Abrupt extinctions at end of Triassic

As the Triassic period closed, about 200 million years ago, large families of animals started dying off in record numbers, forever changing the character of life on earth and paving the way for the age of the dinosaurs. Scientists previously had thought that these extinctions occurred gradually over the course of 15 million to 20 million years. However, an analysis of fossil finds in Nova Scotia now suggests that they happened quite suddenly and may have resulted from the impact of a large meteor.

Paul E. Olsen of the Lamont-Doherty Geological Observatory in Palisades, N.Y., and his colleagues report in the Aug. 28 Science that "the disappearance of dominant Triassic forms was abrubt, occurring in less than 850,000 years." Indeed, according to coauthor Neil H. Shubin from the University of California at Berkeley, "That's an absolute max. Our gut feeling is [that it was] a lot less."

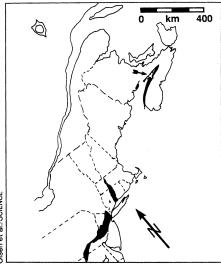
Olsen's group is basing its conclusions on four groups of fossils found two years ago in the McCoy Brook Formation along the Bay of Fundy (SN: 2/8/86, p.86). These fossil assemblages of tetrapods, or four-legged creatures, date to the earliest years after the end of the Triassic and the beginning of Jurassic period and have proved extremely valuable in measuring the duration of the extinctions, says Shubin.

The tetrapod remnants are unusually abundant and were discovered in a diverse range of environments including lake beds, lake shores and sand dunes. Olsen's group found that many Triassic animals were conspicuously absent from the fossil assemblages, which means that they had already disappeared by this early date in the Jurassic.

These conditions — rich groups of fossils found in different environments — help assure researchers that the absence of certain species actually represents an extinction rather than a fluke of fossilization. "It gives you a lot of confidence," says Shubin.

The lake environments will also help scientist refine the dates and duration of the extinctions. Because cycles of the earth's orbit affect the rates of sedimentation in large lakes, scientists can actually resolve the ages of sedimented layers down to 20,000 years. Using these lake cycles, Shubin hopes to demonstrate that the extinctions happened over a much shorter period than 850,000 years.

Berkeley geologist Mark H. Anders, another coauthor of the paper, is currently seeking to determine whether the sudden mass extinctions at the end of the Triassic are related to the roughly contemporaneous impact of a meteorite, which punched a 70-kilometer-wide hole



Arrow in Nova Scotia shows location of fossils, in part of a rock formation known as the Newark Supergroup (shaded areas). A nearby impact crater (upper left) may be related to Triassic extinctions.

in the ground of nearby Quebec. In the last decade, geologists and paleon-tologists have debated whether an extraterrestrial impact caused a later bout of mass extinctions at the end of the Cretaceous period, about 66 million years ago (SN: 5/16/87, p.309).

Olsen's group believes that the Triassic-Jurassic boundary provides an excellent opportunity to conduct a general test of the impact theory. They reason that the impact in Quebec must have left some marks in the well-preserved stratigraphic record in the McCoy Brook Formation and elsewhere. If the researchers find shocked quartz or other evidence of an impact directly prior to the disappearance of many species, this would provide those who are tracking the cause of the extinctions with a so-called smoking gun. However, a null result would not necessarily disprove the impact theory.

While many hail the Nova Scotia finds as significant contributions to the sketchy fossil record for this time, some paleontologists are reserving judgment on the duration of the Triassic mass extinctions. "It's quite possible," says Berkeley paleontologist Kevin Padian, "that the extinctions along the East Coast [of North America] occurred fairly quickly, but whether that has general applications around the world is still an open question."

East Coast extinctions might be unique because this area was experiencing intense geologic upheaval at the end of the Triassic, notes Padian. At this time, all the continents of the earth were part of one large supercontinent that was in the process of breaking apart. As North America began to tear away from North Africa, volcanic activity and extensive faulting ripped open a valley in between —a valley that later developed into North America's East Coast. — R. Monastersky

Bringing back fading memories

There are both joyous and sad memories. And there are those lost as a person ages — a loss that is exacerbated if the person has Alzheimer's disease. An inability to recall recent events can be disabling and frightening, and researchers are seeking ways to halt such age-and disease-related failures of short-term memory. Scientists in Sweden and the United States report this week that a substance called nerve growth factor may help improve impaired memory.

