

some general way ought to look like what we see [in newer crust]. But our observations suggest that's not true."

Either the processes shaping the earth have changed since the planet's early history, or there are some as-yet-un-

discovered processes that act on the crust and the upper mantle over very long time periods, changing the crust from the kind of structure scientists see under the Cordillera to that of the craton just to the east of it, says Nelson.

One way to understand the processes that have formed the Moho is to look at many different areas and hope a pattern emerges, he says. "Right now we're just getting a hint of a pattern. But we don't know the reasons for it." — *S. Weisburd*

Alzheimer's disease: Scientists report research advances

Medical scientists cannot tell you the cause of nor administer a cure for Alzheimer's disease, which slowly erodes the minds of 5 to 10 percent of people over 65. But there was excitement and optimism at an Alzheimer's disease research forum held last week in Washington, D.C., at the 16th annual meeting of the Society for Neuroscience. In an unscheduled, last-minute presentation, Dmitry Goldgaber of the National Institute of Neurological and Communicative Disorders and Stroke (NINCDS) reported the isolation, localization and characterization of a protein and gene possibly associated with the illness. Also notable was the disclosure by two scientists from the Albert Einstein College of Medicine in the Bronx of their discovery of what may be the first accurate diagnostic indicator of Alzheimer's.

In time, these and other research findings reported at the forum could lead to an understanding of the causes of the disease, which affects an estimated 2.5 million elderly people in the United States at an inestimable cost in human suffering and which levies an economic burden soaring into the billions of dollars.

Goldgaber reported results of ongoing experiments that he is conducting in collaboration with Carleton D. Gajdusek, also of NINCDS, and Michael Lerman, Wesley McBride and Umberto Saffiotti, all of the National Cancer Institute. The scientists have identified and localized a gene on chromosome 21 as coding for part of a protein that may be a precursor of amyloid, a small protein that is unusually abundant in the brains of Alzheimer patients. While some scientists have long suspected that the disease is genetically based (SN: 7/2/83, p.5), Goldgaber's research marks the first time a gene has been experimentally associated with the disease.

The researchers have not yet published their results, nor could they have submitted them — the report did not yet exist — before the deadline for this year's Society for Neuroscience meeting. But the importance of their laboratory findings compelled them to circumvent the more orthodox and slower avenues. About a week before the meeting, Goldgaber called symposium chairman John H. Morrison of Scripps Clinic and Research Foundation in La Jolla, Calif., and informed him of his group's results. Morrison agreed that the presentation should be included at the meeting.

Goldgaber emphasized in the news

conference that neither amyloid's action in cells nor its abundance in Alzheimer brains is understood. But now that they know the cellular address of the gene coding for amyloid, scientists will be able to compare these genes from Alzheimer patients with those from healthy people, Morrison told SCIENCE NEWS. Any differences in the genes and the gene products would strongly suggest that the gene is at least partly responsible for the devastating disease. Goldgaber says it is too early to say that the gene definitely plays a role in Alzheimer's.

Other research reported in two presentations of the symposium could lead to early diagnosis of the illness, an ability that would be essential for any cure that may be developed in the future. Presently, the only way to make a positive diagnosis of Alzheimer's disease (except in rare cases when brain biopsies are done) is to examine brain cells obtained after the death of the patient. Pathologists look for specific cellular signs that Alzheimer's disease, and not some other disease, was the cause of the patient's symptoms. Such signs include tangles of tiny filaments inside neurons, chaotic assemblages of cellular components and indications that abnormal brain-cell death had occurred before the patients themselves had died.

Peter Davies and Benjamin Wolozin of the Albert Einstein College of Medicine reported their discovery of what might become the first unambiguous early indicator of the disease. Using an antibody probe, the two researchers observed high levels of a protein they call A68 in eight of nine brains from people who had succumbed to Alzheimer's disease. In addition, brain samples obtained from biopsies of two living patients suspected of having Alzheimer's disease had high levels of A68, indicating that the protein accumulates early in the course of the disease.

The scientists developed a technique that allows them to detect A68 in the cerebrospinal fluid of Alzheimer patients. Davies says he hopes the procedure can be used as a diagnostic test, which will greatly facilitate clinical research. In addition to the diagnostic possibilities, the Bronx scientists' work reveals more of the biochemical orchestration that underlies the disease. A68 has "protein-kinase" activity, which makes it like a middle-management protein that regulates the activity of other cellular proteins and enzymes. Also, electron microscope

studies show A68 to be associated with neurofilaments in Alzheimer brains.

This evidence, says Wolozin, suggests that A68 may play a role in the process that leads to the tangles and disorganized plaques observed in certain areas of Alzheimer brains. Scientists suspect that these are signs of neural disorganization that underlie the mental deficits characteristic of the disease.

In other research on diagnostic indicators for Alzheimer's, scientists from the National Institutes of Health and the National Institute on Aging found that Alzheimer patients are not good at discriminating smells. This olfactory impairment, they say, is detectable in "the very early stages of the disease" and therefore might be useful as a signal for the presence of the disease.

Ten other scientists at the forum reported a wide range of findings that describe how brains of Alzheimer victims differ from those of healthy people. A group of scientists from Scripps Clinic and Research Foundation and The University of Psychiatry in Geneva, Switzerland, found that the tangles and plaques seen in Alzheimer brains during autopsies are associated with those cells in the cortex that project long fibers to other cortical cells. Three scientists from the University of British Columbia in Vancouver reported low levels of hippocampal RNA in Alzheimer brains. Other groups from U.S. universities, hospitals and research institutes described possible immunological and viral involvement in the disease, the extent and range of disorganization in neural structures, animal models for studying the illness, and abnormalities in Alzheimer brains of the distribution of proteins, neurotransmitters, hormones and other biological molecules.

These presentations come on the heels of a report of a drug treatment that appears to help alleviate some symptoms of Alzheimer's disease (SN: 11/15/86, p.308).

Although no cure for Alzheimer's disease will precipitate directly from the findings disclosed last week, many medical researchers are optimistic that early diagnosis is in the offing. However, Morrison and other scientists emphasize that they are still very much in the phase of discovering and describing the details of the disease in order to determine exactly what it is they are contending with.

— *I. Amato*

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