BES/Advanced

Department of Energy
Washington, DC 20585

FEB 23 1990

Dr. Chase N. Peterson, President
The University of Utah
203 Park Building
Salt Lake City, UT 84112

Dear President Peterson:

Admiral Watkins has asked me to respond to your February 2, 1990 letter, and to assure you that your points are understood.

The most important concern we all have is to ensure that we do not lose sight of the fact that there continue to be unexplained experimental results that we need to understand.

From the very beginning of the "cold fusion" excitement, Admiral Watkins called for a thorough examination of the results and, as you well know, resources of the Department and our national laboratories were rapidly deployed to examine every facet of the results being reported from different quarters.

The review by our Energy Research Advisory Board, which was reported in the article enclosed with your letter, was requested by Admiral Watkins. His purpose was to ensure as thorough a peer review as we could muster, and, as you know, we are continuing support at several universities and laboratories of research in this area.

Thank you for taking the time to write and for the material forwarded.

Sincerely,

James F. Decker
Acting Director
Office of Energy Research

Dr. Chase N. Peterson, President
The University of Utah
203 Park Building
Salt Lake City, UT 84112

Dear President Peterson:

Admiral Watkins has asked me to respond to your February 2, 1990, letter.

In making his comment regarding the absence of "proper peer review" of the original Fleischmann/Pons paper, the Secretary reflected a belief widely held within the scientific community. If nothing else, just an examination of the paper's submission date, March 13, 1989, (in revised form March 22, 1989) must raise questions about the depth of the peer review that paper has received prior to the March 23, 1989, press conference. Accordingly, I believe the Secretary's point regarding lack of a proper (emphasis added) peer review of the paper prior to the public disclosure was well taken.

I also fully agree with Admiral Watkins' other statement, that the confusion of recent months could have been avoided had the University of Utah announced its findings in a scientific journal rather than at a press conference. It is true that announcements of scientific results through press conferences are not unheard of, and the two examples you cite illustrate that well-known fact. Please note, however, that the Secretary did not take a position as to whether this means of disseminating scientific information is or is not appropriate; that issue, under our system of government, should best be left to the scientific community at large to decide. All the Secretary said was that "confusion could have been avoided." I could not agree with him more.

Thank you for taking the time to write Admiral Watkins.

Sincerely,

James F. Decker
Acting Director
Office of Energy Research

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FROM PETERSON, CHASE N UT 0
PRESIDENT
THE UNIVERSITY OF UTAH

SUBJ: NUCLEAR
FUSION
FWDS INFORMATION PERTAINING TO
NUCLEAR FUSION, THE CYSTIC
FIBROSIS GENE AND THE MASS OF
THE Z PARTICLE

REMARKS:
Prepare reply for signature of Dr. Decker.

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2				
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THE UNIVERSITY OF UTAH

Chase N. Peterson
President

2 February 1990

The Honorable James D. Watkins
U. S. Energy Secretary
Department of Energy
7A-257 Forrestal Building
1000 Independence Avenue, S.W.
Washington, D.C. 20585

Dear Secretary Watkins:

I read with interest your recent interview comments that "fusion confusion" might have been avoided had there been proper peer review of the Pons/Fleischmann paper before the announcement. As a matter of fact, it was stated at the press conference that the paper had been submitted to the Journal of Electroanalytical Chemistry and had been reviewed and accepted. Furthermore, we obtained permission of the editor to release the information before the actual publication date.

We have repeatedly stated that the principal reason for the press conference was that the results were beginning to leak out and that because of the potential practical applications the story would excite considerable interest, which is of course what happened. We felt it was important to release the full story to the general public accurately and at one time.

Interestingly enough, a similar sequence of events occurred subsequently last September in connection with the announcement of the discovery of the cystic fibrosis gene defect. As the enclosed article from Science relates, scientific papers were submitted and accepted, but the story started to become known and the investigators quickly held two press conferences before publication. Here again, the importance to the general public was the key issue.

You may also be interested in the enclosed other story from Science concerning the announcement of the mass of the Z particle by the Fermi National Accelerator Laboratory. This was done by press conference in the absence of any scientific publication. Obviously, since the mass of the Z particle is of little interest outside of the scientific community, the event went nearly unnoticed.

