Behavior

Environmental roots to parkinsonism

New support for the theory that Parkinson's disease is caused by environmental factors has come from six Canadian families. Each family has several members who developed the disorder within less than five years of one another, despite large age differences, say Donald B. Calne of the University of British Columbia in Vancouver and his colleagues.

The appearance of the neurological disorder among only a portion of the parents and their children undermines a purely genetic explanation, say the researchers.

"Whatever the environmental factors were, they must have been transient," says Calne. "There may be a range of environmental toxins stimulating Parkinson's disease, including some in the diet, but at the moment this is very speculative." In one family, for instance, the only healthy member was also the only one who had abstained from a rigorous "health food" diet that included large quantities of sunflower seeds. Whether that diet contained toxic elements is unclear, says Calne.

Parkinson's disease usually occurs among people over 40 years of age and is marked by tremors, muscle rigidity and weakness and a shuffling gait.

In the Canadian families, an average of only 4.6 years separated the onset of symptoms in different generations, while the average age difference between children and parents who contracted the disorder was 25 years. Two of the families contained identical twins, and only one member of each pair developed Parkinson's disease, report Calne and his coworkers in the August CANADIAN JOURNAL OF NEUROLOGICAL SCIENCES.

The children, whose symptoms developed by age 40, had generally lived apart from their parents for two or more decades. Any common environmental causes must have occurred before they left home, conclude the researchers. This inference is in line with a previous hypothesis that environmental hazards first cause "clinically silent damage" to the brain early in life, followed by normal cell loss with aging that triggers Parkinson's disease (SN: 10/5/85, p.212).

Their data dovetail with a report that an amino acid found in the seeds of the false sago palm produces parkinsonism in monkeys and may have caused an increase in neurological diseases on two west Pacific islands (SN: 8/8/87, p.94).

Don't panic, you're pregnant

For 10 years, the 33-year-old woman had experienced recurrent panic attacks when in confined places or large crowds. Panic symptoms, including nausea, tremors and a sense of being out of control, caused her to avoid people and limit her driving. During three pregnancies, however, the attacks dramatically eased and she became less socially inhibited.

Her case, as well as those of two other women whose panic attacks showed marked improvement during pregnancy, raises the possibility that pregnancy has an antipanic effect, report David T. George and his colleagues of the National Institute on Alcohol Abuse and Alcoholism in Betheseda, Md. The improvement cannot be explained by medication, psychotherapy or stabilization of the womens' life situations, add the researchers in the August AMERICAN JOURNAL OF PSYCHIATRY. One possiblity, they suggest, is that pregnancy blunts activity of the sympathetic nervous system, which accelerates the heartbeat and secretes adrenaline during panic attacks. Or, hormonal changes in pregnancy may alter barbiturate receptors in the brain and create antianxiety chemicals. Finally, the sense of purpose and self-esteem fostered by pregnancy may help to quell panic attacks. If the finding holds for a larger number of panic patients, say the researchers, the relationship between pregnancy and panic attacks should be fully explored.

Biomedicine

Drug shows potential as MS treatment

A synthetic substance that mimics muscle protein significantly reduced the number and severity of disabling neurological attacks in selected multiple sclerosis patients during a two-year study, scientists reported last week. But the researchers emphasized that the study must be considered preliminary, and an official of a national multiple sclerosis organization called for extreme caution in interpreting results.

Researchers at the Albert Einstein College of Medicine in Bronx, N.Y., and the Weizmann Institute of Science in Rehovot, Israel, used a drug called Cop 1, similar in structure to the myelin basic protein found in the sheaths surrounding nerves. In multiple sclerosis, lesions on nerve sheaths usually lead to progressive weakness and tremors, which often follow a cyclic course of exacerbations and remissions. Patients with this so-called exacerbating-remitting form of multiple sclerosis took part in the recently reported study.

According to a paper in the Aug. 13 NEW ENGLAND JOURNAL OF MEDICINE, daily injections of Cop 1 stabilized or improved the assay scores used to measure a patient's neurological function. Nearly 85 percent of the 25 patients given the drug showed beneficial effects, compared with 30 percent of the 23 control subjects injected with saline as a placebo.

After taking into account initial scores and previous exacerbation rates, the authors conclude that a patient taking saline was four times more likely to show progressive symptoms than was a Cop 1 patient. The greatest improvement, however, occurred in patients who had less severe disease at the beginning of the study.

In the Aug. 20 NATURE, Byron H. Waksman of the New York-based National Multiple Sclerosis Society emphasizes that both the unpredictable course of the disease and questions about the study reported last week make expanded studies of Cop 1 mandatory. There have been, he says, about 150 "therapies" proposed for multiple sclerosis thus far.

Steroid helpful in muscular dystrophy

Using the steroid prednisone in patients with Duchenne muscular dystrophy (DMD) may delay by an average of two years their confinement to a wheelchair, say scientists at the Johns Hopkins University School of Medicine in Baltimore. Daniel B. Drachman and his coauthors report in the August ARCHIVES OF NEUROLOGY that the 16 prednisone-treated patients studied were able to walk and stand until an average age of 12, whereas the 38 untreated subjects lost their mobility by an average age of 10.

A genetic disease that affects young boys, DMD causes progressive weakening of the muscles, resulting in the need for a wheelchair by the age of 9 to 11 years. To assess the long-term effects of prednisone, the Baltimore study followed some patients as long as 14 years until they lost mobility. Among those patients who received prednisone for the longest period of time, loss of mobility was postponed more than three years compared to the control group. The treatment, however, does not stop the disease, and can produce side effects such as weight gain, hyperactivity and a rounded face.

Despite its limitations, researchers say that prednisone may prove itself as the first available treatment for the disease, which affects 1 in 3,000 live male births. In a six-month study also reported in ARCHIVES, 33 Duchenne patients treated with prednisone at four other medical centers showed definite improvement in muscle strength, although eventual immobility was not prevented. How prednisone acts to improve or slow the onset of symptoms is unclear. Authors of the reports suggest that prednisone's known anti-inflammatory capabilities may play a role, as may its ability to stabilize leaky muscle-cell membranes.