A protein produced by nerve cells, nerve growth factor was isolated in the early 1950s. Later, scientists observed that degenerative changes in the brains of Alzheimer patients occur in the same regions as those affected by the growth factor. On the basis this relationship, researchers at the University of Lund in Sweden and the University of California at San Diego recently tested the effects of infusing nerve growth factor into the aging brain, by studying changes in memory retention among aged rats.

As described in the Sept. 3 NATURE, the scientists repeatedly placed 2-year-old rats in a tank of water made opaque by a white powder, and observed the length of time it took for each rat to swim to a submerged platform on which it could stand. The "water maze task" is an established measure of how well rats retain prior knowledge of the platform's location, Anders Bjorklund of Lund told Sci-ENCE NEWS. He says that inability to learn the task is directly related to the degree of atrophy seen in the cholinergic system of a rat's brain. The network of cholinergic nerve cells - which release the chemical acetylcholine during message transmission between cells - is also affected in Alzheimer patients.

Two months before taking the water maze test, aged rats had been placed in the tank and categorized by whether or not they could memorize the platform's location. Those that could not were placed in the "impaired" group. About half the rats in this group were implanted with pumps containing nerve growth factor, while the remaining impaired rats served as controls, receiving a common blood protein as a placebo. After implantation, the rats were tested twice during a 28-day infusion period: about one week, and then three weeks, after nerve growth factor (or placebo) therapy began.

There was no change during the first week, but by the third week of therapy, performance of the rats given nerve growth factor was as good as that of the nonimpaired group, say the authors. They attribute this to "improved retention" of information learned during the first week's test. Along with the improved memory among the treated rats, the

SEPTEMBER 5, 1987 149

researchers found that cholinergic nerve cells on the side of the brain holding the implant increased in size.

Results from the short-term tests do not, however, show that nerve growth factor can improve the ability to learn new tasks or stop the gradual deterioration of overall memory, says Björklund.

This report follows an annnouncement last month that clinical trials are about to begin on another potential drug for Alzheimer's disease. Called tetra-hydroaminoacridine (THA), the drug was synthesized nearly 80 years ago for other

purposes. Last year, scientists reported that THA had improved memory function in a small group of patients (SN: 11/15/86, p.308). During the two-year clinical trials, about 300 Alzheimer patients will take the drug orally at U.S. research centers. THA is thought to block or slow the breakdown of the acetylcholine. Even if results from the clinical study confirm memory improvement due to the drug, experts have cautioned that THA probably will not stop the progressive destruction of brain cells that is characteristic of Alzheimer's.

— D.D. Edwards

Faces of emotion: Social or innate?

It is common finding that, in the United States, females are more facially expressive than males. This advantage now appears to be importantly influenced by cultural factors and social training, since women from Taiwan and mainland China do not show more facial expressiveness than their male counterparts, according to a study reported in New York City this week at the annual meeting of the American Psychological Association.

At the same time, report psychologists Ross Buck and Wan-Cheng Teng of the University of Connecticut in Storrs, there are substantial differences in facial expressiveness between subjects from Taiwan and mainland China. This suggests, in their view, that "political and social changes taking place within a few generations can profoundly affect the spontaneous emotional expressiveness of the population of a nation, with far-reaching personal and cultural consequences."

The researchers showed emotionally loaded color slides to 44 foreign students at the University of Connecticut, 24 from Taiwan and 20 from mainland China. In addition, 20 U.S. students were shown the slides. The slides included scenes of nude males and females, pleasant landscapes, severe burns and facial injuries, unusual photographic effects and people familiar to the students. Unknown to the subjects, their facial and gestural responses to each slide were recorded by a hidden videocamera. Later, after the presence of the camera was revealed and their permission to use the videotapes was granted, each subject attempted to match the appropriate slide to taped facial expressions obtained from the other students.

