Drop time: Manipulating liquids in space

An orbiting spacecraft can serve as a unique laboratory for studying the behavior of liquid drops. In a setting in which the effects of gravity are practically negligible, researchers can observe details of how a drop responds to various forces and how drops of different liquids interact and combine.

The U.S. Microgravity Laboratory, carried aloft last month by space shuttle Columbia (SN: 11/11/95, p.308), includes an apparatus called the drop physics module. With this equipment, a shuttle crew member can use sound waves to levitate and manipulate liquid drops about the size of golf balls.

This approach provides large spherical drops—undistorted by gravity and unaffected by container walls—as the starting point for experiments. "Whatever sample you put in there is in contact with nothing but air," says Arvid P. Croonquist of NASA's Jet Propulsion Laboratory in Pasadena, Calif.

One set of experiments, put together by Taylor G. Wang of Vanderbilt University in Nashville and his coworkers, involved drop dynamics. The researchers studied the breakup of rapidly rotating drops and the transition to chaotic motion as sound waves forced stationary drops to oscillate.

In one case, the researchers observed a succession of extreme shape changes that went on for nearly half a minute. "This was much more rich and varied than the usual oscillations we see," Croonquist notes.

Wang and his colleagues also looked at the behavior of compound drops produced by injecting one liquid into another. They were particularly interested in observing fluid motion within the liquids as the compound drop's outer surface oscillated, causing its inner surface to move correspondingly.

Such investigations may lead to improved methods for encapsulating spherical objects—whether groups of living cells for hormone therapy or deuterium targets for fusion experiments.

In addition, the researchers observed the formation of a membrane at the interface created when two drops of different chemicals collided. "We can't say how good or bad the membrane is until we examine it in detail," Wang says. "But at first glance, it looks very pretty."

Several experiments developed by Robert E. Apfel of Yale University and his colleagues focused on the surface characteristics of liquid drops containing traces of compounds called surfactants. These additives are widely used in such products as detergents and in many industrial processes, including pharmaceutical manufacturing and oil recovery enhancement.

To get a better understanding of the molecular forces acting in the surface layer of a water drop laced with surfactant, the researchers studied the oscillations created when a drop is squeezed acoustically, then released. — *I. Peterson*

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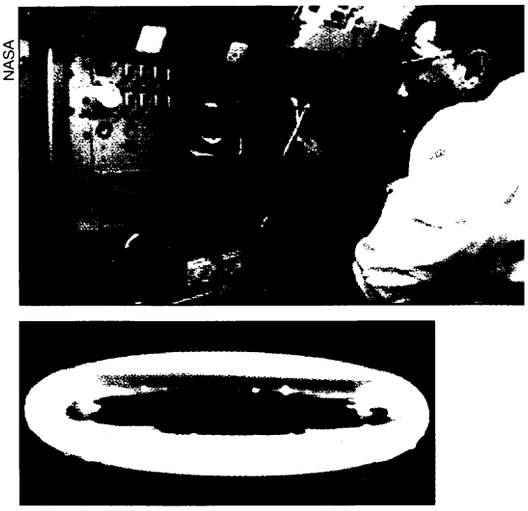

Top: Payload specialist Albert A. Sacco Jr. monitors an experiment in the drop physics module aboard space shuttle Columbia. Bottom: Sound waves deform a liquid drop into an elongated shape.

Molecules bind mutant huntington proteins

Using mutant proteins from people with Huntington's disease as bait, investigators have reeled in a novel brain protein that may play a role in this neurodegenerative disorder. And in a finding that may offer physicians a new diagnostic tool, a group of French researchers has found an antibody that binds tightly to the mutant Huntington's proteins and to similar ones made by people with other brain disorders.

Both discoveries, reported at this week's Society of Neuroscience meeting in San Diego and in the upcoming Nov. 23 NATURE, advance the study of CAG-repeat diseases. These neurodegenerative disorders occur when certain genes contain too many repetitions of a brief DNA sequence abbreviated CAG. The mutant genes code for proteins with abnormally long strings of the amino acid glutamine, leading to the death of specific populations of brain cells (SN: 6/10/95, p.360).

In 1993, researchers identified the mutant gene that causes Huntington's, but they have not yet identified the function of the protein, called huntington, that the normal version of the gene encodes. Most tissues manufacture huntington, which makes it difficult to explain why mutant versions of the protein kill only certain brain cells.

Now, researchers led by Christopher A. Ross of the Johns Hopkins University School of Medicine in Baltimore have identified for the first time a brain-specific molecule that binds to huntington. They screened for such molecules by putting into yeast the gene for huntington and the genes for the tens of thousands of proteins made in rat brains. "The yeast turns blue when there's a protein interaction," says Ross.

The investigators found a protein, hap-1, that grabs normal versions of huntington but binds even more tightly to the glutamine-rich mutant forms. The human version of hap-1 acts similarly.

The rat studies suggest that only brain tissue makes hap-1, though synthesis of the protein is not limited to the kind of brain cells killed in Huntington's disease. "We're still left with the problem of selective neuronal loss," says Michael Hayden of the University of British Columbia in Vancouver.

Hayden and Ross expect that investigators will soon identify other brain proteins that bind to normal and mutant forms of huntington, which should clarify how mutant versions lead to cell death. Researchers may then find ways to treat Huntington's disease by halting or slowing that death, says Ross.

In the other CAG-repeat advance, a French group identified an antibody that seems to bind only to forms of huntington that include enough glutamines, around 40 or more, to cause illness. The antibody also recognizes the glutamine-loaded mutant proteins of two other known CAG-repeat diseases, says Frédéric Saudou of the University of Strasbourg.

At a certain threshold of glutamine repeats, he suggests, huntington and the two other proteins undergo a change in shape. That transformation, which allows the antibody to attach to the mutant proteins, may provide a clue to why they cause disease, Saudou adds.

In addition to discriminating between normal and mutant versions of known proteins, the antibody recognized novel proteins in patients with two brain disorders thought to result from CAG-repeat mutations—even though the mutant genes responsible for the disorders remain unknown. Identifying those proteins may lead quickly to the genes, says Hayden.

The antibody's ability to spot abnormal glutamine strings may someday help physicians diagnose patients. "That's exciting. It allows one to rule out or rule in CAG expansion as a cause of disease," says Hayden. — *J. Travis*