PSYCHOBIOLOGY

A Lead On Schizophrenia

Delicate chemical research indicates schizophrenic brains may produce antibodies against their own cells.

A major step toward understanding one of the most refractory of all mental illnesses—schizophrenia—has been taken by Tulane University researchers.

Their new evidence that schizophrenia—particularly acute schizophrenia—is an autoimmune disease typifies the advances made over the past decade in understanding the brain.

Only 10 years ago, schizophrenics languished in hospitals, immune to most of the psychotherapeutic measures taken against their disease, and filling a good percentage of the nation's hospital beds. Then medical scientists discovered the drug chlorpromazine with its startling ability to reduce schizophrenic confusion. Hospitals began releasing patients, some of whom had spent their entire lives on the wards. But chlorpromazine is, after all, only a medication, not a cure, and taken too long, can damage the eyes and discolor the skin.

Meanwhile, Dr. Robert G. Heath and his associates at the Tulane School of Medicine pursued increasingly refined chemical studies, believing schizophrenia to be a disease in which the body manufactures antibodies against its own brain cells. Their 10 years of research, published in the January Archives of General Psychiatry, indicates that these antibodies combine with cells in specific brain regions to impair their work.

Most likely, they propose, disruption occurs in the cell's capacity to pass on correct information, thus producing schizophrenic behavior.

Dr. Heath believes the guilty antibody to be a protein called taraxein, which has, in fact, been found in the blood of schizophrenics, first by Dr. Heath and then confirmed by others.

His next step, reports the Tulane scientist, is to search for an antidote to this protein and thus find a chemical cure for schizophrenia which afflicts some half a million Americans.

Though several U.S. scientists have been probing into the chemical subtleties of the brain for an answer to schizophrenia, Dr. Heath's group has been the only one to approach it as an immunologic disease.

Now, it seems likely others will take his lead. Should they be able to confirm his evidence, schizophrenia, or at least some forms of it, will no longer be the elusive, stubborn condition it has remained over the years, despite treatments ranging from psychotherapy to LSD to vitamin B3.

But two major questions remain unanswered, according to Dr. Hudson Hoagland, executive director of the Worchester Foundation for Experimental Biology in Massachusetts.

One concerns the taraxein substance. That it is a factor in the blood of schizophrenics and that it will disturb the behavior of animals which have been injected is well established. But whether the results are actually schizophrenic episodes needs to be more firmly established, says Dr. Hoagland. He says he did observe the effects of injecting taraxein into human volunteers and was impressed. The evidence is good, but it needs "to be tied up more tightly."

The other problem concerns schizophrenia itself. Dr. Hoagland, like most physiologists engaged in this research, does not believe the disorder is a single disease. While Dr. Heath maintains that taraxein is present only in schizophrenics, not in normal people, the evidence is not yet conclusive. Taraxein does appear in two-thirds of schizophrenics, leaving a questionable one-third and an unclear dividing line between normality and schizophrenia.

But the Heath work is a highly promising lead that will be followed intensively by others, says Dr. Hoagland. "I think he has something darned good."

In his latest experiment and the cap to his former work, Dr. Heath experimentally created an antibody with many of the characteristics of taraxein. When he injected it into well monkeys, it not only produced catatonic-like behavior, but left traces in precisely those brain areas—the septal-caudate—found to be abnormally changed in schizophrenics.

Earlier Dr. Heath had studied brain tissue from 14 schizophrenic patients within 10 hours after their deaths and had discovered an antibody attached to the nerve cells in 12 of them. But 19 normal people showed no such abnormal protein.

It is these septal-caudate cells, believes Dr. Heath, that are uniquely capable of producing the guilty antibody, taraxein. The antibody carried by blood then acts back upon the cells, not to destroy them but to combine with them, possibly disrupting the passage of information from one cell to another.