

Fermat proof flaw: Fixing the details

The process of smoothing out and filling in the technical details of the celebrated proof of Fermat's last theorem, announced last June, has turned up a gap in the proof's logic.

Earlier this month, Andrew Wiles of Princeton University admitted in an electronic message to colleagues that reviewers of the proof had pointed out a number of problems, one of which remains unresolved. Nonetheless, he noted, "I believe I will be able to finish this in the near future."

"Many people have assumed that because the verification hasn't come quickly, there's actually a hole in the proof," says Andrew J. Granville of the University of Georgia in Athens. But "it's not a hole so much as something that needs filling in."

Fermat's last theorem asserts that for any whole number n greater than 2, the equation $x^n + y^n = z^n$ has no solution for which x , y and z are all whole numbers greater than zero. Despite the assertion's simplicity, proof of its validity eluded mathematicians for more than 350 years — until Wiles followed up several discoveries made by other mathematicians during the 1980s. These insights linked Fermat's last theorem to important ideas in number theory.

Wiles took advantage of these links in his announced proof of part of the so-called Taniyama-Shimura conjecture, which in turn establishes the truth of Fermat's last theorem (SN: 7/3/93, p.5).

But filling in the technical details of the proof is a matter of some delicacy. The snag that Wiles has encountered involves calculating a precise upper limit on the size of a mathematical object called the Selmer group. Without confirming that this group is small, the proof remains incomplete.

"I am still optimistic that the problems will be worked out," says Karl Rubin of Ohio State University in Columbus. "I can't say how long it will take, but I would expect a complete proof before long."

Rubin is one of only about half a dozen mathematicians who have copies of the preliminary, 200-page manuscript of the proof. Some mathematicians have complained that Wiles' reluctance to circulate additional copies until his work is finished has hindered the checking process and spawned rampant speculation about where things stand.

Wiles plans to present a full account of his work in a series of lectures at Princeton starting in February.

— I. Peterson

Repairing Hubble: Now a waiting game

The surgery went smoothly, but will it restore the celebrity patient's eyesight? An anxious public must now wait several weeks before specialists can determine the outcome, but experts are guardedly optimistic. After all, four high-flying astronauts — including a former surgeon — repaired the myopic Hubble Space Telescope with surprising ease last week during five space walks.

The apparent success of the \$674 million Hubble mission may usher in a rosier future for manned space flight as well as buoy NASA's sagging reputation. In a call to the astronauts, President Clinton hailed their efforts as "one of the most spectacular space missions in our history."

"It's extremely difficult to keep from getting excited now," says Hubble scientist David S. Leckrone of NASA's Goddard Space Flight Center in Greenbelt, Md.

Nonetheless, the repair mission — aimed at restoring Hubble's mechanical and optical health during an overhaul in the cargo bay of the space shuttle Endeavour — had its anxious moments. During the first space walk, astronauts found they couldn't completely shut two doors housing the gyroscopes they had just replaced. The gap between the doors could allow light to leak into the telescope and ruin observations. Using a strap to pull the doors closer together, the astronauts managed to shut them just minutes before the end of their walk.

Another cliff-hanger came during the third space walk, on Dec. 7. Story Musgrave and Jeffrey Hoffman had gently slid the old Wide-Field and Planetary Camera (WFPC) out of its compartment on Hubble, and Musgrave was about to remove the "lens cap" protecting a key, pristine mirror on the new camera, exposing it to the environment of space for the first time. Removing the cap, says Hubble project scientist Edward J. Weiler, "was delicate; he couldn't touch it, and we were watching it at the control room. And sure enough, just as he reached for it, we lost the [video]." But minutes later, the astronauts radioed that they had removed the cap and installed the camera.

Later that night, as the astronauts clamped new magnetometers onto the old ones at the top of the telescope, the cover of one of the old magnetic detectors came loose. Members of the shuttle crew crafted makeshift covers out of extra insulation. Then, with the blue-white marble of Earth clearly visible behind them, Hoffman and Musgrave attached the covers during the final space walk two days later.

The aftermath of the fourth space walk was like a roller-coaster ride. First, astronauts Kathryn Thornton and Tom Akers deftly installed COSTAR, a device that sharpens the blurred light bouncing off

'Good cholesterol' helps more than heart

A new contender to fight sepsis, a life-threatening bacterial infection, just entered the ring. It's high-density lipoprotein (HDL), alias the "good cholesterol," an established heavyweight in the heart disease arena.

Sepsis can prove deadly when the body's immune system overreacts to the endotoxin molecules released by the bacteria that flood the bloodstream. Half of the 300,000 to 400,000 people who develop sepsis each year die of it. The search for drugs to fight the condition has yielded little success, and doctors must rely on antibiotics (SN: 8/15/92, p.104).

HDL is noted for binding to and helping the body dispose of artery-clogging cholesterol. Also, studies have shown that HDL and other lipoproteins will bind to and neutralize endotoxins, thereby preventing the immune system's overreaction to them. Now, an *in vivo* study of mice given an endotoxin finds that animals with higher concentrations of HDL in their blood are less apt to die.

In the study, Daniel M. Levine of the Rogosin Institute at New York Hospital-Cornell Medical Center and his colleagues injected an endotoxin from one of two common bacteria into 232 mice. They genetically engineered half of the mice so the animals would have different concen-

trations of HDL in their blood. Their results appear in the Dec. 15 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES.

Almost 40 percent of the mice whose HDL ranked in the top quarter survived at least 48 hours after the endotoxin injection, compared to 8 percent of the animals in the lowest quartile, they write. In other mice, the researchers injected an endotoxin and a synthetic HDL. These mice had two to four times the survival rates of untreated mice given the endotoxin, more HDL-endotoxin binding, and milder immune responses.

Very preliminary studies suggest that people with high HDL do better at fighting infection, two team members say.

The researchers have patented the use of the synthetic HDL for treating sepsis and hope to test the drug on humans by the end of 1994. A pharmaceutical company has licensed the drug and will fund further research, including the human trials.

Joseph H. Rapp of the University of California, San Francisco, Medical Center praised the work. He and his colleagues are looking at chylomicron, a very-low-density lipoprotein that clears the body more rapidly than HDL. They reported earlier this year that chylomicron protects rats against sepsis.

— T. Adler