

Pandas mate at last but zoo aids nature

Champagne flowed this week at the National Zoo in Washington as the keepers celebrated the first successful mating of the only pair of giant pandas in the United States. The pandas, Ling-Ling and Hsing-Hsing, have been sexually mature for eight years; with legendary ineptitude they have frustrated all previous attempts at natural breeding. But this year, after Ling-Ling went into heat with much bleating and chirping, a mating was finally achieved.

The zoo reports that on Friday, March 18, at 11:45 a.m. the pandas copulated for about two minutes, then separated and slept until 2 p.m. In the afternoon Hsing-Hsing became uncharacteristically aggressive. The next day he attempted to mount again, behavior that led only to further unfriendly interactions.

To maximize the chance of a pregnancy, later Saturday zoo veterinarians artificially inseminated Ling-Ling with fresh sperm brought from Chia-Chia, the male panda at the London Zoo. They repeated the artificial insemination on Sunday. (In 1981 Chia-Chia himself was shipped to Washington, but failed to mate with Ling-Ling.)

Devra Kleiman, the zoo's reproductive zoologist, says that the artificial insemination should not jeopardize any natural



Jessie Cohen, National Zoo

To increase the odds of a panda pregnancy, Ling-Ling, anesthetized, is prepared for the first of two artificial inseminations.

conception. "The Chinese have done the same thing successfully," she says. Although the zoo officials "don't really care" which insemination succeeds, if a cub results, it may be possible to identify the father by genetic analysis, a procedure not previously performed on pandas, Kleiman says. Last year Ling-Ling raised hopes when, after artificial insemination, she showed some gestational behavior, but it was later thought to be due to a false pregnancy. For this year, signs of pregnancy are not expected until later summer, shortly before the cub would be born.

—J.A. Miller

Is DOE ignoring the fusion energy act?

Ambitious. That's how analysts characterized the Magnetic-Fusion Energy Engineering Act upon its passage in 1980. However, notes the General Accounting Office in the draft of a forthcoming report, "Citing budgetary constraints, the [Department of Energy] no longer plans to follow through the Act's intended development strategy." In fact, charges Rep. Fortney H. (Pete) Stark Jr. (D-Calif.), all of the [magnetic-confinement] fusion programs are suffering" as a result of major program cutbacks, project delays and funding well below levels envisioned under the act.

Magnetic-confinement fusion derives its name from the concept of "bottling" fusion plasmas that have been heated to millions of degrees within the melt-proof walls of intense magnetic fields. Under the fusion-engineering act, federal research, development and demonstration of magnetic-fusion energy would be accelerated. The act recommended a timetable for increasing federal spending over a seven-year period, in the hope of making possible a scientific and engineering assessment of fusion's commercial prospects by the early 1990s.

"This is the third year that the administration has submitted budgets in magnetic fusion well below [up to 27 percent below] those envisioned by the ... act," Stark

noted in congressional testimony March 17 before the House subcommittee on energy research and production. The act also requires that DOE make special reports annually on the status of its activities under the act. Yet, the GAO says, "DOE has yet to comply with this requirement." As a result, Congress is largely unaware of the extent to which DOE is altering fusion development policy and possibly subverting the intent of Congress, Stark contends.

Alvin Trivelpiece, DOE's director of energy research, says his agency intends "to maintain the U.S. leadership" in magnetic-fusion through its "balanced scientific program." In fact, scientific programs are "fairly healthy," with the exception of the Elmo Bumpy Torus-P project (which DOE proposes canceling), notes Stephen Dean, president of Fusion Power Associates (a group representing private-sector interest in fusion power). Dean says DOE's engineering programs, however, are another matter; "they are suffering." Though DOE may want to transfer fusion development to the private sector, Dean says, DOE hasn't committed itself to building the full line of research devices needed to provide sufficient engineering data to fuel industry interest in "putting private money into fusion."

—J. Raloff

Chemical key to emphysema found

The chemical roles cigarette smoke or air pollutants play in causing or accelerating the lung damage associated with pulmonary emphysema have been pinpointed by Ines Mandl and colleagues of Columbia University College of Physicians and Surgeons in New York City. Mandl's findings, which were presented this week at the American Chemical Society meeting in Seattle, have long-range implications for research on possible treatments for the disease.

Emphysema, a deterioration of lung function characterized by damaged alveoli (air cells), now afflicts about 11 million people in the United States; the disease kills about 56,000 persons each year in this country. During the past decade, scientists have learned that the first step in emphysematous deterioration is breakdown of lung elastin — an elastic connective tissue protein. Scientists also know that individuals with a genetically determined deficiency of alpha₁(α_1)-antitrypsin — a chemical that can inhibit the action of elastase, an enzyme that degrades elastin — are predisposed to the disease.

To shed more light on the disease process, Mandl induced emphysema in about 100 hamsters and then exposed some of those animals to cigarette smoke. First, she found, the cigarette smoke can oxidize (take electrons from) and thus inactivate the "elastin protector" α_1 -antitrypsin. Other oxidizers such as air pollutants are expected to act similarly, Mandl reported. This supports the contention that such irritants can cause disease in persons who are not genetically predisposed to it or aggravate an already high-risk situation.

Mandl also found that an increased amount of elastase was released by white blood cells into the lungs of the smoke-exposed animals. Finally, she discovered, smoke-exposed animals showed no expected increase in lysyl oxidase — an enzyme essential for the crosslink formation of new elastin and whose concentration normally rises in response to lung injury.

Recognizing the mechanisms by which agents cause or contribute to destruction of lung elastin is opening the door to a "rational approach" to possible treatments, Mandl reported. For example, Ronald G. Crystal and colleagues of the National Institutes of Health now are testing whether elastase attacks on lung elastin can be inhibited when α_1 -antitrypsin (or a chemical that leads to increased levels of this elastin-protector in the body) is administered to individuals deficient in the antitrypsin chemical.

Says Mandl, "The hope is ... that we will someday be able to reverse or at least arrest the process [of emphysema]."

—L. Garmon