ban it completely) on a national but not on a local basis. "I personally deplore the introduction of personalities instead of issues into this discussion of recombinant DNA. I regard such actions as an indication of bankruptcy of argument and I will not engage in such."

The strength of reaction against the second wave of critics seems in some ways disproportionate to the critics' actions. With some notable exceptions, their language has been moderate, and most of their arguments in favor of stricter guidelines, whether right or wrong, are not inherently extreme or unreasonable; 24 percent of biologists and 32 percent of other scientists think the guidelines are "probably insufficiently cautious," according to a straw poll conducted by the Federation of American Scientists. The warmth of the hostility is perhaps explained, at least in part, by an understandable nervousness that the public might use the critics' arguments to shut down research completely. Mayor Vellucci's threats to do just that in Cambridge were certainly alarming, and the citizens' faith in the value of the research, as expressed in the review board's recent report, could perhaps not have been foreseen. "The scientific community seems to have a tremendous sense of living in a hostile environment, of being a little enclave of rationality in a hostile world," notes Rae Goodell, an MIT historian who has followed the gene-splicing issue from the beginning. "There are rigid rules about what a scientist should and should not do. It's fine to be critical in private but not in public. If you want to express social responsibility, it is fine to do so in Washington but not on the street. Pressure on the critics in this issue would seem absolutely inevitable," Goodell observes.

Pressure is hard to measure and easy to exaggerate. Yet however high tensions may have risen at Cambridge, there is no sign that any facts or arguments on the critics' side of the case have failed to reach the public record. The public must have confidence, said the NIH, in an environmental impact statement prepared last year, that the goals of the gene-splicing research accord with social values, and "A key element in achieving and maintaining this public trust is for the scientific community to ensure an openness and candor in its proceedings." The hostility toward the critics is the one shadow on what has otherwise been a notably open and candid process.—NICHOLAS WADE

Legion Fever: "Failed" Investigation May Be Successful After All

Scientists at the Center for Disease Control (CDC) in Atlanta, having labored for months, seemingly in vain, to find the cause of Legionnaire's disease, think that they finally have cracked the case. On Tuesday afternoon, 18 January, at a hastily called press conference that had a decidedly celebratory air about it, CDC officials announced that they have isolated an as yet unidentified organism as the probable cause of Legion fever and the related "Broad Street pneumonia," whose victims were within a block of the Bellevue-Stratford hotel on Broad Street in downtown Philadelphia at the time the two diseases struck last summer. The organism is about the size of a bacterium, is shaped like a bacterium, and stains like a bacterium. So, CDC is calling it a "bacterium-like organism" for now, while gearing up for a series of tests to characterize it in detail. So far, it has no name, but that is a minor matter to researchers whose investigation is beginning to go somewhere.

Legionnaire's disease is one of those mysterious and terrifying things that comes from out of the blue, strikes its victims, and is self-limiting. After a short time, it simply vanishes. It has happened before. CDC files list close to a dozen such instances that are still unsolved. But not many are as conspicuous or attract as much national attention as Legion fever (*Science*, 3 December). It struck 180 individuals who were associated in one way or another with a Pennsylvania state American Legion convention headquartered at the Bellevue-Stratford last July. Twenty-nine of its victims died, as did the hotel after frightened patrons stayed away in droves. At about the same time, there was another mini-epidemic in Philadelphia—38 cases of Broad Street pneumonia were recorded.

Amidst accusations that the investigation was being bungled in every conceivable way, epidemiologists from CDC and state and local health departments tried everything they could think of to find out what was going on. First, they looked for swine flu virus, then other infectious agents—bacteria, viruses, and fungi. Then, and some charge belatedly, they searched for inanimate toxic agents. All they managed to do was rule things out and, by fall, many of the investigators were ready to concede that the cause of Legionnaire's disease might never be found. But they diligently kept on looking just the same, and it appears that their patience has paid off. As yet, they have no absolute proof that their bacterium-like organism is the culprit, but they have the next best thing to it. And Louis Weinstein of Harvard, an acknowledged leader in infectious diseases and a member of a panel of outside reviewers called in to assess the way CDC is doing its job, says "Now the burden of proof is on those who want to say that this organism is *not* the cause of the disease."

According to Weinstein, the review panel, satisfied that CDC officers had done everything they could to detect a conventional organism or agent as the culprit, suggested that they start looking for something unconventional. As it turns out, that is what they seem to have found.

In late December, Joseph E. McDade of CDC's leprosy and rickettsial disease branch decided to look once again at slides that had been prepared about the time of the epidemic and studied thoroughly. He found something that had not been noticed the first time around—in a slide of lung tissue from a dead Legionnaire he saw tell-tale signs of what he thought might be rickettsiae. It was during the course of following that lead to an infectious agent that investigators came upon the "bacteria" that they now suspect caused the disease. How it got to Philadelphia and from where, nobody knows.

Some organisms that cause disease can be identified as pathogens by their behavior in various laboratory test systems. Others reveal their pathogenicity only in living animals. Trying to tease the culprit out of hiding, McDade and Charles C. Sheparel inoculated some guinea

pigs with a suspension of lung tissue from a deceased victim of Legionnaire's disease and others with a lung tissue suspension from a patient who had Broad Street pneumonia. Within 1 to 2 days both those animals that were inoculated with Legionnaire's tissue and those inoculated with Broad Street tissue became ill with fever, watery eyes, and prostration. Some organism in the suspensions of lung tissue was making the guinea pigs sick.

