ern Methodist University, had turned up evidence of eight pachyrhinosaurs from a quarry not 16 meters square.

Paleontologists were aware of the dinosaur bone area, named the Kikak-Tegoseak bed, but not its remarkable density. Previously Alaska's horned dinosaurs have been discovered one at a time. "Finding that many skulls [of] ceratopsians stacked one on top of another is a pretty unusual situation," says Roland Gangloff, curator of earth sciences at the University of Alaska Museum, a co-sponsor of the expedition. Gangloff and his colleague David W. Norton, operator of the Arctic Rim Research in Fairbanks, first found the bed in 1994, after following a trail of bone fragments from the river's edge up a sheer, eroding bluff.

This year's discovery of eight individuals makes the site the largest collection of ceratopsians ever found above the Arctic Circle. "It's probably a huge bone bed," Fiorillo says, "and we're looking at a little, tiny part of it." Gangloff goes further, adding that the entire Colville region "will someday be recognized worldwide as one of the greatest dinosaur fossil accumulations in the world." The full size of the find remains to be seen. This summer's excavation was limited by time, manpower and the rigors of working on isolated tundra. Reaching the bed required daily climbs up a mud-slicked bluff. Work was also slowed once by a midnight visit from a bear. Such rugged conditions meant in the past that many fossils went uncollected. But this year a heavy-lifting Chinook helicopter and the cooperation of the U.S. Army made the extrications possible.

Fiorillo's team retrieved parts of at least three skulls and other skeletal material, including leg bones, ribs and vertebrae, much of it so jam-packed that researchers were finally forced to set aside delicate tools for pickaxes and to sacrifice surrounding bone to retrieve a reasonable sample.

Among the assortment were eight bones that looked like boccie balls. These occipital condyles—distinctive, spherical bones characteristic of ceratopsians—were part of the ball joint that supported the horned dinosaur's weighty head. Each condyle discovery represents another skull—and likely skeleton—lying below the earth, Fiorillo concludes. His preliminary examination suggests that the Arctic pachyrhinosaurs were close in age and probably died together in a catastrophe, such as a flood. It provides the first evidence that horned dinosaurs north of Alberta, Canada, behaved gregariously.

"We've been waiting for this for some time," says ceratopsian expert Peter Dodson of the University of Pennsylvania, referring to Fiorillo's find, which has yet to be published in a scientific journal. Although horned dinosaur remains have previously been found in the Arctic, Dodson notes, "we had not really learned very much other than that they were present." Now, he remarks, "we're going to learn something definitive."

Sonya Senkowsky, a writer based in Anchorage, was also a volunteer on the expedition.

HEADING NORTH FOR DINOSAURS

The Kikak-Tegoseak site, between 69 and 70 degrees north latitude, lies about 20 miles from where Shell Oil geologist Robert Liscomb found the region's first dinosaur bones in 1961. Liscomb died in a rock slide the next year, and his discovery went unnoted for decades. Paleontologists finally confirmed in the mid-1980s that dinosaurs were in the Arctic.

The unprecedented opportunity to examine a group of Arctic horned dinosaurs could also answer questions about how high-latitude dinosaurs adapted to the Cretaceous Arctic. (The climate was more temperate then, similar to weather today in southern Alberta, Canada.) It will also add to a picture of how all dinosaurs responded to climate change.

IMMUNOLOGY

Subduing Suppressors

SILENCING CERTAIN IMMUNE CELLS COULD DEFEAT DISEASE **BY LISA MELTON**

For decades, scientists have tried to manipulate the immune system to fight disease, but finding the right tools to crank up or slow down immune cells hasn't been easy. Now immunologists may have finally struck gold, in the form of a white blood cell known as a regulatory, or suppressor, T cell. Such cells are the levers that quiet the immune system. Keep them subdued, some scientists predict, and it soon will be possible to wipe out intractable pathogens that cause hepatitis C, HIV/AIDS and tuberculosis and even annihilate cancer cells.

Numerous laboratory studies in the 1970s

proved that suppression existed; unfortunately, there was nothing to distinguish these cells from other, similar T cells in the body. And because the experiments were hard to reproduce, immunologists eventually concluded that suppression did not exist.

