In 1974, Nancy Kaiser made the mistake of coming down with an illness that didn't fit any of the available diagnoses. The Albuquerque housewife was just 38, an avid golfer and swimmer, and under no particular stress. Yet she felt like she was dying. She was weak, profoundly tired and plagued by constant bladder infections. Her muscles ached. Her mood shifted unpredictably. Her memory seemed to be failing. "If this is menopause," she remembers thinking, "this is horrible, worse than I ever imagined." Her doctors could offer no better guess, so after seven awful years she agreed to a hysterectomy. When her health didn't improve, physicians referred her to psychiatrists, who announced she was mourning her lost uterus. One suggested she have an affair.

By 1987, Kaiser had been to 212 different experts. Her eyesight was fading and she was having a dozen minor seizures every day. She often lacked the strength to stand up. Her concentration and memory had so deteriorated that she couldn't get through a TV show. Her IQ was down by 22 points. Still, because she lacked a known disease, no one could quite accept that she was sick. Her weary family started pulling away, and doctors hinted that she might be better off in a mental institution.

Eventually she found an expert who was familiar with the problem she described, and interested in helping. After trying several unsuccessful treatments, Kaiser found relief in an experimental AIDS drug, Ampligen. She now leads a nearly normal life, but her ordeal remains a fresh source of outrage. "We're desperate for care," she says of her fellow sufferers. "The medical profession simply doesn't want us."

You wouldn't know it to read the mainstream medical journals, but Nancy Kaiser's strange affliction is today a major public-health concern. Though no one has precise numbers, epidemiologists guess that 2 million to 5 million Americans have been stricken. The illness has spawned four national patients' organizations and some 400 local support groups. Officials at the Atlanta-based Centers for Disease Control (CDC), the federal agency responsible for tracking infectious disease, say they receive 1,000 to 2,000 calls about the condition every month. Dr. Jay Levy, a San Francisco AIDS researcher, calls it "the disease of the '90s."

The ailment goes by a range of different names. The British and Canadians know it as myalgic
encephalomyelitis, or ME. The Japanese call it low natural killer cell syndrome. In this country, patients' groups call it chronic fatigue immune dysfunction syndrome (CFIDS), but it is generally known as chronic fatigue syndrome (CFS). Some of the common symptoms--fevers and lymph-node swelling, night sweats, persistent diarrhea, joint and muscle pain--have a disturbingly familiar ring. But unlike AIDS, this gray plague doesn't kill people, unless they take their lives in despair. It simply turns them into confused invalids. Many patients suffer mood swings or panic attacks and most develop a low-grade dementia. Sleep disturbances are common, as are vision problems. Some sufferers lose their hair, or their finger-prints, or they develop acne for the first time. And though the illness sometimes lifts after a few hellish months, it can linger for years--or recede only to return.

Still, no one is quite sure what it is. "Something is going on," says Dr. Walter Gunn, the CDC investigator in charge of a four-city survey of the phenomenon. "But whether it's one disease or several, one cause or more, is not clear." The CDC defines chronic fatigue syndrome as a distinctive array of unexplained symptoms that cause 50 percent reduction in activity for at least six months. Diagnosis is largely a matter of ruling out other diseases--AIDS, cancer, multiple sclerosis and so on. Treatment proceeds by trial and error.

But the outlook is rapidly changing. Mounting evidence suggests that CFS is an immune-system disorder in which the body works frantically but inefficiently to control common viral infections. In a flurry of small studies, immunologists have found clear breaches in the body's defense system. Neurologists have discovered physiological abnormalities in patients' brains. And a couple of dogged small-town doctors, aided by a Philadelphia virologist, have recently glimpsed what may be the agent behind the affliction. The story of their quest is in one sense a fable about the medical establishment's indifference to problems that defy comfortable assumptions. But it's also a good mystery whose most tantalizing chapter is just now unfolding.

