

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.

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.

Spot check on measles

In 1958, the United States was bespeckled with nearly 800,000 cases of measles. Although that number fell by half the following year, real gains in controlling the disease didn't come until after the licensing of a vaccine in 1963.

Although the cases reported in 1986 represent only a fraction of those prevalent in prevaccination days, the total more than doubled from the previous year. A total of 2,822 cases were reported in 1985, compared to more than 6,200 reported in 1986, with no deaths recorded, according to the May 29 MORBIDITY AND MORTALITY WEEKLY REPORT. This is the highest figure of any year since 1980, when 13,506 cases were reported.

New York City alone accounted for 945 reported cases, followed by New Jersey with 911 and Illinois with 710. More than half of all cases originated from 10 outbreaks of more than 100 cases each. The highest incidence was among children less than 5 years old, who accounted for 40 percent of all cases in 1986.

The Atlanta-based Centers for Disease Control (CDC), which published the report, cited two major reasons for the increase in cases: unvaccinated preschool-aged children and vaccine failures in school-aged children. If more preschoolers had been properly vaccinated and other preventive measures had been taken, 36 percent of last year's cases could have been averted, according to CDC. In addition, revaccination during selected outbreaks may be a strategy to combat vaccine failures.

Bridging the gap

In the last 10 years, scientists have shown that cells in the peripheral nervous system — those outside the spinal cord and brain — are capable of limited regeneration. But it has remained much more difficult to get damaged cells in the central nervous system to heal (SN: 3/29/86, p.204).

Now, scientists at the University of California at San Diego report that they have successfully induced nerve cell regeneration in the brains of rats — marking the first time such regeneration has been achieved with the use of human tissue as a nerve growth medium. The researchers implanted tiny "bridges" made of human placental tissue into surgically inflicted gaps in the animals' neuronal bundles. The research team, led by neuroscientist Fred H. Gage, used placental tissue because it is rich in "promoting factors," which normally stimulate the growth of fetal nerve fibers. The neurons grew across the placental bridge and reconnected with neurons on the other side of the surgical gap, raising hopes that some types of brain damage may someday be repairable. However, the team reports in the May 29 SCIENCE, further tests will be needed to show that the regenerated nerve cells function normally.

Targeting tumors

Scientists attending the annual meeting of the American Association for Cancer Research in Atlanta last month expressed optimism about a newly developed chemical that selectively kills tumor cells. J. Martin Brown, director of the radiation biology division at Stanford University School of Medicine, reported that he and his colleagues have designed a new compound that is naturally metabolized into a toxic form in the oxygen-depleted environment characteristic of many tumors. Solid tumors are often low in oxygen, or hypoxic, because they have outgrown their own blood supply.

Like other so-called bioreductive agents, the chemical, SR 4233, has been shown to release DNA-damaging free radicals at tumor cells in mice, especially when administered with other drugs that make tumors even more hypoxic than usual. Brown says the new chemical is "at least an order of magnitude more selective for hypoxic cells than previously used compounds," and so is less apt to harm healthy cells.

Energetic electrons: An ozone killer?

High-energy electrons riding the earth's magnetic field lines, 40,000 kilometers above the surface of the planet, have already been implicated in the malfunctions and failures of several satellites. Now, scientists from the Los Alamos (N.M.) National Laboratory propose that these electrons rain down from their high-flying orbits and possibly contribute to the loss of stratospheric ozone over Antarctica each year (SN: 5/23/87, p.326). Scientists are concerned with stratospheric ozone because it protects life on earth by absorbing harmful ultraviolet radiation from the sun.

Large populations of these electrons regularly appeared in the earth's magnetosphere every 27 days from late 1981 to 1984, a time that corresponds to a minimum in the 11-year cycle of sunspot activity. In recent years, as the solar cycle builds toward a maximum, the Los Alamos scientists have measured smaller, less periodic fluxes of these electrons, which travel at nearly the speed of light.

Most of the electrons remain trapped in the magnetosphere, but a small portion of them could precipitate out near the earth's poles and penetrate as far down into the atmosphere as 40 km above the surface, Los Alamos's Dan Baker reported at a recent meeting in Baltimore of the American Geophysical Union. These electrons would then ionize air molecules and in turn produce odd nitrogen compounds, which catalytically remove ozone from the stratosphere. In support of this theory, Baker points to a correlation between the years of greatest ozone loss and largest electron fluxes.

Researchers are unsure of the source of the accelerated electrons. In one proposed mechanism, solar winds energize electrons already present in the earth's magnetosphere. A rival theory relies on electrons that originate outside the magnetosphere and are accelerated by Jupiter's magnetic field.

Variations on a Pacific theme

Since the 1983 El Niño, when abnormal winds and temperatures wreaked havoc on the Pacific climate and on those whose lives are affected by it, many studies have focused on year-to-year variations in the climate of the tropical Pacific (SN: 1/24/87, p.55). Researchers have devoted much less energy to studying long-term variations in that climate. However, scientists are increasingly turning to records of winds and temperatures kept by merchant ships in order to decipher changes in the climate on the scale of decades.

By sifting through millions of wind observations from the years 1920 through 1983, a group of British researchers has discovered several decade-scale variations in the wind patterns of the tropical Pacific. In the May 21 NATURE, researchers from the Hooke Institute for Atmospheric Research in Oxford report that from 1940 to 1944 the easterly trade winds, which travel with an average speed of 5 meters per second, slowed to an average speed of 4 m/s. The researchers also report that the years 1950 to 1981 showed a trend toward increasing trade winds over the Pacific.

This later trend, say the researchers, could explain other long-term climate variations such as the dramatic decrease in rainfall in the Sahel region of Africa, which caused the disastrous African droughts of the last two decades.

Studies of decade-scale trends will help scientists identify subtle human effects on the climate by providing information on the climate's natural variability. However, many climatologists are wary of the merchant marine data, as they were compiled by sailors rather than scientists, and say that future studies must verify these observations by analyzing independent sets of data. Researchers have raised the possibility, for instance, that the trend in the 1940s could be an artifact of poor data coverage during the Second World War.