Biology

Tina Adler reports from Providence, R.I., at the annual meeting of the Ecological Society of America

Saving cheetahs: Adults come first . . .

Cheetah populations worldwide are declining, and researchers continue to debate whether to blame genetic or environmental factors.

While some scientists argue that this speedy creature is quickly inbreeding its way to extinction, other investigators disagree (SN: 9/25/93, p. 200). They say predation—particularly of the young—has caused the animal's population woes. Lions and hyenas kill about 75 percent of the wild cubs.

Polarization around the cheetah controversy has escalated, yet rigorous quantitative analysis has not established the role of either predation or genetics in cheetah demography, says biologist Kevin R. Crooks.

So Crooks and his coworkers at the University of California, Santa Cruz, developed a computer model to predict what would happen to the cheetah population if the animals reproduced with varying degrees of success and died at certain ages and rates. The researchers used rates ranging from the average observed in nature to extreme possibilities.

In a variety of scenarios, including the complete prevention of predation, survival of adults—not of cubs—had by far the largest influence on population growth, they assert. Adults are key, because after losing a litter, females produce another within a year.

Crooks

Protecting cheetahs by focusing on safeguarding the infants, as some scientists have recommended, would therefore prove not only difficult and expensive, but also unsuccessful, the Santa Cruz team predicts.

A cheetah at the San Diego Wild Animal Park in California.

. . . and gophers offer valuable clues

The idea that cheetah populations are dangerously inbred comes in part from experiments showing that unrelated cheetahs can accept skin grafts from each other. Their immune systems don't reject the grafts, because the animals are so genetically similar. Critics, however, have argued that these grafting experiments suffered from many methodological shortcomings, casting doubt on the genetic similarity of cheetahs.

A new report confirms that in some natural populations, individuals from different families can be sufficiently similar to accept skin grafts from each other. It also sheds light on the ability of genetically matched populations to survive in the wild, despite their uniform immune systems, assert M.A. Sanjayan and his colleagues at the University of California, Santa Cruz. Their report has been accepted for publication in CONSERVATION BIOLOGY.

The team tested how well 44 members of three genetically dissimilar, well-established pocket-gopher populations accepted skin grafts from animals in their own group.

Two of the groups were highly inbred, and their members were the only animals to accept grafts from each other, the team found. However, the inbred individuals readily rejected skin taken from gophers outside of their population, which demonstrated that their immune systems were functioning reasonably well.

Although the individual gophers' immune systems responded normally, the inbred animals had very similar genes that encode immune-system proteins. Inbred groups, whether gophers or cheetahs, therefore may risk losing many members to a single infectious agent, the team notes.

Biomedicine

A risky cure for sickle cell disease

Like 18-wheelers unable to navigate sharply curved roads or pass under low bridges, the misshapen red blood cells of a person with sickle cell disease cannot travel easily through blood vessels of the body. For some, this means years of swollen limbs and severe pain. For others, it means an early death, as vital organs suffer irreparable damage. A drug, hydroxyurea, can ease symptoms but provides no cure.

Bone marrow transplants, long used to cure people with leukemia and other cancers of the blood, offer children with sickle cell disease a fighting chance to be free of their illness, report Mark C. Walters of Fred Hutchinson Cancer Research Center in Seattle and his colleagues in the Aug. 8 NEW ENGLAND JOURNAL OF MEDICINE. After receiving transplants of bone marrow from a sibling, 16 of 22 children with severe sickle cell disease were cured, the researchers found. The donor bone marrow contains stem cells that can supply healthy red blood cells to replace the defective ones made by the original marrow.

The expensive treatment, first attempted for sickle cell disease in 1984, does not guarantee a cure and is not without risks. Four of the children in the recent study rejected the donor bone marrow. Another two died from complications of the transplant, in which the patient's own bone marrow must be destroyed with chemotherapy.

Until the risks of bone marrow transplants fall, parents and physicians will have difficulty deciding whether the procedure is worth the gamble. "The disease is extremely variable... We're not able to identify those individuals who are at risk for early death," notes Helena O. Mishoe of the National Heart, Lung, and Blood Institute in Bethesda, Md.

Lose a gene, lose some weight—in mice

Don't you just hate people who eat tons of ice cream and unlimited pizza but remain svelte? G. Stanley McKnight has equally annoying mice in his laboratory at the University of Washington School of Medicine in Seattle. McKnight's mice, which have been genetically engineered and bred to lack a specific gene, recently went on a 58 percent fat diet for 4 months and still stayed trim and fit.

The missing mouse gene encodes an enzyme subunit, called RII-beta. The enzyme, PKA, comes in several versions, and the one with RII-beta is largely made by brain and fat cells. Mice deprived of their RII-beta gene are forced to substitute another subunit, RI-alpha, to construct the PKA enzyme.

That simple switch has a dramatic effect. Compared to RII-beta, RI-alpha binds more easily to certain signaling molecules, so PKA is more readily activated in the mice. The enzyme's increased activity, in turn, sets off a biochemical cascade that ultimately stimulates fat cells, particularly those known as brown fat cells, to convert calories to heat rather than to stored fat. The final outcome is a mouse with high body temperature and metabolic rate, a low percentage of body fat, and resistance to diet-induced obesity.

Other than their enviable leanness, the mice lacking RII-beta seem ordinary. Though their body weight is about 10 percent less than normal, the loss seems due to smaller fat cells rather than a decrease in muscle mass. Furthermore, the mice eat only slightly more than normal mice, remain fertile, and betray no obvious behavioral problems, McKnight's group reports in the Aug. 15 NATURE.

Do these trim mice offer any clues about people, either thin or obese? "There could be humans out there with abnormalities in PKA," says Bradford B. Lowell of Beth Israel Hospital in Boston. The genetically engineered mice also provide encouragement to Lowell and other researchers who have just begun examining whether compounds that stimulate PKA activity in mouse fat cells can become effective antiobesity drugs.