Chronic fatigue syndrome is probably not a new disease. The same baffling symptom complex has turned up both sporadically and in local clusters for more than a century (Florence Nightingale and Charles Darwin may both have been stricken). But until recently it has been rare. The current round of concern started in Incline Village, Nev., a prosperous little resort town snuggled against the north shore of Lake Tahoe. A pair of young doctors named Paul Cheney and Daniel Peterson had been enjoying a quiet practice when, in the fall of 1984, teachers at two local high schools started coming down with what seemed an extremely tenacious flu. Cheney recalls telling them it was just a virus and would surely pass. But within a few months, nearly 200 of the town's 20,000 residents had developed the same symptoms, and no one seemed to get better. Most of the sufferers were mass-producing antibodies to Epstein-Barr, the herpes virus that causes infectious mononucleosis. But mononucleosis is rare in adults and epidemics of adult mono are unheard of. So Cheney called the CDC to report the strange outbreak.

In September 1985, after much prodding, the agency dispatched two investigators, who concluded in a published report that Epstein-Barr was not necessarily the problem. That virus infects nearly everyone by adulthood, and healthy people sometimes exhibit the high antibody counts that Cheney and Peterson were recording. Moreover, Epstein-Barr was not the only bug these patients seemed to be fighting; blood tests revealed high antibody counts to herpes simplex and the cytomegalovirus as well. Rather than fretting over Epstein-Barr, the men from CDC suggested, doctors with sick patients should focus on "more definable, and possibly treatable, conditions."

David Bell still remembers the day in 1986 when he came across a news article about the Lake Tahoe epidemic. He was sitting in his modest office in Lyndonville, N.Y., a tiny farm town on the southern shore of
Lake Ontario. Dr. Bell and his wife, Karen, had moved there in 1979, fresh out of medical school, and he had spent several placid years giving checkups and vaccinating babies. Now his practice was being consumed by the strangest illness he’d ever seen: a persistent flulike condition with no apparent cause. He gathered that the Nevada resort town had been hit by "Yuppie flu," a fashionable form of hypochondria, and that two local doctors were blaming it on the Epstein-Barr virus. "I knew that whatever we were studying had nothing to do with Yuppies," he says. Indeed, Bell’s sickest patients were children, and a quarter of them had tested negative for Epstein-Barr. So he turned the page.

Bell’s troubles had started in November 1986, when his secretary, Jean Pollard, and her husband rushed their 13-year-old daughter to the hospital for an appendectomy. Their three younger girls stayed with David and Debbie Duncanson, family friends with eight boys of their own. Within two weeks all three Pollard girls and five of the Duncanson boys were sick. "One or two of them had slightly enlarged spleens, and their lymph nodes were swollen," Bell recalls. "It looked a lot like mono." But some classic symptoms were missing. Bell guessed it was flu and would soon pass.

Instead, it got worse "At first it was just sore throats and stomachaches and vomiting," Debbie Duncanson says of her sons’ condition. "Then they started walking around crying, holding their arm out straight because their lymph nodes were so sore." The three Pollard girls hardly walked at all for three months. "The! just lay on the couch," their mother recalls. "Their eyes hurt so bad they couldn’t watch television." They were feeling somewhat better by the following summer, but by Bell had roughly 30 cases of the same illness on his hands. It was striking children and grown-ups alike, and he was no closer to knowing "what it was.

After trying in vain to get the state health department involved, Bell and his wife, an infectious-disease specialist, developed a survey to seek out common threads among his patients. The survey covered everything from pets and heating systems to socioeconomic status. When the Bells administered it to 63 local kids, several intriguing patterns emerged: the stricken children were more likely to have a history of allergies to have an afflicted family member and to have been drinking unpasteurized milk (the Pollards and Duncansons had been drinking from the same goat). Unfortunately, the symptoms didn’t match those of any common allergy or bacterial infection. "We went through seven or eight possibilities," Bell recalls. "The upshot was that nothing fit."

By the spring of 1987, the caseload was still growing. He tried referring his patients to specialists, but the specialists could see nothing wrong with them. He and Jean Pollard also tried calling the CDC, but to no avail. Then Bell came across a most unusual patient. She had a classic case of what he was calling the Lyndonville syndrome, but she wasn’t from Lyndonville. She was from California, and she knew her illness as the chronic Epstein-Barr virus syndrome. Bell realized in an instant that he wasn’t alone--that the Lake Tahoe doctors he’d read about were wrestling with the same disease.