It was, and is my position that some scientific discoveries may prove to have such significance that there is a greater obligation to the general public than to the scientific community.

I have enclosed a copy of your interview as it appeared in the Deseret News last Friday.

Sincerely,

Chase Peterson

Chase N. Peterson
President

Attachment

Office of the President

203 Park Building
Salt Lake City, Utah 84112
(801) 581-5701

The Cystic Fibrosis Gene Is Found

Researchers have identified the major gene defect that causes cystic fibrosis. The discovery should lead to better diagnosis and perhaps improved therapies for the now fatal disease

THE RACE TO FIND the cystic fibrosis gene is over. In three papers to be published in the 8 September issue of *Science*, researchers from Toronto and Ann Arbor report that they have cloned the gene and pinpointed the gene defect that causes most cystic fibrosis cases. "The data are virtually irrefutable that they have the right gene," says Louis Kunkel of Children's Hospital Medical Center in Boston, a cloning expert who led the successful search for the gene causing Duchenne muscular dystrophy.

Gene sleuths. *Lap-Chee Tsui (left), Francis Collins, and their colleagues tracked down the cystic fibrosis gene.*

Cystic fibrosis researchers have looked long and hard for their gene—and with good reason. The disease is the most common genetic disorder of Caucasians. In the United States, it strikes one child in every 2000. An estimated 30,000 people have the disease today, and their prospects are grim. Most will die before their thirtieth birthday. Perhaps not surprisingly then, news of the gene discovery began to leak out before the scheduled publication of the papers describing the research, and this in turn prompted the editors of *Science* to drop their normal embargo policy (also see box on p. 924).

The discovery means that scientists can improve cystic fibrosis diagnosis, including prenatal diagnosis, and also devise better screening tests for people who carry a defective copy of the gene and run the risk of having children with disease. It also raises hopes for better cystic fibrosis treatments, perhaps new drugs or even gene therapy to replace the defective gene itself.

None of this could have even been considered until scientists could get a handle on the basic protein defect that causes cystic fibrosis. "Now we can really study what the basic defect is and we may be able to treat the defect directly, not just the symptoms," says Lap-Chee Tsui, the leader of one of the groups that cloned the gene. No one can now predict, however, how long it might take to do this or even if it will prove to be possible.

The search for the cystic fibrosis gene has been highly competitive, if not out-and-out contentious at times (*Science*, 8 April 1988, p. 141, and 15 April 1988, p. 282). But in the end, a collaborative effort by the groups of Tsui and John Riordan at Toronto's Hospital for Sick Children, together with Francis Collins at the Howard Hughes Medical Institute at the University of Michigan, bagged the gene.

The researchers appear to have a clear victory. "We have a lot of papers in press, but we don't have the gene," says chief competitor Robert Williamson of Saint Mary's Hospital Medical School in London, who has also been rumored to be close to cloning the cystic fibrosis gene. "If we couldn't get it, we're very pleased that Francis and Lap-Chee were the ones to do it."

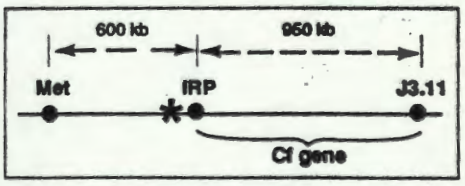
The collaboration between Tsui and Collins began in the fall of 1987, when the two researchers, who had previously been working independently, got together in San Diego at the annual meeting of the American Society for Human Genetics. "It was clear by then that this was a very hard problem that was not going to be solved without a great deal of labor," Collins says.

The cystic fibrosis gene was such a tough nut to crack because, in the absence of information about the protein it encodes, researchers did not know what they were looking for among the estimated 100,000 genes in the human genome. Researchers have managed to clone a few other genes without knowing what their products were—the Duchenne muscular dystrophy gene is one of them—but some of the defects responsible for the malfunction of these genes were large rearrangements that made them relatively easy to spot once their approximate locations in the genome were known. The cystic fibrosis gene di not carry any such convenient tag unfortunately.

