Astronomy

Ron Cowen reports from Houston at the annual Lunar and Planetary Science Conference

Matching meteorites with their parents

Astronomers report the first direct link between a set of meteorites and a particular location in the main asteroid belt, which lies between Mars and Jupiter.

Michael J. Gaffey, Kevin L. Reed and Michael S. Kelley of the Rensselaer Polytechnic Institute in Troy, N.Y., made their chance discovery after observing the asteroid Apollo (3103) 1982BB as it passed within 30 million kilometers of Earth in July 1991. Obtaining the asteroid's spectra with the NASA Infrared Telescope atop Mauna Kea in Hawaii, the researchers found evidence that the body contained a type of iron-free, magnesium-rich silicate known as enstatite. That mineral also predominates in a group of meteorites called enstatite achondrites. Gaffey's team believes these meteorites represent fragments of the kilometer-size asteroid they studied.

At first glance, that identification wouldn't seem to link meteorites with main-belt asteroids, since (3103) 1982BB does not now reside in the belt. But it may have once, says Gaffey, since this asteroid's orbit intersects the Hungaria region, one of the innermost parts of the asteroid belt.

Researchers had previously suspected that enstatite achondrites came from the Hungaria region, but they discounted the idea because the surfaces of meteorites falling to Earth directly from this locale would have been bombarded by cosmic rays for tens of millions of years. Cosmic-ray studies, however, have shown that enstatite achondrites experienced such bombardment about 10 times longer.

Gaffey's team conjectures that the Apollo asteroid represents a chip off an old rock, a runaway fragment of a larger resident of the Hungaria region. As such, it could have served as a reservoir for the enstatite meteorites, which at some point broke off its surface, accounting for their longer exposure in space. Thus, Hungaria asteroids indeed appear to be the parent bodies of these meteorites, Gaffey concludes. The link may enable researchers to infer the composition and temperature of this part of the solar system during the formation of the planets and asteroids, he notes.

Riding the rapids on Mars

Come one, come all, on a grand tour of a major Martian waterway! Ride the rapids in Maja Canyon, spend lazy days fishing in the lakes of Lunae Planum and Xanthe Terra. Why not take a relaxing swim in the waters of western Chryse Planitia?

Such a scenario might not have seemed farfetched during a brief period several billion years ago — an era, some researchers believe, when water carved the now-dry canyons and channels that scar the Martian surface. To understand the nature of these proposed ancient waterways, Rene A. De Hon and Eric A. Pani of Northeast Louisiana University in Monroe focused on Maja Valles, a system of canyons and channels some 1,600 kilometers long.

The researchers used standard fluid-flow equations, accounting for the region's slope and Martian gravity, to calculate how long it would take water collecting at Juvantae Chasm — an uphill canyon — to reach the Chryse Planitia basin, where it emptied. Nowadays, notes De Hon, such a journey would span a mere 44 hours, thanks to the direct path apparently carved by the ancient flow. But the initial rush of water would have taken a far more circuitous and time-consuming route to its final destination. Water would have temporarily collected in ponds, craters and depressions along the way until it spilled over onto adjacent regions.

De Hon and Pani calculate it would have taken some 430 Earth days for 62,000 cubic kilometers of water — the minimum amount believed held by the Juvantae Chasm canyon — to move through Maja Valles. Such a time scale appears long enough to have significantly eroded the Martian surface, they say.

Biomedicine

Enzyme dissolves cystic fibrosis phlegm

An experimental drug that cystic fibrosis patients inhale in aerosol form can cut through the thick mucus that clogs their lungs, allowing them to breathe more easily and deeply, according to the results of a preliminary study.

A team led by National Institutes of Health researcher Ronald G. Crystal found that twice-daily inhalations of the drug, called DNase I, improved the lung functions of all 16 of the adult subjects. Moreover, the treatment had no noticeable side effects, the researchers report in the March 19 NEW ENGLAND JOURNAL OF MEDICINE.

DNase I is a naturally occurring body enzyme that chops up DNA, a major constituent of the sticky phlegm that accumulates in the lungs and other organs of cystic fibrosis patients. The DNA is left behind by infection-fighting white blood cells that migrate to the lining of these organs, only to die after finishing their job. Because DNA is very viscous, it makes the mucus hard to cough up, increasing patients' vulnerability to further lung infections.

DNase I treatment thinned the lung mucus of all of the patients participating in the new study, Crystal and his colleagues found. They also detected higher levels of degraded DNA in the patients' sputum following DNase I therapy.

"This is very exciting," says Robert J. Beall, vice president for medical affairs of the Cystic Fibrosis Foundation in Bethesda, Md. "It's the first [mucus-degrading drug] for which we've had such encouraging preliminary data."

Crystal and his colleagues reported early results from the DNase I study at a conference on cystic fibrosis last year (SN: 3/2/91, p.132). Genentech, Inc., the South San Francisco, Calif., biotechnology company that produces DNase I through genetic engineering, is now expanding tests of the drug to include 900 cystic fibrosis patients at 50 U.S. medical centers. If the additional tests confirm DNase I's efficacy, the company plans to file for Food and Drug Administration approval of the drug early next year.

Defect in cystic fibrosis protein found

Biochemists studying a specific genetic mutation that causes most cases of cystic fibrosis have come up with a new lead in understanding the disease.

Peter L. Pedersen and his colleagues at the Johns Hopkins University School of Medicine in Baltimore have found that the mutation leads to the deletion of a crucial amino acid from the protein involved in the disease. The protein, cystic fibrosis transmembrane conductance regulator (CFTR), usually helps cells maintain normal levels of chloride. But Pedersen's group found that when the protein lacks this amino acid, it doesn't fold into its correct shape and loses its function. The mucus buildup of cystic fibrosis results. The researchers report their finding in the March 25 JOURNAL OF BIOLOGICAL CHEMISTRY.

Scientists first discovered the CFTR gene more than two years ago (SN: 9/2/89, p.149). Further studies indicated that mutations in the CFTR gene lead to defective CFTR proteins that cannot move from the interior of a cell, where they are made, to their places in the cell's outer membrane (SN: 11/24/90, p.324).

Pedersen's group confirmed that mutant CFTR proteins have a three-dimensional shape that differs from normal CFTR proteins. They also showed that this altered shape prevents the mutant proteins from binding to ATP, the molecule that usually provides them with chemical fuel.

Pedersen says his group's finding suggests that structure-correcting proteins called "chaperonins" offer promise as drugs to straighten out the CFTR defects behind cystic fibrosis. Chaperonins play a natural role in reshaping mutant proteins and in keeping newly made proteins from tangling.