

into each of the catalyst's carbon rings. To each boron they then attached oxygen atoms linked to other small hydrocarbon groups. Borons are themselves electron-hungry, so they try to swipe electrons from neighboring atoms. Oxygen is reluctant to give up any electrons, so the borons end up snatching them from the zirconium, leaving it even more starved of electrons. "The zirconium atom needs to do something to compensate," says Bazan, and so it tries even harder to swipe a hydrogen from the growing polymer chain. It usually succeeds quickly, causing the polymer chain to break off almost as soon as it starts growing, thereby creating the short-chain α -olefin. "That's something no other metallocenes

have done thus far," says Bazan.

The Rochester team believes that it may also be able to fine-tune the catalyst to produce α -olefins of a particular size. Changing the organic groups linked to the catalyst's oxygen atoms should change the electronic state of the zirconium atom and influence which α -olefins it produces. "It's a pretty unique system that you can tune the electronics," says Kemp. Bazan says he hopes to tune the catalyst to make just large α -olefins consisting of 10 or 12 ethylene units, which are currently expensive to produce.

The new catalyst still has a few hurdles to overcome before it gets to the market. For a start, although current-generation catalysts are less efficient at producing

α -olefins, they are "dirt cheap," says Bazan. The new metallocenes, however, require several complicated and expensive synthesis steps just to insert boron atoms into the all-carbon rings. Also, Maurice Brookhart, a catalysis expert at the University of North Carolina, Chapel Hill, notes that the boron-based catalyst is slow compared to conventional catalysts. But Kemp believes that tinkering with the reactor conditions will likely improve its speed. "There's almost always room for optimizing a catalyst," he says. If that proves true in this case, metallocene catalysts may soon find themselves in command of a whole new market.

—Robert F. Service

EVOLUTIONARY BIOLOGY

Males Mutate More, Bird Study Shows

You can't choose your parents. But if you could, you might want to pick a father who is young. For the last 90 years, geneticists, including the illustrious J.B.S. Haldane, have noted that children of older fathers tend to suffer more from genetic diseases. The standard explanation has been that fathers pass on more genetic mutations because their sperm-producing cells divide throughout their lifetimes—as many as 400 times in a 30-year-old man. This provides many more opportunities for mistakes to occur as the DNA copies itself than in egg cells, which only divide about 24 times. The notion has been hard to prove directly. But in this month's *Nature Genetics*, a pair of Swedish researchers present evidence that in a father, age can have a genetic cost.

By analyzing a gene found on both the male and female sex chromosomes of birds, evolutionary geneticist Hans Ellegren of the Swedish University of Agricultural Sciences in Uppsala and his graduate student Anna-Karin Fridolfsson showed that mutation rates really are higher in males. "These data show that male-driven mutation appears to be a general phenomenon" in both mammals and birds, says population geneticist Brian Charlesworth of the University of Edinburgh in the United Kingdom. Not only does the result support the idea that older males are a source of the mutations that lead to genetic disease, but it also suggests that males have more input into evolutionary change than females do.

The Uppsala team turned to birds in order to avoid a problem that has confounded efforts to resolve the issue of why males have higher mutation rates. The most obvious way to determine the relative mutation rates in the two sexes is to look at genes found on the sex-

determining X and Y chromosomes. But there are fewer Y chromosomes in the population overall (only one Y for every three X's), and in population genetics, a decrease in population size renders natural selection less effective. Consequently, the likelihood that deleterious mutations on the Y will slip through to the next generation is greater than the corresponding chance for mutations in genes on the X.

But in birds, it's the females that have the mismatched sex chromosomes, designated "W" and "Z," while males have two "Z's." So in birds, the chromosome arrangement should drive down the apparent mutation rate in the



Flighty genes. A new finding indicates that a male collared flycatcher (right) passes on more mutations to his offspring than a female does.

male chromosome—the Z. If the male mutation rate is still higher, the elevated rate is likely to be a real effect. "Using birds is a really nice way of sorting this out," says evolutionary biologist Linda Partridge of University College, London.

Before the Uppsala researchers could do the experiment, they needed a gene found on both bird sex chromosomes. Such a gene did not turn up until 1996, when it was shown that the CHD gene, which makes a protein involved in gene control, occurs on both the W and Z chromosomes. "Suddenly," Ellegren recalls, "we could test the hypothesis" that mutation rates are higher among males. And that's what they found.

The Ellegren team sequenced the CHD gene from males and females of birds, includ-

ing warblers, flycatchers, and yellowhammers. Applying a statistical method for analyzing mutation rates, they then showed that the gene accumulated mutations in males as much as 6.5 times faster than in females.

Charlesworth cautions, however, that more work will be needed to verify the Ellegren team's conclusion. Indeed, other recent findings have "muddied the waters," Partridge says. In the 27 March issue of *Nature*, Gilean McVean of Cambridge University and Laurence Hurst of the University of Bath, both in the United Kingdom, reported that genes on the X chromosomes of mice and rats have significantly lower mutation rates than genes on the nonsex chromosomes. That finding suggests that the apparently lower female mutation rate the Swedish researchers found could be just a peculiarity of the female sex chromosome.

But Ellegren notes that that is not necessarily the case. He points out that if McVean and Hurst's finding about a lower mutation rate on the X chromosome applies to the Z chromosome of birds, it would tend to counteract the mutation-enhancing effect

of more DNA replication in males, as males have two Zs. "In spite of [the expected reduction in male mutation rates], we found the male bias," says Ellegren. Charlesworth suggests that the Swedish researchers now need to compare the mutation rates of the CHD and other Z-linked genes to those on nonsex chromosomes in birds to verify that the excess mutations arise in other genes as well—a task on which Ellegren's laboratory is working. Already, though, the new results should tell baby boomers and birds alike to beware: There may be unintended consequences to siring a family when you are a senior citizen.

—Steven Dickman

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