In 1985, however, 2 years befor Tsui and his colleagues joined force with Collins and his team, the Toron to group had provided a big boost to efforts to find the gene when they mapped it to chromosome 7. Williamson and Ray White of the Howard Hughes Medical Institute at the University of Utah in Salt Lake City further narrowed its location by identifying two "markers," the *met* oncogene and a DNA sequence designated J3.11 that flanked the gene (see diagram).

Once flanking markers have been identified, they can serve as starting points for zeroing in on a target gene. But *met* and J3.11 are almost 1600 kilobases apart—too great a distance to be traversed in a reasonable time by standard chromosome "walking" techniques. (A researcher walks a chromosome by identifying overlapping cloned fragments of DNA until the final destination is reached.)

Tsui consequently decided to use a brute force approach known as saturation mapping to find new marker sequences that were closer to the cystic fibrosis gene than either *met* or J3.11. To do this, the Toronto workers had to look for identifiable DNA sequences that are inherited along with the cystic fibrosis gene in the members of families with cystic fibrosis. The frequency with which a particular marker is transmitted along with the target gene gives an estimate of how close together they are. "Lo and behold, after screening 250 markers," Tsui says, "we found two that happened to be between *met* and J3.11." The genetic studies



At the start of the search. *The cystic fibrosis gene was known to be somewhere between the IRP gene and J3.11, but closer to IRP. The asterisk marks the location at which the successful hunt for the gene began.*

indicated that both were closer to the cystic fibrosis gene than either *met* or J3.11.

It was at that point that Tsui joined forces with Collins, who had developed a technique called chromosome jumping that can skip over lengthy segments of DNA. Not only is jumping faster than walking, but it also has the advantage of being able to move over unclonable DNA sequences. The human genome is studded with such sequences and they can stop a walk in its tracks.

It would have been ideal if the two new markers identified by Tsui had flanked the cystic fibrosis gene. But they didn't. They were located close together between *met* and another gene that the Williamson group had originally identified in the spring of 1987.

At the time, the London workers thought that they had the cystic fibrosis gene itself and said as much in an article published in *Nature*. By the fall of that year, howev their hopes were cruelly dashed when ad tional work showed that it was not. But t gene, which became known as the IRP (i *int*-related protein) gene because the prote it encodes resembles the product of the oncogene, did prove to be the closest mark yet for the cystic fibrosis gene.

Despite some initial disappointment the location of the new markers, Tsui, Cc lins, and their colleagues decided to plun ahead. They began walking and jumping one marker, moving at first in both dire tions. "You have to move in both directio until you cross a landmark that tells yc which way you are going," Collins explain The IRP gene was one of the first landmar crossed, and the researchers knew that the were heading in the right direction.

The researchers had to jump and wa across 280 kilobases of DNA before er countering the beginning of the cystic fibr sis gene. Along the way they searched fc potential genes by comparing the DN sequences they were traversing with DNA from other organisms. If structurally relate sequences could be found in other organ isms, that would mean that the sequence ha been conserved during evolution, a goo indication that it has an essential function They found three conserved DNA se quences but were quickly able to eliminat two of them as candidates for the cysti fibrosis gene.

The third proved to be the key to the prize—but not without some initial anxiety. Genetic studies in cystic fibrosis families indicated that the potential gene segment was in the right location, but when the researchers looked for signs that it might be actively expressed, they could not find any after an extensive search. "That was quite disappointing," Tsui says. It looked as if the DNA sequence was not part of any active gene.

And this is the point where Tsui's Toronto colleague Riordan made an essential contribution. The Riordan group had made "libraries" of DNAs copied from the messenger RNAs present in sweat gland cells, which is one of the cell types in which the cystic fibrosis gene is supposed to be expressed. Each DNA copy corresponds to an active gene, and one of those from the sweat gland library proved to contain a segment matching the conserved sequence that Tsui, Collins, and their colleagues had found.

The extent of the match-up was quite small. The two DNAs shared only 113 base pairs of sequence, a circumstance that may explain why the researchers originally had so much trouble showing that the conserved sequence was part of an active gene. Further analysis showed, however, that those 113

The CF Gene Hits the News

When scientists attack problems cooperatively, they often hasten solutions. But there's at least one downside to this. It may fatally wound the hallowed tradition of the publishing embargo. Some secrets are apparently just too good to keep.

