

Social ties boost immune function

Monkeys whose social lives unfold in groups that continually lose established members and replace them with new recruits experience marked stress that apparently weakens the ability of their immune systems to fight disease, according to psychologist Sheldon Cohen of Carnegie Mellon University in Pittsburgh and his colleagues. However, monkeys who cultivate social bonds with their peers under these difficult circumstances inoculate themselves against immune decay, the researchers report in the September *PSYCHOLOGICAL SCIENCE*.

This project represents the first experimental study of any animal's immune response to long-term social upheaval.

Cohen's team studied 43 healthy, adult male macaques housed for 14 months in unchanging groups of four or five. The monkeys were then assigned at random to one of two conditions for the next 26 months: 21 lived in "unstable" groups, in which three or four monkeys departed each month to reside with another group, and 22 remained in their previous group.

Weekly observations indicated that "affiliative" behavior, such as friendly touching or grooming of another animal, occurred more often in unstable groups and may represent an attempt to dampen social stress, Cohen's team contends. High levels of affiliative behavior appeared in 14 animals living in unstable groups, compared with eight in stable groups.

In the three weeks that followed the study, monkeys in unstable groups also displayed, on average, a weaker proliferation of white blood cells in response to a substance that induces such cells to divide. Sluggish white blood cell responses appeared most pronounced among residents of unstable groups who had engaged in few affiliative gestures.

These findings may not apply to humans, the scientists note. But they suspect that macaques will provide a good animal model for understanding how the human immune system responds to psychological and social stress.

Happy birthday, and so long

Birthdays, beloved by children as an annual bonanza of gifts and sweets, may assume even more powerful meanings for adults. Here's a case in point: The approach of a birthday appears to prolong life for a short while among women and hasten death among men, assert sociologist David P. Phillips of the University of California, San Diego, and his co-workers.

Their analysis of 2,745,149 deaths from natural causes recorded in California from 1969 to 1990 found 3 percent more deaths than expected among women in the week following their birthdays, the highest female death rate for any week of the year. In contrast, male deaths reached a similar peak in the week before birthdays and then immediately leveled off, the team reports in the September-October *PSYCHOSOMATIC MEDICINE*.

The findings suggest that birthdays serve as a "lifeline" for some women and a "deadline" for some men, Phillips argues. U.S. women may cherish the increased attention from family and friends evoked by birthdays and therefore may somehow postpone death until just after the event. In a prior study, Phillips charted even larger "lifeline" effects, with death rates falling just before and rising just after Passover among religious Jews, as well as just before and after the Harvest Moon Festival among Chinese-Americans.

Men may use birthdays to take stock of the disparity between career and economic goals and achievements, thus creating a sense of dread as the annual event looms, Phillips contends. Any annual event that rubs in perceived failures may accelerate death among those with a serious preexisting medical condition, he theorizes.

However, the biological mechanisms by which birthdays or other symbolic anniversaries prolong or shorten life remain unknown, Phillips adds.

A method for earlier genetic testing

After plucking a single cell from an eight-celled embryo conceived through *in vitro* fertilization (IVF), geneticists have successfully determined whether the embryo carries the genetic mutation that causes cystic fibrosis.

The advance offers a means of detecting some genetic diseases much earlier than is possible using amniocentesis or chorionic villus sampling, two widely used techniques for diagnosing common genetic abnormalities. Unlike these tests, the new technique — called preimplantation diagnosis — is performed before a pregnancy begins.

A team led by Alan H. Handyside of Hammersmith Hospital in London, England, removed a cell from each of five embryos that had been conceived in a laboratory dish three days earlier. Both parents were carriers of the mutation responsible for cystic fibrosis and therefore had a one-in-four chance of having a child affected by the disorder, which clogs vital organs such as the lungs with infection-prone mucus.

Handyside collaborated with a group led by Mark R. Hughes of the Baylor College of Medicine in Houston to examine the five isolated cells for mutations. They found that two contained two copies of the mutant gene responsible for cystic fibrosis, one was a carrier of the disorder, and two others were unaffected.

Handyside's group implanted a carrier and an unaffected embryo into the womb of the mother. One developed, and the woman gave birth to a healthy baby, both teams of researchers report in the Sept. 24 *NEW ENGLAND JOURNAL OF MEDICINE*.

Preimplantation diagnosis "represents substantive progress" in genetic testing, Joe Leigh Simpson and Sandra Ann Carson of the University of Tennessee in Memphis comment in an editorial accompanying the report. Because the technique involves embryos that have not yet been implanted in the mother's womb, they write, couples may avoid the agonizing decision of whether to abort an affected fetus.

Simpson and Carson caution, however, that preimplantation diagnosis is technically difficult. They also question whether the technique's \$2,000 price tag makes it a practical way to conceive a baby — especially since it can only be administered to embryos conceived through the already costly IVF process.

Alzheimer's protein in healthy brains

Two teams of scientists, working independently, have found that normal brain cells can make beta amyloid, a primary constituent of the plaques that pock the brains of Alzheimer's patients.

The findings support the theory that Alzheimer's disease arises as a result of an imbalance between two naturally occurring processes for breaking down amyloid precursor protein, the forerunner of beta amyloid (SN: 3/7/92, p.152). Many scientists believe this imbalance may sometimes result from a genetic mutation that affects one of the processes.

The two research teams — led by Dennis J. Selkoe of Brigham and Women's Hospital in Boston and Steven G. Younkin of Case Western Reserve University in Cleveland — report their results in the Sept. 24 *NATURE* and the Oct. 2 *SCIENCE*, respectively. Both groups found beta amyloid secreted from healthy cells grown in laboratory culture. Selkoe's team also found beta amyloid in cerebrospinal fluid taken from both healthy individuals and Alzheimer's patients.

Selkoe's group suggests their finding may lead to a test for detecting elevated beta amyloid levels. However, in an editorial in the Sept. 24 *NATURE*, John Hardy and Mike Mullan of the University of South Florida in Tampa caution that "much more work is required before the relationships between [causative] factors for [Alzheimer's] disease and biochemical markers . . . are elucidated."