BEHAVIOR

FAS tied to occasional binge drinking

Five or six strong cocktails consumed in a single session early in pregnancy might be enough to instigate the mental retardation and many of the facial defects associated with fetal alcohol syndrome, suggest scientists from the University of North Carolina at Chapel Hill. Most previous research has indicated that the syndrome results from a woman's chronic abusive drinking throughout pregnancy (SN: 7/25/81, p. 53). But the study of pregnant mice by Kathleen K. Sulik, Malcolm C. Johnston and Mary A. Webb in the Nov. 20 SCIENCE suggests that there may be a two- or three-day "critical period" as early as a few weeks after conception when the developing human fetus is especially vulnerable to alcohol effects.

"Many women are not aware of their pregnancy at this stage," the scientists report. "Those who are aware may not realize that social or binge drinking so early in pregnancy may be as deleterious to the embryo as constant heavy drinking."

The researchers injected two fairly large doses of 25 percent ethanol four hours apart in mice that were seven days pregnant. A mouse's metabolism and rate of embryonic development is much faster than a human's, Johnston told SCIENCE NEWS. Thus the double dose of .015 milliliters of alcohol per gram of body weight on the seventh day roughly corresponds to two consecutive days of binge drinking for a woman three weeks pregnant. Alcohol levels in the blood of female mice rose to 215 mg per 100 ml of blood, which in humans is twice the maximum that most states permit as safe driving levels.

While examining the fetuses a week later, the researchers found facial defects, eye malformations and abnormally small brains in nearly 50 percent of the mice. Doses given produced a "staggering drunk," Johnston says, and were not meant to reflect the intake of occasional social drinkers.

Teaching an old chimp new math

Send a chimpanzee to school and she probably won't come home fluent in a human language. But she may pick up a few analytical skills that could help scientists understand the way children learn abstract reasoning.

"Although language training does not instill human language [in chimps], it does appear to make some overall cognitive changes," University of Pennsylvania researcher David Premack told SCIENCE NEWS, "particularly in the area of analytical reasoning." Turning aside from the fray over an ape's ability to learn and use language (SN: 5/10/80, p. 298), Premack and colleague Guy Woodruff tested the mathematical abilities of five chimps. Four of the apes, accustomed to learning simple tasks but unschooled in language skills, flunked the researchers' tests. But "Sarah," a 16-year-old graduate of 18 months of language training, demonstrated a good grasp of small numbers and simple fractions in the study reported in NATURE (Vol. 293, No. 5833).

In a typical test of the apes' understanding of numbers, the researchers placed two dishes in front of each subject — one containing three wooden disks, the other containing four disks. At the same time, the chimps were shown three cups of tinted liquid. Pushing the dish with three disks toward the tray with three cups constituted a correct response. The researchers admit that rather than counting, Sarah, who matched the numbers correctly in 91 percent of 120 trials, may have "subitized" — perceived at a glance the similarity between small numbers of items. (Human children are thought to attain such skills in infancy.) But the chimp seemed to demonstrate more complex reasoning in a second round of tasks, the researchers say, when she correctly matched wooden disk fractions (one-fourth, one-half, three-fourths and one) with partially filled jars of liquid in 88 percent of the trials.

BIOMEDICINE

Fetal surgery out of the womb

This year some spectacular advances have been made in fetal medicine by a handful of California scientists (SN: 5/23/81, p. 326; 8/1/81, p. 70; 8/22/81, p. 118). Now three of those scientists — Mitchell Golbus, Michael Harrison and Roy Filly of the University of California at San Francisco — report another first: treating a human fetus out of the womb.

The fetus in question was discovered by ultrasound, during the 21st week of pregnancy, to have a blocked urinary tract that could lead to a potentially fatal backup of urine into the bladder and kidneys. The condition was too advanced to be corrected by inserting a catheter through the mother's abdomen to drain the fetus's bladder. So with the consent of the mother, the researchers cut into her womb, withdrew the lower half of the fetus and extended the ureters (the tubes that carry urine from the kidneys to the bladder) outside the fetus's body to bypass the obstruction and allow drainage. The fetus was then returned to the womb and carried to term.

The operation was not successful, however, in that it was performed after the blocked urinary tract had irreversibly damaged the unborn child's kidneys, and the child died soon after birth. Still, the researchers are optimistic that they can perform the same operation on other cases in time to save lives.

Environment key to central nerve repair

Damaged nerves in the central nervous system don't repair themselves, unlike those in the peripheral nervous system. One reason is that the axons of damaged central nerves grow only short distances while the axons of damaged peripheral nerves grow long ones. Growing evidence from Albert J. Aguayo of McGill University in Montreal and his colleagues suggests that this is due to the environment in which the axons are located, not to intrinsic properties of the axons themselves.

In 1979 Aguayo and co-workers reported that injured peripheral axons with a known capacity to grow didn't do so when placed in an environment of glial cells from the central nervous system. (Glial cells are nonneuronal, supportive cells found in both the central and peripheral nervous systems.) Last year Aguayo and colleagues reported that if peripheral nervous system material was put in the central nervous system, damaged central axons would grow into the material. And now Aguayo and colleague Samuel David report in the Nov. 20 SCIENCE that when the central nervous system glial environment was replaced by a peripheral nervous system one, damaged central axons grew unprecedentedly and remarkably long distances.

A missing growth hormone gene

For the first time, the total absence of a gene has been found to cause a human disease other than some blood disorders. The disease is growth hormone deficiency.

Although the causes of most human growth hormone deficiencies are unknown, some have been identified through family pedigree studies as being genetic. John A. Phillips III of Johns Hopkins University School of Medicine in Baltimore and colleagues took DNA from four patients whose growth hormone deficiency was known to be due to inheritance, separated it into various fragments and attempted to match the fragments to a strip of DNA known to contain the normal human growth hormone gene as well as some other genetic material. As the scientists report in the October PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES, the patients' DNA fragments matched areas of the control strip of DNA but not the areas known to represent the normal human growth hormone gene, showing that a lack of this gene caused the patients' deficiencies.