Gill net changes can prevent bird drownings

A test of modified fishing nets has revealed ways to make gill nets friendlier to seabirds, reducing the number that get entangled underwater and drown, according to Washington scientists.

These efforts represent the first fix for protecting birds from gill nets without closing a fishery, says Ed Melvin of the Washington Sea Grant Program in Seattle.

Other gear, such as trawls, have devices to protect wildlife, but technology has lagged for the gill net, Melvin points out. In the December CONSERVATION BIOLOGY, he and his colleagues describe modifying gill nets used to catch sockeye salmon in Puget Sound.

Full-size gill nets stretch 1,800 feet in length and dangle 60 ft deep. Their monofilament line, hard to see underwater, snags birds when they dive.

Previous bird-saving experiments haven't worked out well, Melvin notes. Some Japanese boats fishing for flying squid sank nets about 2 yards below the water's surface. Fewer birds died, but the squid catch shrank to as little as 5 percent.

In Washington State, concern about bird entanglement rose in 1992 after the marbled murrelet, a bird that forages at sea, was listed by the federal goverment as a threatened species, explains Jon Anderson of the Washington Department of Fish and Wildlife in Olympia. Observers on salmon boats in 1994 saw only one murrelet entangled but recorded 3,500 other birds snared, mostly common murres and rhinoceros auklets.

"Some by-catch problems can be solved if you sit down and listen to people," Melvin says. Talks with fishing crews led to the tests reported this week. Two boats replaced the top 7 ft of their nets with white, multistrand mesh. Two other boats replaced the top 15 ft.

Both white-topped nets reduced murre entanglement by about 40 percent. The auklet deaths decreased similarly, but only with the net topped by the wider white band. Unfortunately, that net also cut salmon take by more than half.

Another two boats put pingers on nets to warn birds. "They sound a whole lot like those annoying beepers on watches you hear in movie theaters," Melvin says. The pingers halved the accidental murre deaths but seemed to attract seals, as if a dinner bell signaled free salmon.

Even before the scientific paper was published, Washington fisheries managers ordered the salmon boats they regulate to top their gill nets with 7 ft of white mesh and not to set their nets during the rhinoceros auklets' busy feeding time at dawn. Melvin's team had found that this time limit would reduce auklet entanglements by 60 percent.

The regulations do not apply to tribal fishing boats. The nontribal fleet protested the new regulations at first, but a court ordered them into effect.


Melvin

White mesh reduces bird entanglements.

"You couldn't argue the science," comments John Grettenberger of the U.S. Fish and Wildlife Service in Lacey, Wash. He says the study's solid design and its involvement of fishing crews eased the regulatory process.

Canada has not adopted similar rules for its fleet that takes sockeye from the same run, as the fish head north to Canada's Fraser River. Melvin estimates that in 1996, tribal and Canadian fleets caught 99 percent of the sockeye. Yet there are no data on bird by-catch in Canada.

"We can't blithely say we don't have a problem because you can't say that until you've looked," acknowledges Ken Morgan of the Canadian Wildlife Service in Sidney, British Columbia. He's starting to train observers to identify bird by-catch when monitoring boats for other purposes. "We're really years behind what Ed has done," he laments. —*S. Milius*

Gene may alter Ritalin's effects in ADHD

Many children diagnosed with attention-deficit hyperactivity disorder (ADHD) calm down and otherwise improve when given the stimulant methylphenidate, or Ritalin. Others derive no benefit from the same medication.

Reasons for this variation in methylphenidate's impact on ADHD remain unclear. A new study, however, suggests that the drug fails to help youngsters who have inherited a specific form of a gene involved in dopamine transmission in the brain.

This finding stems from an analysis of 30 African-American boys, ages 7 to 11, who exhibited moderate-to-severe ADHD symptoms. The work sets the stage for larger genetic studies of methylphenidate response, say Bertrand G. Winsberg of Brookdale University Hospital and Medical Center in New York and David E. Comings of City of Hope National Medical Center in Duarte, Calif.

If the psychiatrists' linkage of this particular gene variant to poor methylphenidate response holds up, it may provide guidance to mental health clinicians faced with difficult decisions about prescribing drugs to children with ADHD (SN: 11/28/98, p. 343).

Of 14 boys with ADHD who showed little or no improvement after taking methylphenidate pills for 3 months, 12 had inherited a long form of the dopamine transporter gene (*DAT1*) from both parents, the researchers report in the December JOURNAL OF THE AMERICAN ACADEMY OF CHILD AND ADOLESCENT PSYCHIATRY. This version of *DAT1* contains 10 copies of a particular amino acid sequence; other forms have five, eight, or nine copies.

In contrast, only 5 of 16 boys with ADHD who benefited from medication had inherited copies of the long *DAT1* version from both parents.

Several teams have previously linked the gene *DAT1* to ADHD, but not to children's responses to stimulants used to treat the condition.

Researchers know little about the functions of *DAT1* and its various forms. Mice engineered to lack this gene become hyperactive. Stimulants, which act directly on dopamine-transporter molecules, nonetheless calm these genetically engineered mice. They are ineffective, however, if the mice also receive a drug that enhances the availability of another neurotransmitter, serotonin.

The new link between methylphenidate and *DAT1* underscores the complexity of genetic influences on individual development, remarks psychiatrist Edwin H. Cook Jr. of the University of Chicago in a commentary published in the same journal. Further exploration of the properties of different forms of the *DAT1* gene may open new treatment options for ADHD, Cook contends.

Variants of two genes for brain receptors for dopamine, known as *DRD2* and *DRD4*, didn't appear to affect youngsters' responses to methylphenidate in Winsberg and Comings' study. Other investigations have implicated versions of these genes in ADHD, the researchers say.

Genetic research with larger, ethnically diverse groups of children diagnosed with ADHD may find that certain forms of *DRD2* and *DRD4* help or hinder methylphenidate's effectiveness, Cook says.

Winsberg and Comings have essentially launched the study of the relationship of genetic variation to the medication responses of children with psychiatric disorders, he adds.

"The reality is that whether any individual child with ADHD will respond to stimulants is largely an issue of luck because we have not measured all of the reasons for variability in response," Cook concludes. —*B. Bower*