That certainly was the case with the discovery of the cystic fibrosis gene. With some two dozen researchers at two institutions and perhaps a half-dozen funding agencies involved in two countries, it may come as no surprise that someone leaked the news to the press. Reports began appearing more than 2 weeks before the papers describing the achievement were scheduled to be published, prompting *Science* editor Daniel Koshland to take the highly unusual step of lifting the embargo that is normally imposed on data in press at the journal. And that's not all the leaks did.

The papers are to appear in the 8 September issue, a near record 5 weeks after they were first received. In the normal course of events, a press conference would have been held on 7 September. The patent agents for the two institutions where the work was done wanted to file the patent applications before then.

But those plans changed on 22 August when Reuters News Service put out a story on its wire that said—correctly—that Francis Collins of the University of Michigan and Lap-Chee Tsui of the Hospital for Sick Children in Toronto had discovered the cystic fibrosis gene. "Once Reuters had broken the story we thought it was unfair to the rest of the press to withhold the information," Koshland says.

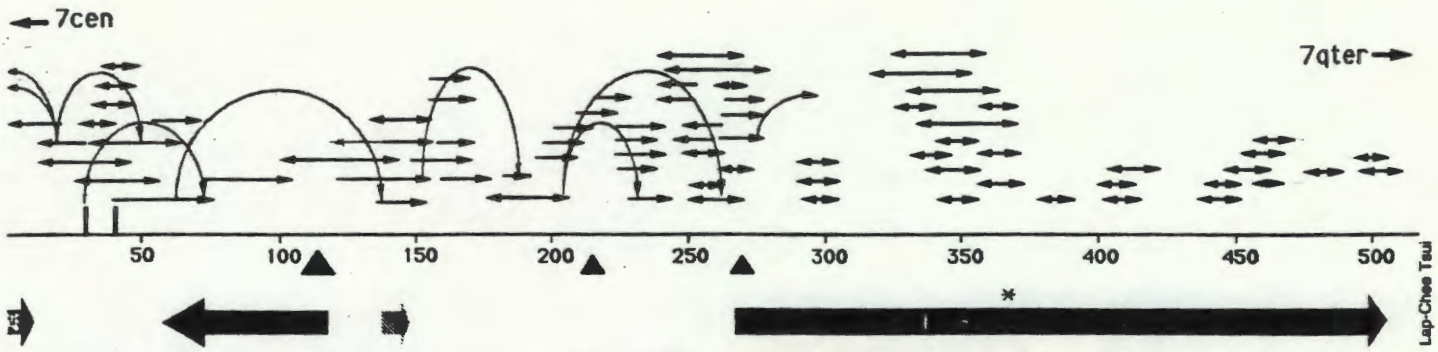
The researchers then held two press conferences, one in Toronto and one in Washington, on one exhausting day: 24 August. Howard Hughes Medical Institute, which supports Collins' work, provided private jets that made this possible. *Science*'s own press office was under siege by reporters who were not pleased to learn that the actual papers, still grinding their way through the editorial mill, would not be available for another week. At least one veteran reporter grumped that it was just like cold fusion all over again. It wasn't, of course; the three papers had been accepted.

Meanwhile, on learning that the press conference was going to be moved up, patent officials at the University of Michigan became concerned because the patents on the gene discovery and its applications had not yet been filed, and they were afraid that public disclosure of the research might jeopardize the awarding of patents, especially in Europe and Japan.

According to Robert Gavin of Michigan's Office of Intellectual Properties, the patent rights will be jointly held by the Hospital for Sick Children and the University of Michigan, with Howard Hughes receiving a share of Michigan's royalties. The discovery could be worth a lot of money because it should make it possible for the first time to screen members of the general population for defective cystic fibrosis genes. Since an estimated 1 person in 20 is a carrier, the potential testing market is large.

So with the press conference date moved up, the patent agent for the Hospital for Sick Children had to move quickly, filing the patent at the U.S. patent office at 11:45 p.m. on 22 August. Gavin was still concerned that the descriptions of the applications were not sufficiently detailed, however. James Friesen, the director of the Research Institute at the Hospital for Sick Children, concedes, "We knew that what we put in was not very polished. We thought we had another week and a half." They didn't, but patent applications can be amended.

