El Niño's Atlantic Counterpart

Call it son of El Niño — or, perhaps, brother or cousin. Scientists have found in the Atlantic Ocean a phenomenon that is the image of El Niño — the periodic change of water temperature and winds that occurs in the Pacific Ocean, triggering ecological upsets worldwide.

Scientists report in the July 17 NATURE that they measured El Niño-like conditions in the Atlantic in 1984. Near the equator, the surface water was unusually warm and winds were very light. The band of clouds that spans the ocean from South America to Africa hung over the equator longer than usual that spring — causing heavy rains in eastern Brazil and western Angola and intensifying drought to the north, near the Amazon and in western sub-Saharan Africa.

This Atlantic phenomenon occurs periodically, though not as often as El Niño, according to George H. Philander, senior oceanographer at the Geophysical Fluid Dynamics Laboratory at Princeton (N.J.) University, who wrote the first in a series of six articles in NATURE describing the 1984 event. Whereas El Niño occurs every three to five years, the last Atlantic event prior to this one appears — from weather and ocean temperature records — to have been in 1963, Philander reports.

As El Niño in 1982-83 decimated the anchovy, sardine and mackerel populations off southwest Peru, the warm Atlantic in 1984 killed the same kinds of fish near Angola, according to biological oceanographer Richard T. Barber of Duke University's marine lab in Beaufort, N.C.

And as El Niño altered weather around the world (SN:2/26/83,p.135), the Atlantic event affected rainfall and temperatures in western Europe and Africa. However, these effects were relatively mild, Philander says, because the Atlantic is three times smaller than the Pacific.

Although the cause of oceanic warming spells is not well understood, scientists believe it has to do with the interaction of sea-surface temperatures and winds. Normally, in both the Pacific and Atlantic, the water is warmer on the west side of the basin than on the east, and the surface winds blow westward.

During 1984 in the Atlantic — as during El Niños — the eastern basin warmed to match the west, and winds were unusually weak. Near Angola, where in June 1983 the water averaged 21°C, the average temperature in June 1984 was 26°C — the same as the water near Brazil.

The question is, which comes first, warm water or weak winds? A meteorologist would say it's the water, according to Philander, because winds are drawn to the warmest areas. But an oceanographer would say the winds change first, because without them, the warm water does not move west. "It's a chicken-and-egg situation," Philander says.

The weather changes because the warmer ocean holds the east-west band of clouds near the equator. The band usually migrates about 15° northward in March, as the sun warms the water to the north. In 1984, the Atlantic clouds did not move north until late June.

However, a second major weather change caused by El Niños did not occur in the Atlantic. During El Niño, clouds over the western islands are drawn eastward to the warmer ocean, causing drought in Australia, Indonesia and the Philippines. In 1984, the clouds over Brazil did not similarly move east — probably because Brazil's large, warm land mass was enough to hold them, Philander says.

"It's as though, in the Atlantic, it was half an El Niño," he says.

Philander hopes the new data will help scientists understand El Niños. "By comparing the Atlantic and ... Pacific events," he says, "we should be able to understand both of them better."

— M. Murray

Second T cell receptor found

In the complex orchestra of the immune system there are instruments whose roles are unknown, and there are sounds with no known source. One instrument emerged from behind the curtains at last week's 6th International Congress of Immunology in Toronto. While it generated quite a bit of excitement at the meeting, researchers do not yet know whether it plays a minor or major role in the immune system.

The newly discovered instrument is a protein molecule on the surface of T cells. One of the two major arms of the immune system, T cells are primarily involved in ridding the body of foreign organisms — fungi, parasites, cancer cells, foreign tissue and virally infected cells. Each T cell recognizes and works against one subtype within these groups, through a specific receptor on its cell membrane.

At the Toronto meeting, Michael B. Brenner of Harvard University and Leonard Chess of Columbia University in New York City presented research by their laboratories indicating that there is a second class of T cell receptors. The experiments are also described in the July 10 NATURE.

The second receptor, says Brenner, is "a cousin but clearly distinct" from the first. While it opens up a whole new area of investigation for immunologists — detailing the function of the receptor and its role in disease — Brenner and Chess say it is too early to predict the clinical applications of the finding.

The second receptor had been hotly pursued following the discovery of a "mystery gene" by Haruo Saito and Susumu Tonegawa at the Massachusetts Institute of Technology in Cambridge in 1984. Unlike the two genes that together code the first receptor, this gene is active in T cells at the time of their development. Like the first two genes, it can create many different proteins by rearranging itself. Prior to last week's announcement, the gene's function was unknown. Now that the product of the gene has been found, researchers are seeking the protein's "sound."

Brenner's and Chess's laboratories took different routes to their discoveries. Since the MIT work suggested the gene was active early in T cell development, and since the other T cell receptor appears relatively late, Chess's group worked with clones of immature cells. They looked for an intermediate step in protein production based on the genetic sequence of the mystery gene and found what they were looking for.

Brenner's laboratory used monoclonal antibodies on a preparation of T cells to "cleanse" it of the known receptor; they found a novel protein on the remaining cells that matched up with predictions of what the mystery gene would produce. In both cases, the new receptor was found in conjunction with another cell-surface molecule that the other receptor requires in order to be viable, indicating that the new receptor may have a similar recognition function.

"It used to be you started with a phenomenon, looked for a protein, then looked for the gene," says Chess. Having taken the reverse route in this case, the researchers now need to determine exactly what the protein does. "We have to know more about its function before we can say anything about its effect. We're hoping it will have some impact on our understanding of the immune system," says Chess.

Says Philip Halloran, an organ transplant specialist at Mt. Sinai Hospital in Toronto, "This is a new window on how T cells work. How important it will be depends on what we can see through the window."

— J. Silberner