Cold fusion saga: Trials and tribulations

In two tumultuous weeks of June, the cold fusion research community encountered a scientific snag, a political embarrassment and a legal surprise with haunting implications.

On the scientific front, nuclear chemist Kevin L. Wolf revealed that he had found tritium – a potential fusion product – in several unused segments of palladium wire obtained from Hoover & Strong, Inc., a metals processor in Richmond, Va. Wolf, of Texas A&M University in College Station, had previously reported intermittent detection of tritium in his cold fusion experiments. He now says those observations may have reflected contamination of the palladium electrodes used in the experiments, rather than some unexpected physical process such as cold fusion.

"The contamination is at a level that is consistent with the bulk of the tritium findings at various laboratories," says David Worledge, research manager of the Electric Power Research Institute in Palo Alto, Calif., an industry foundation that funds several cold fusion researchers, including Wolf.

Some scientists insist palladium contamination does not explain all the tritium observations made to date. For example, Edmund K. Storms of the Los Alamos (N.M.) National Laboratory told SCIENCE NEWS that only one of his 11 tritium-positive experiments involved palladium from Hoover & Strong. "We don't think our wire has been contaminated," he says.

Just days before word of the contamination reached the public through the June 7 Wall Street Journal, a political controversy erupted at the University of Utah, where B. Stanley Pons and Martin Fleischmann first ignited the cold fusion drama 15 months ago.

The Salt Lake Tribune reported on June 1 that $500,000 identified as a grant from an "unnamed source" in the current quarterly report of the university-affiliated National Cold Fusion Institute (NCFI) actually came from a school fund. A state advisory board, which oversees Utah taxpayer dollars earmarked for cold fusion research, relies on these reports to make recommendations to the governor about releasing funds to NCFI.

NCFI Director Fritz G. Will told SCIENCE NEWS he blames the error on high-ranking university officials who led him to believe the money came from a legitimate anonymous source. The inaccurate portrayal of funding sources, creating the appearance that NCFI had attracted substantial external support, outraged many of the university's scientists. A committee of 22 faculty scientists met with University of Utah President Chase N. Peterson on June 1 and demanded financial and scientific audits of NCFI.

The state's advisory board convened June 7 and organized a committee to plan the audits, which also will help the state government make future decisions regarding cold fusion funding, says Randy Moon, a board member and science adviser to Utah's governor.

But legal antics by the lawyer representing Pons and Fleischmann may become this month's most memorable cold fusion *faux pas*.

University of Utah physicist Michael J. Salamon and nine co-workers reported in the March 29 NATURE that the radiation detectors they had set up around Pons and Fleischmann's cold fusion cells had failed to collect any nuclear evidence for fusion reactions. Several days later, Salamon and all but one of his coauthors received letters from C. Gary Triggs, a Morganton, N.C., attorney retained by Pons and Fleischmann. The letter critiques the NATURE paper as factually inaccurate, biased and wrought with so many problems that it "should be voluntarily retracted." Furthermore, Triggs threatens to "take whatever action is deemed appropriate" to protect Pons and Fleischmann's interests.

Salamon views the letter as a move to limit academic freedom. "Triggs' letter was an attempt to intimidate us," he says. In May, the Salt Lake Tribune reported Triggs had received more than $50,000 from the University of Utah for other cold-fusion-related legal services, under an agreement between Pons and the university. In response to the outrage of cold fusion advocates and skeptics alike, Triggs sent Salamon's group a second letter, dated June 5, saying he never intended to limit their academic freedom.

Salamon, Will and others say the letter fails to disarm an unusual attempt by scientists to use legal threats to mute the conflicting conclusions of their peers. Joseph Taylor, the university's outgoing vice president of academic affairs, drafted a policy letter last week assuring Salamon's group that the university will indemnify them if Triggs pursues legal action. Taylor also says the university will try to sever its ties with Triggs. *–I. Amato*

Mystery mechanism keeps nerve cells alive

For decades, researchers have struggled to understand the chemical changes that trigger the death of neurons (SN: 12/5/87, p.360). Some are testing experimental treatments to prolong the life of injured nerve cells or protect them from damage caused by disease. In stroke patients, for instance, clinicians are assessing drugs known as calcium channel blockers for their ability to protect brain cells deprived of blood flow. Scientists base such studies on the knowledge that these drugs can manipulate the extracellular environment, expanding constricted vessels in the brain or preventing a toxic concentration of calcium ions from entering neurons.

But new animal studies have prompted two investigators to propose that some calcium channel blockers may protect nerve cells through an as-yet-unknown, *intracellular* mechanism. If this proves correct, the researchers say, it may suggest new therapies for treating neuronal injury while providing new insights into neuron death.

Keith M. Rich and James P. Hollowell of Washington University School of Medicine in St. Louis tested a calcium channel blocker called flunarizine, which is used in humans to treat migraines and epilepsy and is under study as a treatment for stroke. In newborn rats treated with flunarizine, they found that 71 percent fewer severed peripheral nerve cells died after one week compared with severed cells in untreated rats. And in cell cultures, flunarizine prevented the death of peripheral nerve cells from rat embryos after withdrawal of nerve growth factor, a chemical vital for their survival. But flunarizine protected the cells only at doses far greater than the amount needed to block the entry of calcium ions, the neurosurgeons report in the June 15 SCIENCE. Moreover, they say, their *in vitro* work excludes the possibility that the drug prolongs cell survival through blood vessel dilation alone.

Rich and Hollowell propose instead that flunarizine acts inside the neuron to prolong life, possibly by preventing toxic metabolism of calcium ions.

Some supporting evidence for their theory, Hollowell says, comes from a report in the March 23 SCIENCE by John H. Weiss, Dennis W. Choi and their colleagues at the Stanford University School of Medicine. Those researchers found that the calcium channel blocker nifedipine slowed damage to cultured mouse brain cells exposed to toxins – but only at drug doses higher than believed required to block calcium ions.

Choi emphasizes that the nifedipine dose needed to block calcium ions in the brain cells might be higher than the value he extrapolated from studies of peripheral nerves. But he says an unidentified intracellular action of the drug, perhaps in combination with its calcium-blocking ability, seems a likely explanation for the protective effect.

Even if researchers solve the mystery of the protective mechanism, human applications will require further study, says David H. Martin at the Washington University School of Medicine. But he adds: "The dream of clinical application – that's what motivates all our work." *–R. Cowen*