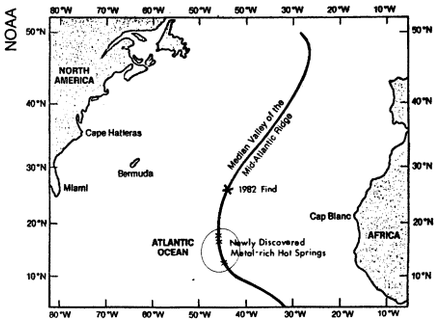
Seafloor vents found in the Atlantic

Imagine trying to find a "mineral deposit on land from a balloon a mile or so higher above the surface of the earth by towing a bucket or camera on a rope through a thick layer of clouds," says Peter Rona, an oceanographer at the National Oceanic and Atmospheric Administration (NOAA) in Miami. That was the kind of problem Rona and his colleagues faced when they spent last August on the Atlantic hunting for undersea hydrothermal vents — hot springs that gush up from oceanic ridges where new seafloor is produced. Rona's group searched for the meter-wide vents along 1,700 kilometers of the Mid-Atlantic Ridge by towing a camera and sensors on 4,000-meter cables from the NOAA ship the *Researcher*.

But the scientists found their treasure: three seafloor vents, bringing the total number of undersea geysers found in the Atlantic to four. The findings, announced last week, show that the Pacific does not have a monopoly on seafloor hot springs. This means that vents at slow-spreading ridges like that found in the Atlantic, which were previously dismissed as being nonexistent or inconsequential, could in fact make a significant contribution to the chemical and thermal structure of the ocean, the researchers say.

NOAA



Three newly discovered seafloor vents lie about 3,000 kilometers southeast of Miami, along the Mid-Atlantic Ridge.

Seafloor vents are found in mid-oceanic rifts where molten rock, or magma, upwelling from the mantle, spreads on both sides of the ridge to form new oceanic crust (SN: 1/12/80, p. 28). Scientists believe that vents are formed when seawater permeates the newly formed crust through cracks or faults, is heated by the hot volcanic rocks and is then spewed out, laden with minerals and metals leached from the crust. Researchers home in on vents by looking for regions of hot water containing high concentrations of manganese and other soluble metals. Vents are marked by surrounding mounds of copper, iron and other ores that are precipitated when the heated mineral solution hits the cold ocean water. In the Pacific at least, they are also surrounded by a community of unique geothermal-based animals.

Since the existence of these hot springs was first proposed in 1965, over 80 vents have been found in the Pacific. One vent in the Atlantic was discovered in 1972 and examined in 1982. Scientists have concentrated their searches in the Pacific because the seafloor there spreads relatively quickly — from 6 to 25 centimeters per year, up to 10 times faster than its slow-spreading counterparts — creating hotter, more violent vents that are easy to spot.

"The visually spectacular vents in the Pacific have detracted attention from the slow-spreading portion of the ridge system, which much of the scientific community wrote off as being unimportant in relation to venting," says Rona. The recent findings show that venting does exist in the Atlantic, and the evidence indicates that these vents must be included in calculations of the ocean's chemical and thermal budget, he adds.

The discovery of Pacific venting provided a "missing" source of ocean chemicals since, until then, scientists had thought that these chemicals came only from the atmosphere and river runoff. However, the Pacific vents don't account for all the gaps in the chemical budget. The contributions from venting at slow-spreading ridges may also be significant, says Rona, since these kinds of rifts make up over half of the ocean's ridge network.

Rona's group, which includes scientists from Cambridge University in England and three other institutions, plans another cruise next July to pinpoint the vent locations and to quantify their chemical and thermal outputs. Meanwhile, the researchers are preparing to publish in the JOURNAL OF GEOPHYSICAL RESEARCH photographs of the vent site discovered in 1972 showing clamlike forms — the first tentative indication that Atlantic vents may be surrounded by life forms like those observed around Pacific vents.

—S. Weisburd

Immunity factor linked to nerve disease

Some scientists have suspected that what brought down baseball player Lou Gehrig and other victims of the progressive neurological disease called amyotrophic lateral sclerosis (ALS) was an autoimmune factor — something the body generates that attacks healthy cells. Such a factor has now been identified, though whether it causes the deadly disease or is a secondary effect remains to be seen.

Motor neurons, movement-controlling nerves that in humans can be as long as 3 or 4 feet, extend from the spinal cord out to muscles. ALS researchers have searched in vain for an antibody against the nerve cell body, located within the spinal cord. Mark Gurney, Jack Antel and colleagues at the University of Chicago found the new factor by looking at what happens at the ends of the nerve cells, out in the muscle where the disease's paralyzing effects are manifested.

The researchers inoculated mice with botulinum toxin, which induces the ends of motor neurons to sprout new growth. They followed that up with injections of blood serum from ALS patients, healthy people and people with diabetes-caused nerve disease.

Less sprouting occurred in mice given serum from 11 of the 25 ALS patients than occurred with the other serums tested, the researchers report in the Oct. 11 NEW ENGLAND JOURNAL OF MEDICINE. The differential reaction convinced them that at least some ALS victims have a nerve-growth inhibiting factor, and the researchers have evidence that the factor is an antibody. But they're not yet blaming the antibody for ALS.

Among the unanswered questions: Why did they fail to find, among the patients whose serum inhibited sprouting, a correlation between the level of inhibition and the progression of the disease, and why didn't the serum from some of the ALS patients show any inhibition?

"The antibody could arise secondarily, so that it's not the primary cause of the disease," says Antel. "Or it may be that the system of measuring the antibody isn't as sensitive as it could be yet."

The researchers also identified a protein secreted by muscle cells in culture that may be the operative growth factor, and found that antibodies to this protein inhibit nerve sprouting. Blood from three ALS patients showed immune activity specifically against this protein.

At last week's Society for Neuroscience meeting in Anaheim, Calif., Gurney reported that a monoclonal antibody against the suspected motor-nerve growth factor suppressed sprouting. Now, notes Antel, "We can once and for all test the theory that growth factors are involved in the disease.

"Once we can isolate and characterize this factor, then we're in a position to see if there is something defective with the factor or at least show that there are autoantibodies associated with ALS. We're in the preliminary stages of looking at all the various places in which an antibody could interfere with a growth factor's effect on its target."

The research has generated cautious optimism. "It's a very exciting observation," notes Carl Leventhal of the National Institute of Neurological and Communicative Disorders and Stroke in Bethesda, Md. "But it needs confirmation."

"If this work can be confirmed, it will have major consequences," says ALS researcher Lewis P. Rowland of Columbia University in New York. "It might be very important, or it might be a bust. We'll have to wait and see."

—J. Silberner