cities," so the agency is "the logical home" for the NCI, says another NCI official.

In the meantime, Europeans are forging ahead with their own plans for an initiative that would plow $10 millon or more a year into the cities. At a meeting later this month in The Hague, Netherlands, they will join with U.S. and Japanese representatives to identify potential projects. "It will be an attempt to prioritize unmet problems," says Segal, and "to try to get countries to commit money." Leading the charge for a European NCI, announced last week at a RANSAC-sponsored meeting in Washington, D.C., is the Landau Network–Centro Volta in Como, Italy, a nongovernmental organization that supports scientific cooperation with the former Soviet Union. The Landau network will follow up discussions at The Hague with its own meeting with nuclear city officials next month in Rome.

If the European effort gets off the ground, it could play a vital role in supporting the nuclear cities until NCI recovers. "It's a daunting agenda," says Segal, "but one for which we'll never be forgiven if we fail."

–RICHARD STONE

GENETICS

Gene Skews Patterns Of Inheritance

In the eyes of Mendelian geneticists, all chromosomes are created equal. In anticipation of sexual reproduction, paired chromosomes split up so that each developing egg or sperm ends up with just one of the partners. In theory, both partners have the same chance of making it into the next generation. But in mice and some fruit flies, reality is not so egalitarian. Sometimes one chromosomal partner consistently wins out over the other, seemingly breaking one of the basic rules of genetics.

Last March, researchers at the University of Wisconsin, Madison, solved part of that mystery in fruit flies. They showed that early in sperm development, while partner chromosomes are still close together, a truncated protein encoded on one copy somehow prevents pre–sperm cells carrying the other from maturing (*Science*, 12 March, pp. 1651 and 1742). Now mouse geneticists have fingered the gene responsible for a similar phenomenon in mice.

Last week at the 13th International Mouse Genome Conference in Philadelphia, Bernhard Herrmann, a geneticist at the Max Planck Institute for Immunology in Freiberg, Germany, described a mouse gene, located on chromosome 17, that can also skew chromosomal inheritance patterns. This gene, which codes for a protein kinase enzyme, apparently works by altering the ability of mature sperm to swim to their target, the egg. (The results also appear in the 11 November issue of *Nature*.)

The finding solves "one of the oldest riddles in mouse genetics," comments John Schimenti, a molecular geneticist at The Jackson Laboratory in Bar Harbor, Maine. It might even have practical uses. Putting the gene on an animal's sex-determining chromosome can alter the sex ratio of its offspring, allowing farmers breeding dairy cows, for example, to produce almost all female calves. "You could save a lot of animals and at the same time enormously increase the productivity," Herrmann says.

Geneticists first noticed the unequal transmission of a then-unidentified chromosome in the 1930s while studying a mutation that produces tailless mice. Mendelian genetics predicted that when the males breed with normal females, 50% of the progeny should have short tails, as a result of inheriting one mutant and one normal copy of the gene. But the crosses produced far fewer short-tailed animals. "This was the first time people saw a distortion in the Mendelian ratio in mammals," Herrmann notes.

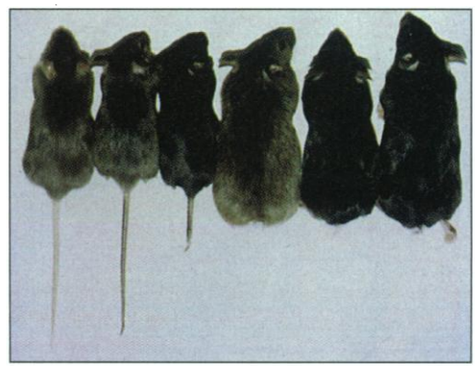
In 1984, Mary Lyon, a mouse geneticist at the Medical Research Council Laboratory of Mammalian Genetics in Harwell, England, took a stab at explaining this distortion after observing strange inheritance patterns of tail lengths in her breeding studies. She suggested that up to four genes had to be involved: one called the responder and as many as three others that she called distorters. She figured out that the responder reduced sperm fitness when not accompanied by distorter genes. As a result, both responder and the presumably closely linked tailless gene would be passed on less than 50% of the time. But when distorter proteins were present, Lyon predicted, the responder could counter their detrimental effects, skewing inheritance in favor of any chromosome carrying the responder. "It turns out that her model is correct," says Lee Silver, a geneticist at Princeton University.

Silver himself had gone looking for the responder gene, working with Schimenti. But although both they and Herrmann came up with candidates for the responder gene, neither panned out. Herrmann's gene, called *rsk3*, provided a lead to the right one, however.

The stretch of chromosome 17 where the *rsk3* gene is located has undergone several duplications and rearrangements. On a hunch, Herrmann decided to find out whether one of those rearrangements might have linked all or part of *rsk3* to the true responder gene. The hunch paid off.

The responder gene Herrmann found, called *Tcr*, consists of the partial *rsk3* gene fused to another gene that resembles genes for sperm-motility kinases, or Smoks. To make sure the new gene was the right one, the Max Planck group inserted it into various mouse chromosomes and showed that it does skew inheritance patterns. When present on the Y chromosome, for instance, the mice fathered far more than the normal 50% male progeny.

Herrmann thinks the responder gene handicaps sperm that carry it by causing their flagella to beat too slowly, while the distorter genes—whose identities are still unknown but which seem to be on the same chromosome—cause them to beat too fast. As a result, only the sperm lucky enough to get both



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Telltale tails. Less than the expected 50% short-tailed mice foretold the unusual inheritance pattern of mouse chromosome 17.

distorter and responder proteins move optimally and are able to beat out the sperm that lack either a responder or distorter.

The discovery in flies and mice of two very different genes that promote their own inheritance—and incidentally that of the chromosome they ride on—suggests that the evolutionary pressure to become such a self-promoter must be quite strong, says Silver. Successful transmission should lead to ever greater representation of that version of a chromosome in a population. Consequently, he adds, "there's probably thousands of examples out there that we can't see," perhaps even in people.

–ELIZABETH PENNISI

ANIMAL REGULATIONS

FDA Report Scores Chimp Research Lab

A federal investigation has found that the country's largest chimpanzee facility has violated dozens of regulations relating to good laboratory practices. The violations, described in a preliminary report detailing the results of an August inspection by the U.S. Food and Drug Administration (FDA), mostly involve inadequate record keeping, but they also include unapproved changes in experimental protocols. Animal activists who obtained the report claim that the irregularities raise questions about the integrity of trials involving potential new drugs and medical devices.

The Coulston Foundation, a private breed-