They weren’t the only ones. The illness was by now turning up throughout the United States and several other countries, and it had attracted a small but devoted cadre of medical researchers. Dr. Anthony Komaroff, chief of general medicine at Brigham and Women’s Hospital in Boston, had conducted an independent study of the Lake Tahoe outbreak and found unequivocal evidence of an organic illness. Dr. Seymour Grufferman, a cancer epidemiologist at the University of Pittsburgh School of Medicine, was studying an outbreak among members of a North Carolina orchestra. The sick musicians were developing cancer at 18 times the expected rate, were other members of their households. Cheney and Peterson had been
denounced as quacks within Incline Village--a spooky new contagion was the last thing the tourist-dependent community to hear about--and Cheney had Charlotte, N.C. His new practice was being overtaken by patients from around the country, who sought him out when no would take them seriously.

Bell soon learned that a Portland, Ore., was organizing a national conference on the illness. At that meeting, held in the fall of 1987, he, Cheney and the others were able to start comparing notes. Though most researchers were still calling the disease chronic Epstein-Barr virus syndrome, many had turned their attention to other viruses, such as a newly discovered herpes virus called HHV-6 and--particularly in Canada and Britain--the enteroviruses (polio, coxsackie and echo). Tests showed that many of these bugs were active in the chronically sick patients. The trouble was that none was active in every patient, and none turned up exclusively in people with the illness. Since Epstein-Barr clearly wasn't the whole problem, the CDC proposed in 1988 that the illness be renamed chronic fatigue syndrome. Its existence could not be denied, but its cause was still a mystery.

The emerging hypothesis held that CFS was at root an immune disorder. Dr. Jay Goldstein, a southern California physician with a large caseload, had long theorized that the illness begins when "agent X"--some unknown chemical or contagion--damages the immune system. According to his model, the initial injury isn't catastrophic, but it enables viruses that are normally held in check to start running amok in the body. Having failed to maintain the peace, the immune system's helper T cells would start churning out chemicals called cytokines to instigate harsher assaults on the resident bugs. And the perpetual flood of cytokines would, in it self, cause many unpleasant symptoms.

That cytokines can make you sick is no secret; one of them, interleukin-2, causes a syndrome resembling CFS when used as a cancer treatment. To see if it was implicated in the illness itself, Cheney and Bell decided in 1988 to mount a collaborative study. They sent blood samples from 104 of their patients to a Los Angeles laboratory. The tests showed that the patients' average interleukin-2 level was 40 times that of healthy control subjects.

Other findings have since filled out the picture of an immune system in disarray. Several studies have shown that CFS patients' natural killer cells, which normally attack anything foreign to the body, are extremely sluggish in a test tube. Dr. Nancy Klimas of the Miami V.A. Medical Center has also found that certain classes of cytotoxic T cells--killers programmed to attack specific intruders--are either under- or overactive. And just as Bell and Cheney found that CFS patients produce a flood of interleukin-2, Klimas found that they often make too little of another cytokine (gamma interferon). None of these imbalances is unique to CFS, but researchers are optimistic about identifying a general blood profile that is. Jay Levy, the San Francisco AIDS researcher, has spent several years developing a CFS blood test and hopes to present the result this month at a CFS conference in Charlotte, N.C.

The search for clinical signs hasn't been confined to the immune system; scientists have linked CFS to specific neurological problems as well. People with CFS perform poorly on certain cognitive tests, notes Curt Sandman, a research psychologist at the University of California, Irvine, and their problems are quite different from those associated with depression (box).

Sandman can only guess at how these problems might relate to events inside the brain. Other researchers are using sophisticated imaging devices to find out. Dr. Ismael Mena, head of nuclear medicine at Harbor UCLA Medical Center, uses an instrument known as a SPECT scan to gauge the amount of blood reaching
various parts of the brain. CFS patients typically show abnormally low blood flow to one of the two temporal lobes. Dr. Marshall HANDLEman, a neurologist at the University of Southern California Medical Center, uses an experimental device, the BEAM scan, to gauge electrical activity in different brain regions. His test reveals a similar dearth of activity in the temporal lobes and the hippocampus. This region of the brain happens to play a central role in the formation of memories.

These findings all represent progress, but they only deepen the central mystery: why is all of this happening? What agent, or agents, might so disrupt people's immune systems, wreaking such havoc on their bodies and brains?

