He sings Dad's songs; she sings Mom's

Like some mammalian musicians, stripe-backed wrens pass down song repertoires within families. Among these tropical wrens, the brothers sing like their father, as distinct from unrelated neighborhood guys, while the sisters sound just like Mom.

These calls are somewhat like human name systems that give clues to paternal and maternal lines of descent, says J. Jordan Price of the University of North Carolina at Chapel Hill. His wren study is the first to document his-and-hers vocal traditions within a family, he adds, but as researchers trace song styles in more tropical bird species, the system may turn out not to be unique.

In many birds of the temperate zones, a young male picks up songs from his new neighbors when he leaves home and establishes a territory, Price says. Among these species, most females don't sing. Females of tropical species are likely to sing, but research on song origins there has lagged behind work on temperate species.

Price's investigation of song patterns grew out of a long-running study that had already documented the genealogy of stripe-backed wrens on a cattle ranch in the llanos, or seasonally flooded savanna, of northern Venezuela. The birds breed cooperatively in groups of up to 14, with adult offspring helping to raise later years' broods. Only the principal female of a group lays eggs, but the whole gang pitches in to build the nest, defend the boundaries, and collect insects for the hungry babies.

One of the males eventually inherits the territory from his father, and most of the brothers in the line of descent stay in the territory, as if waiting for a chance to take over. "It's like the British royal family," Price says.

The sisters are more likely to leave, rushing to a neighboring territory when its reigning female dies or disappears. Claw-to-claw combat determines which female becomes the new top bird. The losers go home to mother and wait for the next vacancy.

Price admits that the calls of these wrens are "not pretty." The main breeding male and female in a territory sing a duet that he calls a "harsh, raspy thing." For the vocal traditions study, he analyzed another call—twisty sounds nicknamed WAYs because they reminded early observers of a distorted human voice whining, "Where are you?" One of their functions, Price speculates, is to help birds keep in touch in dense foliage.

Price taped a total of about 10,000 of these WAY calls from 46 principal males, 22 male helpers, and 14 females. The calls clearly descended along family lines, passed from male to male and female to female, Price reports in the March 22 PROCEEDINGS OF THE ROYAL SOCIETY OF LONDON B. In two cases, he found nearly identical calls in males whose territories lay about a kilometer apart and then discovered that they had shared a great-grandfather 15 years ago.

"I'd be very surprised if the songs weren't learned," Price says. He bases his idea on the complexity of the wren's repertoire. Studies of birds raised in laboratories with different song environments have demonstrated that other species with complicated songs learn their repertoires.

Price's research seems "an important first step" to Rachel Levin of Pomona College in Claremont, Calif. What's needed now, she says, is more analyses of tropical birds.

Levin studies song in the bay wren, another tropical species whose males and females sing duets. "I'm working with extremely precise 'whopdiddly whopdiddly whopdiddly' [calls], where one sex is whopping and the other is diddlying," she explains. Such two-sex song patterns pose complex questions about learning and about how males and females end up with the right parts of the song. —*S. Milius*

AIDS virus may feast on an unexpected meal

HIV is picky about its prey. The AIDS virus attacks only immune cells—and only those with a surface protein called CD4. The virus' preference stems from its need to bind to CD4 before infecting cells.

The discriminating palate of the AIDS virus has been thought to render the other major class of immune cells, which displays a protein called CD8, but not CD4, virtually invulnerable to infection. Three research groups now challenge that dogma with evidence showing that under certain conditions, CD8 cells can make the CD4 protein and consequently become infected by the AIDS virus.

In the body, CD8 cells can transform themselves into killers that seek out and destroy other cells infected by HIV. The virus' unexpected access to CD8 cells may impair these killers, thus speeding the development of AIDS, investigators speculate.

"One envisions that these cells get infected [with HIV] as soon as they start to respond [to it]," says Jerome Zack of the University of California, Los Angeles. "That would be a really smart way for the virus to elude the immune system."

While most scientists have focused on the declining number of CD4 cells that follows untreated HIV infection, others have documented that CD8 cell counts also fall over time, especially late in an infection. Moreover, several recent studies of people with AIDS have shown that their CD8 cells frequently contain HIV genes, evidence that those cells were infected.

Since CD8 cells weren't known to make CD4, a prerequisite for infection by the virus, the puzzling results went unexplained and to some extent ignored.

Now, Robert C. Gallo of the University of Maryland's Institute of Human Virology in Baltimore and his colleagues report that when CD8 cells are stimulated out of their quiescent state, as they would be when responding to an infection, they make CD4.

"This gives a mechanism [for CD8 cell infection]. People now have to pay attention," says Gallo, whose group reports its findings in the March 17 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES.

"Now that there are three labs finding this, I think people are going to believe it," agrees Carl June of the Naval Medical Research Institute in Bethesda, Md. Zack and June presented data at a recent AIDS meeting showing that stimulated CD8 cells produce CD4 and thus become vulnerable to HIV infection. June's group will publish its findings in the April JOURNAL OF EXPERIMENTAL MEDICINE.

June's and Zack's groups have observed that HIV infects naive CD8 cells—those never before exposed to an infection—more efficiently than CD8 cells that have previously been called into action. Naive cells can make CD4, but the veteran cells cannot, says June.

Since most CD8 cells in infants are naive, the fondness of the AIDS virus for those immune cells "may be one reason why children with an HIV infection progress so much faster than adults," notes Zack.

While all three groups have shown that CD8 cells grown in test tubes or obtained directly from tissues or blood can make CD4 and become infected by HIV, none has yet found that this phenomenon impairs the function of the cells in the body. In test tubes, the HIV-infected CD8 cells die prematurely, says Gallo.

"The key question is whether the infection of CD8 cells causes a decline in cytotoxic function," says Peter Simmonds of the University of Edinburgh, who had previously found that CD8 cells in people can be infected by HIV. "If you can show [the CD8 class] is involved, that clearly makes a big difference in trying to understand the immune suppression that AIDS patients have."

Why would CD8 cells begin to make CD4 when stimulated? Zack speculates that the protein, known to mediate interactions between cells, may help CD8 cells make contact with the class of immune cells that teaches them how to identify HIV-infected cells. —*J. Travis*