Among the results: Subjects from all three countries were equally likely to pick the correct slide for facial expressions of both male and female Chinese students; there was a marked advantage in choosing the correct slide for U.S. females' expressions compared with those of U.S. males; and students look-

ing at someone of either sex with pronounced facial expressiveness could identify the appropriate slide regardless of their own cultural background (this ability, however, was stronger for females from each nationality).

The results, although tentative, are consistent with the theory that the ability to interpret spontaneous nonverbal communication is innate and universal to the human species, say the investigators.

Yet the spontaneous facial expressions of Taiwanese students were significantly more indicative of what they were viewing than were the expressions of mainland Chinese students. The greater exposure to worldwide media and emphasis on individuality in Taiwan may promote this difference between people from the same ancient Chinese culture that has split along political and ideological lines, say the Connecticut researchers.

There may be other reasons for the greater expressiveness of the Taiwanese, they add. One of the experimenters was from Taiwan, and relations between Taiwan and the United States have always been cordial, which may have served to make Taiwanese students less defensive and inhibited in the laboratory.

It is also possible, say Buck and Teng, that female subjects in the study were more expressive than they appeared. For example, students from Taiwan and mainland China who rated the videotapes may have expected females to have negative reactions to sexual slides when, in fact, some females may have had positive, amused responses to those scenes. Thus, negative reactions by females may have been pegged inaccurately to viewing a sexual slide.

But for now, say the researchers, it appears that the ability to send spontaneous emotional messages through facial expressions is subject to social influence, while the ability to understand those messages lies outside the social realm.

— B. Bower

'Competition' cause of AIDS dementia?

Structural similarities between AIDS-causing viruses and a protein that stimulates nerve cells may contribute to the dementia common in AIDS patients, and thus may provide a way to reverse that dementia, scientists said last week. The researchers suggest that the similarities create a competition between virus and protein for binding sites on neurons — leading to inhibition of nerve function by the viruses, and in turn causing symptoms associated with dementia.

Since the AIDS-causing HIV-1 virus was first isolated from brain tissue in 1985. there have been multiple reports of the virus found in the nervous system of AIDS patients. Over the same period of time, clinicians began routinely observing AIDS patients for signs of dementia, including memory impairment, apathy and poor concentration. Now recognized in at least two-thirds of AIDS patients, signs of dementia recently were added to a revised definition of the deadly disease (SN: 8/29/87, p.136). But the actual cause of AIDS dementia has been unknown, despite the fact that the amount of HIV-1 in the spinal fluid is related to the severity of dementia.

"Even though there is this dementia in AIDS, it is clear that HIV-1 does not infect the neurons themselves," Mark E. Gurney of the University of Chicago told SCIENCE NEWS. Gurney, co-worker Mark R. Lee and David D. Ho of the Cedars-Sinai Medical Center in Los Angeles studied the interaction between HIV-1 and factors that are known to control neuronal growth and function. What they found, says Gurney, could help explain what is causing AIDS-related dementia.

"[The HIV-1 virus] infects the monocyte cells in the brain," says Gurney. "So how does an infected monocyte, which isn't a nerve cell, cause dementia? There must be an indirect mechanism." That mechanism, Gurney and his coauthors report in the Aug. 28 Science, may be the competition between the virus and a protein called neuroleukin.

First described by Gurney and others in 1986, neuroleukin is secreted by lymphocyte cells, stimulates antibody production by other blood cells and influences the growth of neurons in embryos. In the recent study, the scientists found that a segment of neuroleukin is very similar in structure to the HIV-1 component called gp 120. Experiments using 10-day-old chick embryos showed that the addition of either whole HIV-1 or gp 120 suppressed the activity of neuroleukin.

According to Gurney, the cause of the dementia in AIDS apparently differs from that of other dementias: "In Alzheimer's, you have actual death of nerve cells, and you don't in AIDS....It's as if the [AIDS-