Can the decorum of science publishing be enforced in the future? Koshland certainly prefers this. "Basically we try to keep embargoes because we firmly believe in the principle that people should be able to see all the data at the same time," he avers. But in the new age of high-stakes science and large collaborative groups, that may be more easily said than done. ■ J.L.M.



The long march to the cystic fibrosis gene. *The trek began at a site, shown here at the left of the diagram, that Lap-Chee Tsui's group had identified as close to the cystic fibrosis gene on human chromosome 7. The 280 kilobases of DNA between the start site and the beginning of the gene were covered by a combination of chromosome "walking" and "jumping." The straight arrows above the line represent the DNA segments cloned during the walk and the curved arrows represent the jumps taken. The long arrow on the lower right depicts the cystic fibrosis gene, which spans about 250 kilobases. The dark bars are the 24 exons that specify the amino acid sequence of the protein encoded by the gene. These are separated by the noncoding sequences known as introns. The exon marked with the asterisk contains the mutation found in 70% of defective cystic fibrosis genes. The middle arrow on the lower left denotes the IRP gene that was identified by Robert Williamson's group during their search for the cystic fibrosis gene. The other two right arrows also mark sequences with protein-coding capabilities and the three triangles point to sequences of a type frequently found near gene start sites.*

base pairs were likely to come from the starting end of the cystic fibrosis gene. This provided the probe they needed to clone the whole gene. But even that did not come easily. "To finish the cloning we spent night and day with lots of people," Tsui remarks. "We could only get bits and pieces and then had to fit everything together."

The gene proved to be quite large, extending across nearly 250 kilobases of genomic DNA. Like other genes of higher organisms, it consists of a mosaic of protein-coding exons—24 in this case—separated by nonprotein-coding introns.

Sequence analysis revealed that the protein encoded by the gene contains 1480 amino acids and that it has all the earmarks of a membrane protein, possibly of an ion channel. The protein sequence resembles those of several other proteins known to be involved in transporting substances across membranes. And that, says Robert Beall of the Cystic Fibrosis Foundation, "is very compatible with our current hypothesis of what causes cystic fibrosis."

The patients' main problem is the abnormally thick mucus that they produce, especially in the lungs. As a result, they fall prey to repeated infections that destroy the lung tissue, eventually leading to the patients' deaths. Although researchers had not been able to identify the protein defect causing the excessively thick mucus, recent evidence has indicated that the fault may lie in the inability of the lung cells to secrete chloride ions, and therefore water, into the mucus.

The structure of the cystic fibrosis protein now suggests that it may be a membrane channel for chloride ions. Moreover, Tsui, Riordan, Collins, and their colleagues have found that the gene encoding the protein is altered in cystic fibrosis patients, a change that might well cause the protein to malfunction.

Approximately 70% of the gene mutations are caused by the loss of a single specific trinucleotide codon. As a result, the corresponding protein is lacking just one amino acid, the phenylalanine at position 508. The researchers never see this change in the gene on normal chromosomes. This observation provides the proof that they have the correct gene, Tsui says. They are now looking for the remaining 30% of the mutations that alter this gene.

The site of the phenylalanine deletion may provide some clues as to how the protein malfunctions in cystic fibrosis patients. It affects what may be an important region regulating the protein's activity. The region contains an apparent binding site for adenosine triphosphate (ATP), a compound that provides energy for many cell functions. A nearby region also contains several target sequences for phosphate addition by the protein kinases A and C, both important protein regulators.

The loss of the phenylalanine may therefore interfere with chloride ion transport by preventing ATP binding to the cystic fibrosis protein and depriving it of the energy it needs or by rendering it unresponsive to activation by the protein kinases.

Currently, clinicians can only treat cystic fibrosis patients by attempting to control their infections and other symptoms. But now that the cystic fibrosis gene and protein are in hand it may at last be possible to design more rational therapies aimed at the specific defect itself. One possibility is to develop drugs that can act through the protein to restore normal chloride transport. "This will require a long period of research and development," Tsui says, "but we have at least reached a starting point."

