Parenting frays some feathered friends

Tired human parents who catch every illness going around aren't alone in the animal kingdom. Because of their heavy family responsibilities, certain birds that have large broods to feed fall prey to passing protozoa more readily than their compatriots with smaller families, a new study finds.

This news may comfort, though not surprise, human parents. However, it does counter ornithologists' observations that sickly birds plagued by parasites tend to have fewer offspring.

There's a surprise for nonornithologists too. Only the male parents of large broods have a higher rate of illness than other bird parents, because the males take on more of the chick-care responsibilities, report Heinz Richner of the University of Berne in Switzerland and his colleagues.

Previous studies of mammals and birds have shown that producing a large brood or taking on more of the feeding burden can affect future fecundity, perhaps even survival.

The Swiss researchers suggest for the first time that heavy parenting demands may make birds more susceptible to protozoa, resulting in future fertility problems, says David R. Winkler of Cornell University. "I hope it serves to get more of us ornithologists more interested in parasites."

Philippe Christe

A female great tit.

In addition, he says, researchers rarely do this kind of experimental — as opposed to observational — study of birds.

In their experiments, Richner and his colleagues monitored the health of 118 great tits *(Parus major)* nesting near Lausanne after manipulating the birds' brood sizes, they report in the Feb. 14 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES. The researchers moved enough pairs of nestlings to create similar numbers of diminished, enlarged, and untouched families.

When the chicks reached 13 days old, the age at which they require the most food from their parents, the investigators photographed the nests to find out how often the parents brought food. They then captured and tested the parents for malaria, a common infection in birds.

The males with enlarged broods made about 30 food deliveries per hour — 50 percent more than any of the other birds, including their female partners.

In a recent, unpublished experiment, the team also found that chicks with the worst infestations of blood-sucking fleas beg for food more often than their less heavily infested peers. In response, the males feed them more. The females, however, don't bother.

Males may work harder than females to ensure that their offspring survive, because males outnumber females and may not have as many opportunities to reproduce, Richner speculates. Females may fare better in the long run by not putting all of their energy into one nest.

The busy males pay a price for their efforts, the researchers assert. More than 75 percent of the fathers of the bigger broods became infected with the protozoan that causes malaria — that's twice the infection rate of the other males. Most of the females got off malariafree.

The scientists suggest that the added feedings tax the birds' energy and weaken their immune systems. Alternatively, the birds' schedules may somehow expose them to more of the protozoa.

In an earlier study, Richner and his colleagues found that about one-third of the males in 108 unmanipulated broods had malaria, twice the infection rate the researchers found among the females. A much smaller proportion of infected than uninfected males returned to breed, they report. — *T. Adler*

Philippe Christe

Nestlings calling for dinner.

Honing new weapon to counteract HIV

By teasing apart how a promising class of anti-AIDS drugs hinders the disease, researchers may have found a way to make the drugs more effective.

"The idea behind this analysis is important," says Stephen Hughes, an AIDS researcher at the National Cancer Institute in Frederick, Md. "We don't understand enough about HIV replication," he says of a new study conducted by biochemists Kenneth A. Johnson and Rebecca A. Spence of Pennsylvania State University in University Park and their colleagues.

HIV, the AIDS virus, enters human cells equipped with its own RNA, a template for making DNA. The virus manufactures DNA from this RNA using an enzyme called reverse transcriptase. It then inserts its DNA into that of the host cells, converting them into HIV factories.

AIDS drugs such as AZT and ddC trick the machinery of reverse transcriptase. They bind to a cleft on the enzyme that normally locks onto a nucleoside, one of the building blocks of DNA.

Unfortunately, these nucleoside-mimicking drugs can lead to serious side effects in patients. What's more, HIV mutates, churning out forms of reverse transcriptase that evade the drugs.

A new kind of medication now in clinical trials blocks reverse transcriptase by attaching not at its nucleoside binding site, but in a different spot. These so-called non-nucleoside inhibitors, including Nevirapine, cause fewer side effects than AZT because they're more specific.

The Penn State group halted early stages of DNA reactions in order to unravel the mechanism of three of these inhibitors. They found that the drugs don't stop the enzyme from binding to its target nucleoside.

Instead, they report in the Feb. 17 SCIENCE, the drugs appear to make the enzyme latch onto the nucleoside in a skewed position, hampering the chemical reaction by which the nucleoside attaches to other molecules. Indeed, the inhibitor makes the enzyme bind the nucleoside more tightly.

This supports a new strategy, Johnson says: Join the inhibitor and AZT or a related drug in a single molecule.

"This could give you something with a very high degree of specificity, lower toxicity, and it might be very effective," he says. By acting at two sites, such a drug might also stymie HIV's attempts to evolve a resistant form of reverse transcriptase, he adds.

The problem tackled by the Penn State team involves "horribly difficult analysis," Hughes notes. But "if [the analysis] is right, it's interesting and important." — *J. Kaiser*