The AIDS epidemic raised precisely the same question during the early '80s, and the culprit turned out to be a retrovirus, the notorious HIV. Retroviruses are basically self-contained strands of RNA that can translate themselves into DNA and splice themselves permanently into the chromosomes of certain cells. (They're called retroviruses because RNA is normally made from DNA.) These bugs are common in animals, but only three--HIV, HTLV-1 and HTLV-2--have been found in humans. All three attack immune cells called T lymphocytes and are associated with chronic illness. HTLV-1 has been linked to acute T-cell leukemia and a couple of nervous-system diseases: tropical spastic paraparesis (TSP) and, tentatively, multiple sclerosis. HTLV-2 has been tied to a rare form of leukemia.

By the time Cheney and Bell started their interleukin-2 study, Cheney had long suspected that CFS might involve a retroviral infection. In fact he had been pleading with Elaine DeFreitas, a virologist at Philadelphia's Wistar Institute, to help him find out. DeFreitas wasn't much interested in analyzing blood samples from Cheney's patients, she was busy investigating the role of HTLV-1 in nervous-system diseases. But she perked up when Cheney told her about an Air Force sergeant who had fallen ill after an encounter with a prostitute.

Cheney was treating the sergeant for chronic fatigue syndrome, but some of his symptoms reminded DeFreitas of TSP. Since he would have been only the second American-born Caucasian ever diagnosed with that illness, DeFreitas decided to take a closer look. In March 1988 she agreed to have Cheney send her six blood samples identified only by code numbers. She assumed that one would come from the ailing sergeant, the rest from healthy controls. Instead, Cheney sent her six samples from CFS patients. And when DeFreitas ran her tests, something extraordinary happened: all six showed signs of HTLV infection--an infection that is nearly impossible to find in the general population. The reluctant virologist was suddenly eager to know more.

To clarify the tantalizing link, Cheney and DeFreitas designed a larger, more formal study and invited Bell to take part. That summer the two clinicians started sending coded blood samples to DeFreitas in Philadelphia. Some of the samples came from CFS patients, some came from the patients' close friends and family members and some from the general population. DeFreitas's job was to analyze them all, not knowing which ones were which.

For two years DeFreitas labored over the blood samples. Using an advanced technique called polymerase chain reaction, she searched each one for particular segments of HTLV-1 and HTLV-2. Last summer, results in hand, the three researchers unmasked the blood samples to see whether interesting patterns had emerged. They weren't disappointed. Indeed their results, reported in September at a scientific conference in Kyoto,
Japan, could open a whole new research frontier. Of the 30 samples drawn from CFS patients, fully 77 percent contained a distinctive piece of genetic material found in HTLV-2. Not one of the 20 samples drawn from the general population contained the same viral gene.

Ironically, the findings don't suggest that chronic fatigue syndrome is caused by HTLV-2. For while one of the virus's four genes was very much in evidence, DeFreitas could find no trace of a second. HTLV-2 would be an unlikely suspect anyway, given the way it is transmitted. Like the AIDS virus, it survives only in body fluids such as blood and semen. No one is sure how people get CFS, but it clearly isn't transmitted like AIDS. It's as likely to turn up in a pair of siblings as in a pair of sexual partners. The same is apparently true of the suspect DNA segment. Though it wasn't present in any of the blood that Cheney and Bell drew from the general population, more than a third of the the CFS patients' close, nonsexual associates tested positive.

The question is: positive for what? If the gene associated with CFS didn't come from HTLV-2, where did it come from? It could simply be "junk DNA," homegrown genetic material that happens to resemble a piece of HTLV-2. Or it could be a working part of some other infectious agent--a virus that is related to HTLV-2, that is transmitted differently and that can infect people without immediately making them sick. If the suspicious DNA were just hereditary detritus, it wouldn't normally initiate chemical reactions within the cell. But if it were part of a disease-causing agent, it would sometimes spring into action, manufacturing pieces of RNA in its own image. Symptoms would follow.

Before DeFreitas, Cheney and Bell went to Japan to present their findings, DeFreitas did one last experiment to see if they really meant anything. She gave some of the blood samples to a colleague at Wistar, who used a technique called in situ hybridization to check for newly minted RNA. The suspicious gene was silent in the healthy carriers she tested. But in six out of 12 actual CFS sufferers, it was actively churning out copies of itself. "I'd gone into this as skeptical as anyone," she says. "But when I had everything spread out on my desk, I decided I had to believe my eyes or not believe my eyes. If I believed my eyes, I had to tell people about this."