Ultimately, it may even be possible to use gene therapy to correct the defect. Introducing the normal gene into lung cells should be sufficient to help patients, Beall suggests. They have other symptoms, but these can be controlled. It is the lung defect that kills. Until a way can be found to deliver a functioning cystic fibrosis gene into lung cells, gene therapy will remain something of a long shot, however.

Improved detection of carriers of defective cystic fibrosis genes is a much more immediate prospect. A person has to inherit two bad genes to get the disease. A carrier has only one defective copy and does not have any symptoms by which he or she might be identified. But if two carriers have a baby, their child has a 25% chance of being affected.

Before the cystic fibrosis gene was discovered, carriers could only be detected in families already known to carry the defective gene because some of their members had the disease. Genetic counselors were forced to look for markers known to be inherited with the cystic fibrosis gene, rather than the gene itself, and this procedure requires a knowledge of family genetics.

But the new work should make it possible to identify defective cystic fibrosis genes in anyone. This will require, Tsui points out, that the remaining 30% of the mutations in the cystic fibrosis gene be identified. But the researchers are hard at work on this project, and it may be completed in a year or two.

The long march to the cystic fibrosis gene was obviously arduous. But the successful procedures worked out by Tsui, Collins, and their colleagues for isolating the gene should also be applicable to the identification of the genes causing other genetic diseases. "It was a long task," Kunkel says, "but it shows that it can be done. It can be done again."

■ JEAN L. MARX

Zs For Two: A Critical Mass

As physicists continue their global race to reveal the secrets of the Z particle, one of the pillars of unified field theory, tempers are getting frayed. On 19 July researchers at Chicago's Fermi National Accelerator Laboratory proclaimed via press release that they had surged into the lead by achieving "the most precise measurement" of the Z mass yet: 90.9 ± 0.35 billion electron volts (GeV).

Foul, cried Fermilab's West Coast rivals at the Stanford Linear Accelerator Center (SLAC). "They were deliberately trying to scoop what they knew [would be] a superior result," said SLAC spokesman Michael Riordan in the heat of the moment. According to Riordan, the Fermilab team was well aware that at a SLAC summer workshop on 21 July, the experimenters working at the Stanford Linear Collider were scheduled to present an even more precise result: 91.11 ± 0.23 GeV.

And using a press release to announce results rubbed people the wrong way, said SLAC director Burton Richter, who fired off a letter of protest to Fermilab's new director John Peoples. "I would prefer to do things the normal way, through conferences and publications," Richter told *Science*.

But Peoples isn't apologizing. "So much money has been spent on these experiments by the American taxpayer that when an important result comes out, it's almost an obligation to let people know," he says.

The University of Chicago's Melvin Shochet, a spokesman for the Fermilab experimental group, took exception to SLAC's accusation of "publication by press release."

"We sent our article to *Physical Review Letters* first," he says. On the day it arrived at that journal, moreover, the group gave a detailed seminar on the experiment—at that same SLAC summer workshop—and distributed copies of the paper to everyone who wanted it. "The whole thing got blown up out of proportion," he says.

Indeed it was. The feelings on both sides simmered down almost as fast as they flared up. And yet in retrospect, say the physicists contacted by *Science*, the incident illuminates the deep undercurrent of anxiety at both laboratories. Budgets are tight and, as Richter points out, if the $6-billion Superconducting Super Collider really goes forward, "everybody is nervous about what it is going to do to the rest of the [high-energy physics] program."

SLAC, most notably, is at least a year late with its linear collider, a highly experimental machine using electrons and positrons to create, what Richter promoted as a "Z Factory." It is working now: some 150 Zs have been produced since April. But the laboratory badly needs to restore some of its credibility by skimming the cream of the Z physics—before the Europeans can weigh in with the enormous machine known as LEP, which is still on schedule to start making Zs in quantity this August. So it hurt to have Fermilab beat them, even temporarily.

Fermilab, meanwhile, is fighting a widespread perception in the physics community that proton-antiproton machines such as Fermilab's Tevatron are about as useful as a sledgehammer when it comes to doing high-precision experiments. "We were up on cloud nine" with the Z mass result, says Shochet, "not just because we were first, but because we got such accuracy"—better than the experimenters themselves expected.

