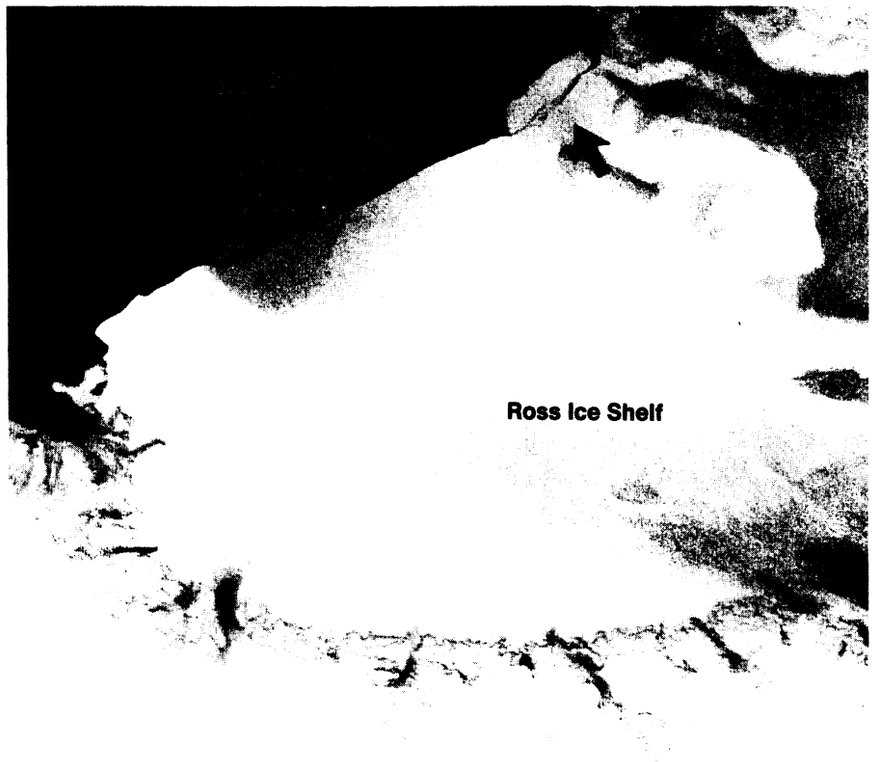
Huge ice cube in Antarctic waters

An iceberg twice the size of Rhode Island has broken off the Ross Ice Shelf in Antarctica, the National Science Foundation reported last week. In this infrared satellite image, taken Oct. 13, the arrow shows the iceberg in the process of separating from the shelf. The iceberg measures approximately 98 miles long and 25 miles wide, with an estimated average thickness of 750 feet.

Since the image was taken, the iceberg has drifted 25 nautical miles to the northwest. At the National Oceanic and Atmospheric Administration, remote-sensing experts have monitored this piece since May and are not sure where it will drift. For now, it presents no hazard to shipping, and it may be years, if ever, before it drifts far to the north.

The Antarctic has several ice shelves, which are thick sheets of floating, freshwater ice that have slid off the Antarctic continent (located below the mountain range in the satellite image). Thousands of years' worth of snow accumulation forms the continental ice cap. At a snail's pace, it flows down to the sea, where it replenishes the parts of the ice sheet that break off.



NOAA/NESDIS/NCDC/SDST

Scientists report that the number of extremely large bergs has dramatically increased in the last year and a half. This piece joins four others that are floating at various points around the Antarctic. Two of these have drifted north, near the Falkland Islands off the coast of South America, and may soon enter shipping lanes. Researchers cannot explain why so many large pieces are now breaking off. They speculate that it may be related to an apparent warming trend in global temperatures.

Lipoprotein findings may solve one riddle . . . and pose another

A surprising similarity in structure between two blood proteins could help explain how some people develop atherosclerosis, or hardening of the arteries, scientists announced this week. But in giving some answers, the new findings raise other questions.

Scientists from the University of Chicago and Genentech, Inc., of South San Francisco report in the Nov. 12 NATURE that they have determined the sequence of amino acids found in apolipoprotein(a), a subunit of lipoprotein(a). Found in the blood, the latter has been tied to the development of atherosclerosis – but the exact mechanism is unclear. Another subunit of lipoprotein(a), called apolipoprotein B-100, already has been fingered as a culprit in atherosclerosis (SN: 2/7/87, p.90).

This week's report contains the first complete sequence of apolipoprotein(a) and offers some insight into the atherosclerosis dilemma. Coauthor John W. McLean of Genentech told SCIENCE NEWS that a report from the group earlier this year had hinted that apolipoprotein(a) might be structurally related to the blood protein plasminogen. Subsequent work has confirmed that similarity, with possible medical consequences, he says.

Plasminogen – the precursor of a blood enzyme that dissolves clots – contains unusual amino acid sequences called kringles, which resemble a Danish cake by that name. Apolipoprotein(a), say the Genentech/Chicago researchers, inexplicably has kringles as well. McLean, who calls the implications of these findings "tantalizing," says structural similarities between the two could mean that apolipoprotein(a) somehow affects plasminogen's role in clot dissolution.

In an accompanying commentary, Nobel laureates Michael S. Brown and Joseph L. Goldstein, from the University of Texas Southwestern Medical Center in Dallas, agree that these "astounding" results could "provide the long-sought link between lipoproteins and the clotting system." Some scientists believe that mini-clots on blood vessel walls promote atherosclerosis.

On the basis of their new data, the researchers are planning studies to investigate the possible physiological functions of apolipoprotein(a) and how the protein interacts with known receptors on blood vessel walls. Of interest, says McLean, is an understanding of why different sizes of apolipoprotein(a) are found in different concentrations among individuals. He adds that the protein's structural similarity to plasminogen also could cast doubts on data from already completed studies that measure one or the other.

Beyond the immediate questions about apolipoprotein(a)'s influence in atherosclerosis is its apparently odd place in evolution. It has 37 repeat copies of one kringle section placed end to end, making the protein larger than what would seem necessary. And thus far, the protein seems useless. The gene coding for apolipoprotein(a) has not been found in species lower than primates, and may have first appeared about 40 million years ago, say the authors. "Why is nature carrying along this huge burden of a protein with no known function?" asks McLean. He speculates that there may be "something in man's history that makes [the protein] important for survival."

The current study does not suggest any potential therapy to prevent atherosclerosis, says McLean. But if there is a correlation between susceptibility to atherosclerosis and apolipoprotein(a) levels, he thinks an assay for the protein could help screen for patients who should adopt exercise routines and watch their nutrition. *– D.D. Edwards*