearer harvests in North America, 1600–1984. Ontario Trappers Assoc., Ontario, Canada. 270pp.
- O'Conner, D. J. and S. W. Nielsen. 1981. Environmental survey of methylmercury levels

in wild mink and otter from the northeastern United States and experimental pathology of methylmercurialism in the otter. Pages 1,728–1,745 *in* J. A. Chapman and D. Pursley, eds. Worldwide Furbearer Conf., Frostburg, Md.

- Ogle, M.C., P. F. Scanlon, R. L. Kirkpatrick and J. V. Gwynn. 1985. Heavy metal concentrations in tissues of mink in Virginia. Bul. Environ. Contam. Toxicol. 35:29– 37.
- O'Shea, T. J., T. E. Kaiser, G. R. Askins and J. A. Chapman. 1981. PCBs in a wild mink population. Pages 1,746–1,751 in J. A. Chapman and D. Pursley, eds. Vol III, Worldwide Furbearer Conf., Frostburg, Md.
- Platonow, N. S. and L. H. Karstad. 1973. Dietary effects of PCBs on mink. Can. J. Comp. Med. 37:391-400.
- Ringer, R. K. 1981. The effects of environmental contaminants on reproduction in the mink. Pages 232–239 in D. Gilmore and B. Cook, eds. Environmental Factors in Mammal Reproduction. Univ. Park Press, Baltimore.
- Sheffy, T. B. and J. R. St. Amant. 1982. Mercury burdens in furbearers in Wisconsin. J. Wildl. Manage. 46:1117-1120.
- S. C. Wildlife and Marine Resources Department (SCWMRD). 1989. 1988–1989 Commercial Fur Harvest. SCWMRD, Columbia, S.C.
- U.S. Environmental Protection Agency. 1982. Inductively coupled plasma-atomic emission. Spectrometric method for trace element analysis of water and wastes-Method 200.7. Environmental Monitoring and Support Laboratory, Cincinnati, Oh. 298pp.
- Veith, G. D., D. W. Kuehl, E. N. Leonard, F. A. Puglisi and H. E. Lemke. 1979. PCBs and other organic chemical residues in fish from major watersheds of the United States, 1976. Pestic. Monit. J. 13:1–11.
- Wobesar, G. and M. Swift. 1976. Mercury poisoning in a wild mink. J. Wildl. Dis. 12:335-340.
- Wobesar, G., N. O. Nielsen and B. Shiefer. 1976. Mercury and mink II: experimental methylmercury intoxication. Can. J. Comp. Med. 40:34–35.
- Wren, C. D. and P. M. Stokes. 1986. Mercury levels in Ontario mink and otter relative to food levels and environmental acidification. Can. J. Zool. 64:2,854–2,859.
 - , D. B. Hunter, J. F. Leatherland and P. M. Stokes. 1987a. The effects of PCBs and methylmercury, singly and in combination, on mink. II: Reproduction and kit development. Arch. Environ. Contam. Toxicol. 16:441–454.

^{----, ----, -----,} and ------. 1987b. The effects of PCBs and methylmercury, singly and in combination, on mink. I: Uptake and toxic responses. Arch. Environ. Contam. Toxicol. 16:441-447.