■ M. MITCHELL WALDROP

Gallo Associate Subject to Investigation

A member of Robert C. Gallo's AIDS research team at the National Cancer Institute is being investigated for possibly violating federal conflict-of-interest laws in connection with the award of contracts to a company where his wife worked.

Syed Zaki Salahuddin, a technician who has worked with Gallo for about 10 years, allegedly recommended that Gallo's lab at the National Cancer Institute do business with Pan Data Systems, Inc., without disclosing that his wife, Firoza Salahuddin, was a founder and director of the Rockville, Maryland, biomedical research firm.

Both Salahuddin and federal health officials have declined to comment on the case, which was first reported in the *Washington Business Journal*. Salahuddin's lawyer, Seymour Glanzer, said he is "unaware of the existence of any government investigation to any purported wrongdoing by Mr. Salahuddin."

Contacted by *Science*, Gallo also said neither he nor Salahuddin had been contacted by any federal investigator. And Gallo said that Salahuddin has never had any financial connection with Pan Data. But Gallo recalls learning several years ago that Salahuddin's wife worked for Pan Data and says he told his technician: "I don't know if it's right or wrong, but I know it's stupid." Mrs. Salahuddin promptly resigned from the company, Gallo asserts, adding that he was astonished and outraged that the case has aroused any outside interest. "He's not a big fellow. This is not a big deal," says Gallo.

Gallo characterized Salahuddin as a hard-working, "totally dedicated" individual who "may be the best in the world at culturing blood cells." Salahuddin is a co-discoverer with Gallo of the HBLV—a B-cell virus—and has been involved in key work on the AIDS virus, HIV.

The General Accounting Office reportedly began an investigation last March which subsequently was taken over by the Inspector General of the Department of Health and Human Services. Earlier this month the GAO informed the office of Representative John D. Dingell (D–MI), chairman of the House investigations subcommittee, that a grand jury investigation had begun. ■ C.H.

Headed for NOAA's Choppy Waters

John A. Knauss

President Bush has nominated academic administrator and oceanographer John A. Knauss to head the chronically underfunded National Oceanic and Atmospheric Administration. For 25 years, Knauss, 63, was Dean of the University of Rhode Island's Graduate School of Oceanography, where he oversaw its growth from a small coastal laboratory to a major research and teaching institution. In 1987, he stepped down to resume his research in physical oceanography.

But now he has been lured away from his lab, all the way to Washington, where he has served on numerous governmental panels including the one that led to the formation of NOAA in 1970. Why leave the pleasures of academia beside Narragansett Bay? "NOAA is a big, exciting organization," says Knauss. "In an era in which more and more people are worried about global change, it has the primary responsibility for measuring and predicting it." And what of NOAA's chronic funding problems? "I don't know if I can turn that around," he says, "but I'm sure going to try."

Knauss's Senate confirmation hearing may not make it onto this session's calendar, but he is expected to face little opposition when it finally is held. ■ R.A.K.

140TH YEAR — No. 226 SALT LAKE CITY, UTAH FRIDAY, JANUARY 26, 1990

Course of fusion race has yet to be set, energy official says

■ Science: DOE leader defends U.S. restraint in funding research of phenomenon that he says is still unproven, despite Japan's charging ahead.

By JoAnn Jacobsen-Wells
Deseret News science writer

WASHINGTON — The United States is not trailing Japan in the international fusion race because no one, as yet, has convincingly proved that cold fusion exists, according to the nation's top energy official.

U.S. Energy Secretary James D. Watkins said he is not concerned, "because I think we have done our homework. I think had we not done all this homework we would have been more concerned. I think it's premature at this time to say that we are losing a race in cold fusion when we have very clearly validated that we are not sure that it's fusion."

In an exclusive Deseret News interview this week, Watkins responded to the pronouncement by a Japanese government scientist that Japan — which boosts 200 fusion researchers in 30 labs — is usurping the United States in the race to turn a Utah-born phenomenon into a practical energy source.

Several U.S. scientists and political leaders agree. They insist that the DOE's refusal to aggressively fund fusion research will result in still another Japanese technological victory.

Watkins doesn't see it that way.

"We have got some uncertainties, so to launch off on something like that would be scientifically unjustified from our point of view. Yet to the tune of $1 million a year, we are not rejecting some of the interesting phenomena that have yet to be resolved," he said.

