K-T mass extinctions: Abrupt or what?

A small, barren island off the coast of Antarctica is revealing a surprising and remarkably detailed picture of the mass extinctions at the end of the Cretaceous period. These new findings, far different from the record found elsewhere in the world, were reported this week in Phoenix at the Geological Society of America's annual meeting. The preliminary results come from the first study of the Cretaceous-Tertiary (K-T) boundary in the Antarctic region, and they may help scientists decide whether a cataclysmic meteor impact killed off the dinosaurs and a significant proportion of life before 65 million years ago.

In the past, most paleontologists who have studied the K-T boundary in Europe, North America and other midlatitude sites have found that species started to die out abruptly and in record numbers at the close of the Cretaceous period. However, William Zinsmeister of Purdue University in West Lafayette, Ind., and his colleagues found a different extinction pattern on Seymour Island, near the Antarctic peninsula.

"In high latitudes," says Zinsmeister, "you just don't see the marked extinctions at the K-T boundary. You see a gradual change, a gradual dropoff."

In the last decade, scores of scientists from the fields of geology, paleontology and even astrophysics have jumped into an often-heated debate over the cause of the K-T extinction (SN: 2/1/86, p.75). Luis Alvarez, a Nobel prize-winning physicist from the University of California at Berkeley, launched the present controversy around 1979 when he proposed that the extinctions resulted from the impact of a comet or meteorite. Such a catastrophic crash would have thrown a cloud of debris into the atmosphere, blocking out sunlight and abruptly killing off much of Cretaceous life.

Recently, however, many paleontologists have argued that the extinctions may not have been so abrupt. And, says Zinsmeister, the Seymore Island data will add to the debate. "It's going to be a hot one," he told SCIENCE NEWS before delivering his paper to the conference.

Zinsmeister adds, though, that a gradual decline of life near the South Pole would not preclude the possibility that species closer to the equator died off abruptly. If Antarctica was meteorologically isolated from the rest of the world, as it is today, "whatever mechanism caused the apparent abrupt mass extinctions at midlatitudes may have been damped by the time it got to the high latitudes," he says.

But the Seymore Island K-T boundary might offer a truer picture of the mass extinction pattern than do the European K-T boundaries. In Europe, because comparatively less sediment accumulated over a given period of time, crucial time periods have been condensed into rock segments only a few centimeters thick, as compared with 30-meter-thick sections on Seymore Island. Says Zinsmeister, "These gradual changes that you see in the Antarctic may actually have occurred in Europe but you don't see them because the picture isn't as clear."

However, Marilyn Kooser from the University of California at Riverside, who also worked on the Seymore Island fossils, cautions that the conclusions are preliminary and that the data await statistical analysis.

According to Kooser, the Seymore Island data also highlight a problem concerning the definition of the K-T boundary. Traditionally, scientists have used several different methods to define the boundary, sometimes relying on the last appearance of large, nautilus-like creatures called ammonites, sometimes relying on other benchmarks, such as a characteristic change in microfossils. However, when the team used the microfossil definition on Seymore, they found ammonites 20 meters above the boundary. — *R. Monastersky*

Interleukin-1's secret message to ACTH

Two decades after scientists began studying the link between immunity and stress, three new reports strengthen the idea that the protein interleukin-1 (IL-1) carries regulatory signals between the two systems. Conflicting scientific data, however, show that the way in which this messenger service operates is still unclear; scientists do not know whether or not IL-1 acts directly on the pituitary, the body's so-called "master gland" nestled under the brain.

A related protein, interleukin-2, has received more attention because of its reported success in treating cancer (SN: 1/17/87, p.44). But studies have shown that IL-1, also secreted by immune system cells, has broad influence within the body. During stress from infection or trauma, it induces fever, inflammation and the need to sleep — all important in healing.

In addition to its immune system functions, IL-1 apparently can induce the pituitary gland to secrete adrenocorticotropic hormone (ACTH) into the bloodstream. Activated by the circulating ACTH, the adrenal glands — which lie atop the kidneys — release other hormones that affect the stress response. In this capacity, IL-1 becomes a primary chemical signal between the body's immune and hormone-release systems during physical, and perhaps emotional, stress.

The three studies reported in the Oct. 23 SCIENCE attempt to clarify whether IL-1 acts directly on the pituitary gland in ACTH release, or acts indirectly by triggering the brain's hypothalamus to secrete chemicals that then activate ACTH release from the pituitary. But the results are contradictory, and while they add insight to IL-1's role in ACTH release, they fail to explain how that process works.

Researchers at Stanford University and the Salk Institute in La Jolla, Calif., incubated pituitary cells from rats with either human or mouse IL-1, and then measured the levels of ACTH in the cultures. Stanford's Robert Sapolsky and his colleagues report that IL-1 did not cause ACTH secretion. But the cells did secrete ACTH when they repeated the experiments using corticotropin-releasing factor (CRF) instead of IL-1. This factor is produced by the hypothalamus region of the brain in the presence of the interleukin.

This, say the scientists, is evidence that IL-1 does not act directly on the pituitary, but instead acts on the brain, which then sends CRF to the pituitary as a secondary messenger to switch on ACTH release. Those findings are supported by a report, also in SCIENCE, from researchers at Free University in Amsterdam, the Netherlands, and at Schweizerisches Forschungsinstitut in Davos-Platz, Switzerland — the group that first reported in 1986 that IL-1 from the immune system somehow induces ACTH secretion during stress.

However, scientists at Walter Reed Army Institute of Research in Washington, D.C., report in the same issue that human IL-1 does stimulate cultured rat pituitary cells to release ACTH. But "the apparent conflicting results" may be two sides of the same coin, says Michael D. Lumpkin of Georgetown University in Washington, D.C. In an accompanying commentary, Lumpkin points out that there are two structural forms of IL-1, and that one may activate the brain, while the other affects pituitary function. Another intriguing possibility, he says, is that the results may reflect sex differences between the pituitary-cell donors used for the studies, since there is evidence that estrogen makes cells more susceptible to substances that stimulate the release of ACTH.

"What's clear to everyone is that the immune system can turn on the stress response," Sapolsky told SCIENCE NEWS. "But everyone's grappling with the mechanism." He also wonders about the "logic" behind the IL-1/ACTH connection, given that some hormones have been shown to decrease immunity. "Why, during stress, should you want to suppress the immune system?" he says. "It is a fascinating issue, because people under stress get sicker more often." — *D.D. Edwards*