A surprising encounter of the NEAR kind

An analysis of pictures and data obtained during last summer's flyby of the asteroid 253 Mathilde reveals that this carbon-rich, heavily-cratered body is only about half as dense as rocky asteroids. This finding—the first accurate measurement of the density of a carbonaceous asteroid—could prompt a reassessment of the geophysical history of these wandering planetesimals.

The Near Earth Asteroid Rendezvous (NEAR) spacecraft skimmed within 1,212 kilometers of Mathilde on June 27, 1997. An initial analysis of the data hinted that the dark body, which reflects light only about half as well as a charcoal briquette, has a low density (SN: 7/12/97, p. 29).

Now, two reports in the Dec. 19, 1997 SCIENCE indicate that the asteroid is highly porous, suggesting that it either was formed from loosely packed fragments or has been pulverized into a "rubble pile" by repeated impacts with other celestial bodies.

One study, led by Donald K. Yeomans of NASA's Jet Propulsion Laboratory in Pasadena, Calif., analyzed the gravitational pull of the asteroid on the NEAR spacecraft as it sped along at about 10 km per second. By measuring the slight acceleration of the craft as it approached Mathilde and the deceleration after it passed, the scientists determined the mass of the asteroid to be about 110 trillion tons.

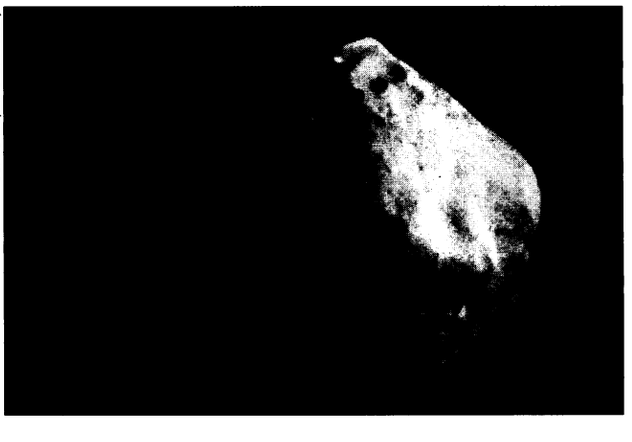
NEAR's optical measurements, part of a study led by Joseph F. Veverka of Cornell University, reveal Mathilde to be a potato-shaped body measuring some 66 km by 48 km by 46 km. Together, the two teams' measurements indicate that the asteroid's density is only about 1.3 times that of water.

A large amount of water ice preserved within Mathilde would help explain its low density, but Earth-based spectroscopic measurements of the asteroid show that there is no water locked within it.

During its flyby, NEAR observed about half of the asteroid's surface and spotted five craters with diameters of 19 km to 33 km. The largest and best-imaged of the craters is wider than the asteroid's mean radius, says Veverka. That crater, whose bottom remained in shadow throughout the 25-minute flyby, may be 5 or 6 km deep, he adds.

Although Mathilde could have been reduced to a pile of rubble by a long history of collisions with other asteroids, it's also possible that its low density is primordial, Veverka says. Mathilde's small size and low gravity create a pressure of less than 2 Earth atmospheres at its center, which is not enough to compress loose materials into a solid.

NEAR's close encounter with Mathilde took place halfway through a 3-year journey to the asteroid 433 Eros, which the spacecraft will orbit for 13 months beginning in early 1999. On Jan. 23, NEAR will swing by Earth and get a gravitationally assisted course correction, says Robert W. Farquhar, mission director at Johns Hopkins University's Applied Physics Laboratory in Laurel, Md. During the maneuver, NEAR is scheduled to pass about 330 miles above the border between Iraq and Kuwait at 10:23 a.m. local time. —*S. Perkins*



Johns Hopkins University

This four-image mosaic of asteroid 253 Mathilde was taken on June 27, 1997, from a distance of about 2,400 kilometers. The apparent slice off the asteroid's upper left surface is actually the rim of one of its large craters viewed edge-on.

Gene pushes cells into forced retirement

As they reach their later years, many people retire, a step that usually involves a significant change in lifestyle. Similarly, when cells grown in test tubes reach the equivalent of old age, many of them undergo senescence, a phenomenon in which cells stop dividing and subtly change their shape and pattern of gene activity.

Investigators suggest that senescence evolved as a way of reducing the risk of uncontrolled cell division—that is, cancer—in long-lived organisms. They also speculate that a gradual accumulation of senescent cells may explain heart disease, memory loss, skin wrinkling, and other age-related changes.

Scientists who study senescence may now have their first insight into the genes a cell needs to persist in this nondividing state.

A newly identified gene on human chromosome 4, when added to cancer cells in the lab, converts the proliferating cells into senescent cells, Michael J. Bertram of the Baylor College of Medicine in Houston reported last month at the American Society for Cell Biology meeting in Washington, D.C.

Bertram is part of a research group led by Baylor's Olivia M. Pereira-Smith that has been searching for the genes involved in cellular senescence. The quest made use of cancer cells, which presumably harbor genetic mutations that rob them of their ability to senesce.

The group found that they could induce tumor cells to senesce by fusing them to normal cells. Further work showed that adding individual chromosomes, such as number 4, produced the same transformation. The scientists then slowly closed in on the location of several putative senescence genes.

The investigators finally struck pay dirt when they found a small fragment of chromosome 4 that could trigger senescence. Within this fragment, they identified a gene that they call *mortality factor-4*, or *morf4*. When added to a line of cancer cells called HeLa cells, *morf4* induces a majority of them to stop dividing.

"It's an exciting finding. [The gene] may be quite useful in thinking about how to control tumorigenesis," says senescence investigator Judith Campisi of Lawrence Berkeley (Calif.) National Laboratory.

The predicted structure of the protein encoded by *morf4* suggests that it may be a transcription factor, a molecule that regulates the activity of genes. "Our hypothesis is that it acts in the nucleus by binding to DNA," says Bertram.

Preliminary studies, including one in which scientists fused a fluorescent tag to *morf4*'s protein, show that it is indeed present in the nucleus.

Campisi says the protein may help cells monitor the length of their telomeres, the protective DNA sequences at the ends of chromosomes. In most cells, telomeres shorten with every cell division and may serve as a clock to tell cells when they should senesce (SN: 11/25/95, p. 362).

The identification of *morf4* may also help scientists probe whether a buildup of senescent cells contributes to the aging of an organism.

"The hypothesis is gaining support, but in my mind it's still speculation," says Campisi.

With its relevance to cancer and possibly aging, cellular senescence has now hit the mainstream, say researchers. "It was kind of an obscure field for quite some time. That's not true anymore," says Campisi. —*J. Travis*