PHOTOGRAPHY/ TOM SMART

James Watkins says fears of Japan taking the lead in cold fusion are premature.

Watkins' conclusions mirror those of the DOE's Energy Research Advisory Board, which in April began a thorough assessment of the new science announced by electrochemists B. Stanley Pons, of the University of Utah, and Martin Fleischmann, of England's Southampton University.

Pons and Fleischmann said they had achieved fusion at room temperature, a process that, if

Please see FUSION on A2

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FUSION

Continued from A1

perfected, could provide the world with a limitless supply of clean and relatively cheap energy.

However, the advisory board's report — completed in November after a cold fusion advisory panel visited several laboratories and studied the results of many others — dampened spirits heightened by the prospect of pollution-free fusion power plants.

Panel members, led by John Huizenga, chemistry professor at the University of Rochester, and Nobel Prize winning chemist Norman Ramsey, concluded that the nation's fusion experiments (reported up to July) didn't present convincing evidence that useful sources of energy will result from the phenomena.

The panel recommended against any special funding or the establishment of special research centers for the investigation of the phenomenon but was sympathetic toward modest support for carefully focused experiments within the present DOE system.

Watkins said his position on the report won't be announced until it's evaluated by James Decker, acting energy research director. However, unless some major flaws are identified, the secretary said the DOE will probably follow it.

"My first reaction is that it's a solid report coming from a solid group of people and generally in line with what my laboratory directors and others are telling me out there in the experimental process," he said. "It also is balanced in the sense that it doesn't pooh-pooh the phenomena that are not understood yet."

Watkins continued, "I am not a scientist. But I have been around the scientific world long enough to be cautious before you reject anything because many times we are right for the wrong reasons or wrong for the right reasons. And we have got to find out exactly where these unknowns — once we solve them — will lead us.

"There are some interesting things going on here. We know that and we don't want to lose sight of that. But what we are not willing to do is go beyond the recommendations at this point in time that a very eminent panel gave us."

What does that mean to fusion researchers hoping to keep their experiments afloat with DOE funds? Watkins said the DOE doesn't have any line item that says, "Cold fusion: so many dollars — even $1 million a year."

But money is available to researchers "trying to find out what this [illegible]."

Fusion programs at the University of Arizona, Brigham Young University and the DOE's Los Alamos, Oak Ridge and Brookhaven labs will continue to receive DOE support, he said. Grants from U. fusion researchers "will be looked at like any other grants."

"I don't know the contents, so it would be premature (to comment)," Watkins said of the four proposals the U. is expected to submit to the DOE for funding. "I don't want to raise expectations that we will approve them, but we will look at them objectively like anybody else that comes in here with research grant requests."

Meanwhile, Watkins said, the DOE is monitoring the activities of other funding organizations such as the Electric Power Research Institute. "We are all hooked up together."

But unless "something exciting" leads them in another direction, the DOE for the time being will do no more than hold the line on fusion at any time.

Watkins said he has just approved the names for the DOE's Fusion Advisory Panel, which will "come together and try to focus our future in fusion in perhaps a new and exciting way."

The secretary's hope is that the mysteries that have shrouded fusion researchers for years will be resolved by the end of the decade, and that the United States will move to a demonstration program that would lead to power generation in fusion by the year 2025.

"That's optimistic probably, but that would be the kind of thing that I hope this panel would be able to come out with. And then really move down that path because we are getting close," he said. "There's no question about it, but that doesn't mean we are ready for commercial application yet. Transition from the science laboratory to commercial use has got to be looked at very carefully and through a very deliberative

U. announced fusion 'the wrong way'

Adm. James Watkins believes the "fusion confusion," of the past 10 months might have been avoided had the University of Utah announced its breakthrough in a scientific journal rather than at a press conference.

The energy secretary said that had there been proper peer review of the Pons/Fleischmann discovery before it hit the press, "we might have all been on the same wave length from the outset and not diverted any monies unnecessarily."

U. vice president James Brophy said a scientific journal had accepted the Pons/Fleischmann paper before news of the experiment was announced to the press. It was announced only then, he said, to avoid inaccurate reports from being leaked.