Behavior

Hyperactivity: No go for amino acid

The theory makes sense: Hyperactive children given strong doses of phenylalanine, an amino acid found in some foods, may show behavior improvements, since this dietary chemical is eventually converted into two important chemical messengers in the brain, dopamine and norepinephrine. Deficiencies in these neurotransmitters have been implicated in hyperactivity, and a recent study suggested that hyperactive children may excrete less phenylethylamine, a metabolic product of phenylalanine.

In practice, however, loading up on phenylalanine appears to have no effect on hyperactivity, report psychiatrist Alan J. Zametkin of the National Institute of Mental Health and his colleagues in the June AMERICAN JOURNAL OF PSYCHIATRY. Eleven hyperactive boys between the ages of 6 and 12 were treated for two weeks with capsules containing a phenylalanine compound and two weeks with placebo capsules. No significant behavior changes, for better or worse, were noted on parent, teacher and experimenter ratings. Scores on tests of attention and memory also did not change with treatment. Although active treatment increased phenylalanine levels in the blood, there was no change in the amount of phenylethylamine excreted in urine.

Despite the amino acid's inability to quell hyperactive behavior, there is an encouraging aspect to the data, say the researchers. The artificial sweetener aspartame contains phenylalanine and is consumed in great quantities by some children, they observe, but it appears that large daily doses have no adverse effects on behavior. Blood levels of phenylalanine among boys in the study were comparable to levels reported for adults considered to be heavy aspartame users. There may, add the investigators, be long-term effects of increased phenylalanine levels that have not yet been examined.

X marks the spot

Evidence is mounting that, in some cases of manic depression, there is a gene near one tip of the X chromosome that predisposes its bearers to the disorder. Scientists who recently studied five families in Jerusalem used DNA-cutting enzymes to locate two genetic markers — one for color blindness, the other for a chemical deficiency that causes anemia — at the end of the long arm of the X chromosome. The markers occurred overwhelmingly among subjects with manic depression or related mood disorders (SN: 3/28/87, p.199).

Julien Mendlewicz of the Free University of Brussels, Belgium, and his colleagues now report that there is another manic depression marker in the same area of the X chromosome. DNA was isolated from 89 individuals, 41 of whom had manic depression or severe depression, in 10 families. A genetic marker for a blood coagulation factor located near the color blindness and anemia markers occurred mainly among family members with the psychiatric diagnoses.

The genetic link was emphasized by the fact that no fathers and sons shared mood disorders, say the researchers. The 23rd pair of human chromosomes consists of two X chromosomes for females and one X and one Y chromosome for males. The Y chromosome is inherited from the father.

There is probably more than one gene involved in predisposing people in different populations to manic depression, note the scientists in the May 30 LANCET. For instance, there is a genetic marker on chromosome 11 linked to manic depression among the Amish (SN: 2/28/87, p.132). But the investigators suggest that the long arm of the X chromosome may hold special promise for tracking down a predisposing gene.

"Banking of DNA samples from high-risk persons may lead to the isolation and sequencing of the [X-chromosome] gene responsible for manic-depressive illness," they say.

Biomedicine

Diane Edwards reports from Washington, D.C., at the Third International Conference on AIDS

Peptide T: Future AIDS treatment?

Scientists in Sweden and the United States are in various stages of preliminary human trials to test whether a small protein that stops the AIDS virus from entering cells will be helpful in treating patients with the disease. The protein, called peptide T, is a short segment found in the envelope of the AIDS-causing virus (SN: 12/20&27/86, p.388).

In Sweden, four patients with advanced AIDS were first injected with the protein last fall. One particularly ill patient, who requested withdrawal from the program, died, but the remaining patients are doing well, says Lennart Wetterberg of the Karolinska Institute in Stockholm. He reported that no toxic side effects have been observed, and weekly treatments are sufficient to maintain improved health. This contrasts with the drug azidothymidine (AZT) recently approved for AIDS patients in the United States, which requires daily doses and produces serious side effects like anemia in some users. Wetterberg says he does not know why the protein causes such dramatic improvement, causing pneumonia and skin lesions in the three AIDS patients to regress. Refusing to speculate on whether peptide T will prove to be a general treatment for AIDS, Wetterberg says case-control human trials must be completed before efficacy of the drug is known. The Swedish group has just started such a study in 36 patients: Half are being given peptide T, and the remainder, a placebo. Wetterberg predicts results will be available within a year.

Preliminary human trials of a synthetic peptide T made at the National Institute of Mental Health in Bethesda, Md., recently were approved by the Food and Drug Administration. Testing is expected to begin within several weeks, an NIMH official said last week.

Blood donation under the AIDS regime

Since mandatory screening of donated blood to detect antibodies against the AIDS virus began in March 1985, scientists have used the test results to assess the dangers of being infected by one of the nearly 15 million units of blood collected in the United States annually.

Between onset of mandatory testing and July 1986, the proportion of blood units positive for the AIDS antibody (seropositive) dropped significantly from .08 percent to .02 percent, according to data from more than 818,000 units collected in Los Angeles, Baltimore and Atlanta, says John W. Ward of the Centers for Disease Control (CDC) in Atlanta. Ward reported last week that a CDC-coordinated study of blood donor demographics shows that the majority of seropositive units in those urban areas came from black or Hispanic men who reportedly were bisexual. Another study, reported by Joel N. Kuritsky of the Food and Drug Administration, concluded that intravenous drug users in particular are a high-risk group that continues to donate blood. The CDC study found that the proportion of seropositive donors who had donated blood previously fell from 73 percent to 55 percent over the test period, indicating that some potential donors have voluntarily ceased donating blood.

Current blood screening tests are based on detecting the antibody, and not the virus, associated with AIDS. Because up to six months may pass between infection with the AIDS virus and the appearance of antibodies in the blood, the American Red Cross in Los Angeles studied the risk of being infected by blood that had tested negative for the antibody after it was collected, but still contained the virus and was "potentially infectious." Previous CDC studies had placed the average risk at 1 in 80,000 units of blood. By identifying seropositive blood donors who also had previously donated blood within the last six months, and the recipients of those earlier units, the Los Angeles group estimates the average risk as 1 in 48,000.