Monkeys Defy Crowding-Aggression Link

For more than 30 years, an influential theory has held that crowding brings out beastly behavior in people and many other animals. But the largest primate study of crowding to date finds that rhesus monkeys maintain a remarkably stable level of aggression across a spectrum of population densities, from confinement in small pens to open living on a small island.

Although generally considered the most fight-prone of monkeys, rhesus individuals reacted to crowded conditions with a surge of "coping" behaviors that kept aggression in check, assert psychologist Peter G. Judge and ethologist Frans B.M. de Waal, both of the Yerkes Regional Primate Research Center in Atlanta. Coping responses included grooming each other, making submissive gestures, avoiding dominant animals, and huddling with relatives after a dispute rather than starting a fight.

"Rhesus are the primate species we would least expect to display effective coping responses under high population density," says de Waal. "The human capacity to adapt to crowding has evolutionary parallels in other primates."

The new findings, submitted for publication, contrast with the tendency of overcrowded mice and rats to attack one another and rapidly die out (SN: 5/31/86, p.346). In close quarters, rodents may rage, but primates try to keep their cool, within the limits of species-typical behavior, Judge and de Waal argue.

The researchers studied nine social groups, containing 413 monkeys, that had lived for at least several years in small indoor pens, in medium-size indoor-outdoor cages, in large outdoor corrals, or on a small island off the coast of South Carolina. Detailed observations of interactions with other group members were gathered for 145 adult males and females in the nine groups.

The frequency of aggressive acts rose slightly in more congested quarters; openly hostile behavior occurred 1.5 times as often in indoor pens as on the island. Yet that disparity was quite small considering that island monkeys had 6,000 times more available space than those restricted to pens, Judge says.

In contrast, crowded conditions brought out large increases in coping behaviors. For instance, monkeys took steps to defuse potential fights more than twice as often in indoor pens as in any of the other living situations.

In previous research, the Yerkes scientists found that when rhesus monkeys are moved suddenly to a much smaller living area, they tend to huddle together with relatives and stay still. The researchers compare this to the tendency of people to avoid eye contact or talking on a crowded subway or elevator.

Yerkes Regional Primate Res. Ctr.

Rhesus monkeys cope adeptly with crowding.

Rhesus monkeys housed with more conciliatory, stub-tailed monkeys also show boosts in coping behaviors, de Waal says.

This category of responses, as well as social "pecking orders" that influence access to food and other resources, may have evolved to rein in aggression and violence in primate groups, he contends.

In a related study, psychologists find that the willingness of city dwellers to help strangers in a variety of ways increases as population density (the number of people per square mile) drops. Population size shows a much weaker link to helpful responses, they contend.

High population density may contribute to sensory overload and a tendency to avoid others' requests, suggest Robert V. Levine of California State University, Fresno, and his coworkers. Larger groups of bystanders may also lessen the sense of individual responsibility toward strangers, they argue.

Experimenters presented six "helping scenarios" to pedestrians in 36 U.S. cities. The situations included asking for change for a quarter and pretending to be unable to pick up a pile of magazines because of an injured leg.

Greater helping tended to occur in cities with lower violent crime rates, the scientists report in the July JOURNAL OF PERSONALITY AND SOCIAL PSYCHOLOGY. Widespread violence may lead to avoidance of direct confrontations with strangers, they contend. Or, as Judge and de Waal might put it, human coping behavior includes avoiding strangers in threatening situations. *—B. Bower*

Fine-tuning gene action in engineered mice

For several years, scientists have been able to "knock out" specific genes and create strains of mice that mimic certain disease conditions (SN: 9/4/93, p.148). To do this, geneticists inactivate a gene in very early embryo cells, then add those cells back to developing mouse embryos. The gene is then missing or not functional in offspring of the mice that develop from these altered embryos.

Now, a multinational research team has fine-tuned this type of genetic engineering. A new technique enables scientists to create mice that lack a particular gene at certain stages of development or in certain tissues, says Hua Gu, a geneticist now at a Rockville, Md., laboratory of the National Institute of Allergy and Infectious Diseases.

"[The procedure] opens up entirely new avenues in scientific research that have never been possible before," says Jamey D. Marth of the University of British Columbia in Vancouver. "Every lab that tries to generate... human disease models will use this."

While working with Klaus Rajewsky at the University of Cologne in Germany, Gu and his colleagues wanted to know how an enzyme called polymerase beta affected the function and development of white cells. They couldn't just knock out the gene for this enzyme, because when they did, the mouse embryos died.

So instead, they borrowed genetic material from a bacterial virus, or phage. Some of that material codes for an enzyme, Cre recombinase, that keeps copies of viral DNA separated from each other, enabling them to infiltrate the bacterium's genetic material more easily. This enzyme homes in on a particular bit of DNA consisting of 34 base pairs. Then it snips out that bit and any genes that follow, stopping just short of the next copy of the 34-base-pair bit.

To create their new mouse strain, the scientists started with genetically altered parent strains. One parent strain carried functional polymerase beta genes sandwiched between two 34-base-pair bits. The other made Cre recombinase.

Marth and Paul C. Orban had created that second strain by joining the Cre recombinase gene to a "promoter" gene, which controls the activation of other genes. That promoter worked only in white cells called T cells, so only they made Cre recombinase.

Consequently, mice born after mating these two strains made Cre recombinase enzyme in their T cells. That enzyme then recognized the modified gene for polymerase beta and cut it out of the T cells' DNA, disabling it. The scientists then observed the effect of this loss, they report in the July 1 SCIENCE. *—E. Pennisi*