Pursuing the organism further, the investigators prepared suspensions of spleen tissue from those sick guinea pigs and injected them into the yolk sacs of hens' eggs to see what effect there would be on the embryos. Within 4 to 6 days, a relatively long period as far as these things go, the embryos died. Most known pathogenic bacteria will kill chick embryos within a day or two. The thought that the organism might be an unusual rickettsial agent made sense. But upon examination, it was apparent that the organism was most likely a type of bacteria. In size, what they found is larger than a rickettsia. Furthermore, it does not react with any standard antigens to rickettsia. However, the bacteria-like organism fails to grow well on standard culture medium, making identification difficult and raising interesting questions about its true nature. "Bacteria by definition grow in artificial medium and we aren't sure this does. We aren't sure of its place in the bacterial world," Shepard noted at the CDC press conference which was piped live to the Department of Health, Education, and Welfare in Washington, D.C.

With their "unidentified" organism in hand as a likely cause of both Legion fever and Broad Street pneumonia, the next step for CDC scientists was to see whether the victims of the two diseases had antibodies to it in their blood. To their utter relief, the answer was yes. Of 33 Legionnaire patients tested as of 18 January, 29 had antibodies, indicating that they had been infected by the organism. Serums from two of four patients with Broad Street pneumonia were positive. Testing of other victims of each disease is continuing. Serums from individuals used as controls showed no significant concentrations of antibodies to the putative "bacterium" The circumstantial evidence is strong.

There is another case in CDC's "unsolved" file that seemed in many ways akin to Legionnaire's disease but, even though researchers were aware of the analogy early in their investigation, there was nothing they could do to pursue it. In 1966, during the summer, an epidemic of a pneumonia-like illness occurred at St. Elizabeth's hospital for the mentally ill in Washington, D.C. Ninety-four patients became ill; 16 of them died. As is its custom, CDC kept on hand frozen serums from many of those patients. By noon on the 18th, just a few hours before the press conference, CDC had tested 14 serum samples for evidence of antibodies to the new Legionnaire's microbe. Thirteen of them did, indeed, have antibodies. CDC may be able to solve two mysteries with a single microbe.

Although there are yet innumerable questions to answer and details to resolve, it is apparent in talking with persons associated with the investigation that the events of the past few weeks have done wonders for morale. Now, while the microbiologists go about characterizing the organism, the epidemiologists are ready to go out into the field again to see what associations they can find between the microbe and human disease. David Fraser, head of the epidemiologic effort, believes it will be possible to identify Legion fever with a new precision that will allow him to separate those cases of real Legionnaire's disease from cases of more ordinary pneumonia. Similarly, he expects to find that some of the individuals who had Broad Street pneumonia will be shown to have had Legionnaire's disease. By the time the study is over, there may emerge a new understanding of certain types of pneumonia generally, though scientists are reluctant to make sweeping predictions.

Even with the new leads, there remains one haunting question to which there is no apparent route to a solution. If the bacterium-like organism is, in fact, the cause of the disease, or diseases, how did it spread? Fraser notes that the one area in which giving the organism a name might make a difference is in thinking about its spread. "If we knew it were something associated with an animal, or dirt, or water, for instance, it might help us find its source," he says.

There is still some feeling, as there was at the outset of the investigation, that the disease-causing agent passed through the air-conditioning system at the Bellevue-Stratford, even though the newly established connection between Legion fever and Broad Street pneumonia argues against that hypothesis. Nevertheless, it will be checked out again in Philadelphia and, back in Atlanta, CDC scientists are considering testing the idea by adding a little motor oil to their culture media to see if that helps their new-found microbe grow. It seems that, at the hotel, a thin coating of ordinary motor oil was rubbed across the surface of some of the air-conditioning filters to help catch dust. It is just possible, investigators reason, that the organism likes oil and may have found the filters a happy place to live and multiply. That, at least, might account for the fact that the majority of persons becoming ill had some contact with the hotel.

Whatever happens next, however, there is still a long way to go before all the questions about Legionnaire's disease are answered—BARBARA J. CULLITON

APPOINTMENTS

James N. Hayward, former professor of neurology, University of California, Los Angeles, to first chairman, neurology department, School of Medicine, University of North Carolina, Chapel Hill. . . . **Thomas W. Tillack**, associate professor of pathology and of anatomy, Washington University School of Medicine, to chairman, pathology department, University of Virginia School of Medicine. . . . **James F. Shepard**, professor of plant pathology, Montana State University, to head, plant pathology department, Kansas State University. . . . **Louis R. M. Del Guercio**, former director of surgery, St. Barnabas Hospital, New Jersey, to chairman, surgery department, New York Medical College. . . . **Richard J. Sauer**, associate professor of entomology, Michigan State University, to head, entomology department, Kansas State University. . . . **Robert E. Lyle, Jr.**, professor of chemistry, University of New Hampshire, to chairman of chemistry, North Texas State University. . . . **Lawrence S. Wrightsman, Jr.**, professor of psychology, George Peabody College for Teachers, to chairman of psychology, University of Kansas. . . . **Ralph R. Rumer, Jr.**, professor of engineering and applied sciences, State University of New York, Buffalo, to chairman of civil engineering, University of Delaware. . . . **Harry N. Beaty**, professor of medicine, University of Washington, to chairman of medicine, University of Vermont. . . . **Stephen L. Wangensteen**, director, surgical research laboratories, University of Virginia, to chairman of surgery, University of Arizona. . . . **Richard R. Goldberg**, chairman of mathematics, University of Iowa, to chairman of mathematics, Vanderbilt University. . . . **Scott G. McNall**, professor of sociology, Arizona State University, to chairman of sociology, University of Kansas.