Evidence for a 'schizophrenia virus'

Classifying an emotional disorder as a medical "illness" or "disease" often carries with it the unspoken duty of researchers to find a cause of the disease — such as a blood abnormality, or perhaps even a virus. Psychiatrists for years have treated the symptoms of schizophrenia as they would those of many physical disorders: with drugs. But while schizophrenia has been associated with imbalances of certain brain chemicals — imbalances which may be genetically transmitted — the ultimate cause or causes of the disorder's thought, mood and behavior disturbances remain elusive.

The possibility of abnormal blood components or viruses has been suggested by a number of scientists, including Harvard University psychiatrist Seymour S. Kety (SN: 10/7/78, p. 244). But there has been little physical evidence to support the existence of any such component.

Now, however, researchers in England report they have found a "virus-like agent" (VLA) in the cerebrospinal fluid (CSF) of 18 of 47 patients diagnosed as schizophrenics. In addition, "VLA was also detected in the CSF of 8 of 11 patients with serious or chronic neurological disease (Huntington's chorea, multiple sclerosis and unexplained alterations of consciousness)," the scientists report in the April 21 issue of THE LANCET.

In a laboratory test, the spinal fluid of the 18 "positive" schizophrenics — and the eight with neurological disease — had a pathological, or disease-like, effect on human cell cultures. In contrast, VLA was detected in only one of 25 patients in a control group. Though the exact nature of the suspected viral agent is not known, the researchers suggest it may be some type of "slow virus," that takes months or years to trigger disease after infecting a person (SN: 10/7/78, p. 245).

Kety has suggested that a form of viral schizophrenia might account for cases that do not seem to be hereditary (about half the cases he has studied in Denmark). But the British scientists speculate that the VLA they have detected has a genetic component. "Our present hypothesis is that the VLA is widely distributed in the community and is pathogenic only in a genetically predisposed subpopulation," they say.

"On purely theoretical grounds," the possibility of a schizophrenia-linked virus — and a group of people genetically susceptible to it — "would be quite reasonable," says Soloman H. Snyder, distinguished service professor of psychiatry and pharmacology at Johns Hopkins University. "Of course, it would solve everything if a simple virus [were found], but I would be very cautious [about the British results]," Snyder says.

Chief among the dangers of such work, he says, is the possibility that the antipsychotic drugs used to treat schizophrenics might falsely color the results. "I would worry ... about whether maybe the drugs or something that the drug does could be responsible for the virus in the tissue culture," says Snyder. "There are many kinds of artifacts that plague this kind of research."

Nevertheless, Snyder says, "If we can demonstrate a viral component to schizophrenia, it would obviously be extremely important." The English researchers not only agree, but believe they may have found the first solid evidence of such a viral agent. "If VLA(s) are found to cause illness there will be a possibility of treatment or prevention by vaccination" or by giving antiviral compounds, they write in the article in THE LANCET. Such a virus, they add, probably is not contagious. "Epidemiological evidence argues against the ready transmissibility of schizophrenia or the other conditions from affected individuals," they say.

The reporting scientists, all from the Clinical Research Centre at Northwick Park Hospital in Harrow, Middlesex, England, are D. A. J. Tyrrell, T. J. Crow, R. P. Parry, I. N. Ferrier, Eve Johnstone, D. G. C. Owens and J. F. MacMillan. □

Putting a check on ozone predictions

Estimates of ozone depletion due to — take your pick — supersonic transport, chlorofluoromethanes or volcanic eruptions seem to fluctuate more than the stock market. But it's not because atmospheric scientists are as fickle as the economic indicators; it's just that atmospheric chemistry and the computer models that predict ozone depletion have undergone a revolution since 1973. And if atmospheric chemistry is evolving so rapidly, how can model-makers be sure that their creations are not spitting out predictions that, in hindsight, will appear outlandish?

What atmospheric scientists need is some way to check their models. And Julius B. Chang, William H. Duewer and Donald J. Wuebbles of the Lawrence Livermore Laboratory think they've found one possible check — the atmospheric nuclear tests of the 1950s and 1960s.

The nitrogen oxides (NO_x) released by those nuclear tests were expected to cause a decrease in stratospheric ozone, which would reach a maximum in 1963. The ozone record for those years, however, shows no change in the ozone layer larger than the accepted 4 percent annual variability. (Total ozone has been monitored from several different stations around the world since 1957.) Any model that produces a 1963 response to the test series that is larger than 4 percent, therefore, "may be in error in a way that seriously affects its reliability in other prognostic applications," the researchers say in the April 20 JOURNAL OF GEOPHYSICAL RESEARCH. In this way, the nuclear test series appears to provide a "negative test" for such models.

The ability of the models to reproduce the actual effects of the nuclear tests has increased, Chang and co-workers show, as knowledge of the chemical reactions that are fed into the computer model has improved. A model that was used in 1973 — the same one that forecast an ozone decrease due to the SST — predicted as much as a 10 percent ozone reduction from the test series. Between 1973 and 1974, several reactions involving HO_x were found to occur and were added to the models. By 1976, reactions involving stratospheric chlorine were added (which produced the first estimates of the effects of fluorocarbons) and the rate of the reaction between OH and HO_2 was revised. As a result of these changes, the predicted effects on 1963 ozone levels dropped, but still remained larger than 5 percent. The most drastic change was due to a 1977 revised estimate of the rate of the reaction involving NO and HO_2, which was increased by about a factor of 40. This addition had direct bearing on the problems of ozone depletion by nuclear testing and the SST, both of which produce NO. Because of this revision, the estimated decrease for 1963 dropped to about 2 percent, an amount that would be lost in the natural variability. (The effects of the SST have also been discounted by these improvements. In fact, Duewer told SCIENCE NEWS, for the last year scientists have suspected that the SST may cause a slight increase in ozone.) More recent, preliminary estimates, that consider rate constants calculated in 1978, indicate the possibility of a slight increase in 1963 ozone levels due to the testing, he said.

Simply because they pass the "nuclear test," the success of current models doesn't exclude room for improvement, the researchers note. "It's my opinion," says Duewer, "that there is latitude for future changes of comparable magnitude to the ones in the past. But the models have gotten substantially better. The expectation of seeing a change is not as great." □

Coming — another DOE?

Bills to create a new Cabinet-level Department of Eduation have passed both of the critical House and Senate committees. The new DOE, staffed with about 22,000 people, would have a budget near $14 billion. Its science component, transferred from the National Science Foundation, totals only $27.8 million in the House, $24.4 million in the Senate version. The House bill focuses on undergraduate science and minorities, the Senate bill on precollege science and minorities. □