Until 1996, that is, when Shimon Sakaguchi of Kyoto University in Japan showed that regulatory cells are present normally in the body as a type of CD4 T lymphocyte (also known as a helper T cell). Crucially, Sakaguchi also found a common identification tag: the CD25 molecule. Nobody yet understands exactly how suppressor cells work, but they protect us from autoimmune diseases by stifling harmful, self-reactive cells.

When it comes to fighting off tumors or chronic infections, however, the influence of suppressor cells is detrimental. "In immunology it's always a question of balance," observes Ethan Shevach of the National Institutes of Health. "In certain situations, it might be better to get rid of them." In chronic infections, for example, "microorganisms may be using T suppressor cells as a window of escape," says Kim J. Hasenkrug of the NIH Rocky Mountain Laboratories in Hamilton, Mont. Indeed, in many viral and bacterial infections, the numbers of suppressor T cells skyrocket—evidently, they are taking the bite out of killer T cells designed to destroy invaders. The trick is to silence regulatory cells and thereby tip the balance to killer cells.

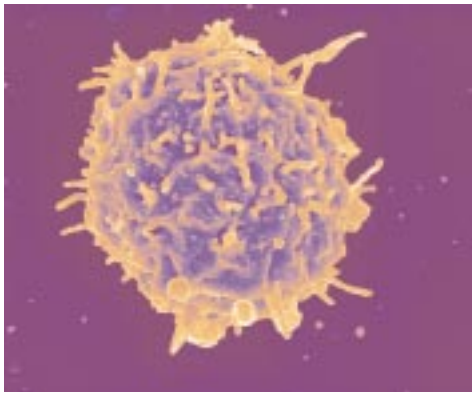
To test different tactics, Hasenkrug infected mice with the Friend leukemia virus, which leads to high numbers of suppressor cells. Using antibodies that block TGF-beta and IL-10 receptors—molecules that regulatory cells need to do their job—proved a winning strategy. The number of CD25 cells plummeted, and mice regained their ability to reject tumors. In skin infections with the parasite *Leishmania major*, Shevach has had similar results using an antibody that depletes CD25 cells.

In the clinic, however, the ideal is to restrain suppressor cells, not eliminate them. Sakaguchi has developed a monoclonal antibody that can manage just that. Rather than deplete the cells, the antibody blocks their function by locking onto a molecule on their surface called glucocorticoid-induced TNF receptor (GITR). This past July at a Novartis Foundation meeting in London, Sakaguchi reported that when mice bearing tumors were injected with anti-GITR, the invigorated immune response shrunk the tumors. "The animal data are very promising, and pharmaceutical companies are pursuing this molecule intensively," he notes.

Some researchers are trying to find out if cancer patients might benefit from silencing suppressor cells. "It is an attractive hypothesis, but we don't know yet," says Jacques Banchereau of the Baylor Institute for Immunology Research in Dallas. In a small clinical trial involving advanced melanoma, Banchereau found that vaccines made from patients' own dendritic cells loaded with bits of tumor protein triggered an immune response to the tumors. The results are dramatic: of the 10 patients injected, nine remained free of disease for 10 weeks, and four are still alive nearly four years after treatment. In other experiments, "we have seen skin, liver, brain and lung tumors disappear," Banchereau claims. "It's something amazing to do with regulatory cells," he thinks, though for the moment, he is unwilling to give away the details.

The hope is that the fruits of such intensive research may soon hit the clinic. "In immunology, we understand a great deal, but unfortunately the number of drugs that have emerged from that understanding is rather small," Shevach acknowledges. But if the 30 years of ignominy are taken into account, then the time may be ripe for regulatory T cells to take center stage.

Lisa Melton, based in London, did her postdoctoral work in immunology during the "dark ages" for suppressor cells.



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VACCINES "HAT FIT TO A "T"

Vaccines would also benefit from having fewer regulatory T cells kicking about. "When you have a suboptimal vaccine, it would be great to get rid of CD25 cells, at least temporarily," points out Ethan Shevach of the National Institutes of Health. Many experimental vaccines, especially for HIV, malaria and mycobacterium tuberculosis, have not made it to the clinic, because they are not protective enough. Keeping the CD25 cells out of the picture would allow the immune system to build up a vigorous response.

CREDIT