Still, the case is far from closed. DeFreitas, Cheney and Bell are the first to admit that their study was small, and that even if other researchers replicate it, the task of isolating a whole virus could take years. If the suspicious gene is eventually linked to a whole virus--no certainty, since some viruses break down inside the body, leaving only scraps like the one already recovered--many questions will remain. Does the agent cause illness by itself--a prospect that Cheney, for one, considers unlikely--or only when helped along by other factors, such as stress, or a second infection, or a genetic susceptibility? Is the ostensible virus specific to CFS, or will it show up in people suffering from other conditions, such as the unusual cancers that Seymour Gruffer-man has seen in North Carolina? If the culprit is a retrovirus, how is it transmitted? Is it an animal virus that people pickup through milk or meat (as Bell's local survey hinted)? Or is there a human retrovirus that, unlike the others, spreads through casual contact? Finally, why has the illness only now become so prevalent?

For all the remaining uncertainty on those fronts, a few things seem clear. One is that there's more to this illness than depression. No one denies that depression can be debilitating, but CFS strikes people whose psyches are demonstrably healthy and promptly ruins their lives. "Depression requires a loss of interest in everything," Cheney observes. "These patients are just the opposite. They're terribly concerned about what their symptoms mean. They can't function. They can't work. Many are petrified. But they do not lack interest in their surroundings."
The other certainty is that the mainstream medical community has been too slow to take the problem seriously. Though federal agencies started funding a handful of CFS studies last year, most of the research completed to date has been funded--and published--by patients' groups, or by obscure doctors struggling to get by in one of the least lucrative specialties in medicine. At least one leading researcher has financed his work by selling his office furniture and working graveyard in a hospital emergency room. Telling people they aren't sick is an easier way out when an illness so defies expectations. Unfortunately, it's a lie.

POLICEMAN'S BLUES: NEW YORK;

No one would accuse Gino Olivieri of lacking gumption. At 28, the former Detroit Lion was raising three kids with his wife, Janet, and relishing his job as a cop in Rochester, N.Y. "I was a canine handler, a SWAT team member, a field training officer. I mean I was go, go, go," he says. His illness came on suddenly in February 1988, after he fell into an ice-covered pond during a late-night burglary chase. He managed to pull himself from the chest-deep water and walk back out of the woods in subfreezing temperatures. After being treated for hypothermia at a local hospital, he developed bronchitis. But instead of receding after a few weeks, it turned into a horrible, unending flu.

Nearly three years later, Olivieri hasn't been back to work. Nor has he been out to dinner, attended a family wedding, observed a holiday or tossed a football with his kids. His wife videotapes the children's school functions so that he can at least see what he's missing. "I'm lightheaded," he says, squinting pale and unshaven through glacier-strength sunglasses. "The fatigue, it's deep in my muscles, deep in my bones. I have five or six different eye problems. I get nausea and lymph-node swelling throughout my body. I'm confused a lot of the time and I have tremendous insomnia. When you've got the flu, that first day's bad, but by the fourth day you're back to work. I don't see any change at all."

A DOCTOR'S VISIT

People with chronic fatigue syndrome suffer an array of unexplained symptoms. Eight of the following must persist or recur over six months for a person to qualify as having CFS.

Chills or low-grade fever

Sore throat

Tender lymph nodes

Muscle pain
Muscle weakness

Extreme fatigue

Headaches

Joint pain (without swelling)

Neurological problems (confusion, memory loss, visual disturbances)

Sleep disorders

Sudden onset of symptoms

SOURCE: CENTERS FOR DISEASE CONTROL

TROUBLE IN THE AIR: TEXAS

Dr. William Harvey, a specialist in aerospace medicine and a seasoned pilot, was about to land his small plane at Moffett Field in Sunnyvale, Calif., when he found to his horror that he couldn't make sense of the commands from the control tower. "It was like I was dyslexic," he says of the 1988 incident. When the same thing happened again two weeks later, he knew his flying days were over. Although physicians couldn't find anything wrong with him, Harvey's illness worsened to the point that he soon had to stop working altogether. After a year of sleeping or "sitting like a vegetable in front of the television," he found relief in gamma globulin, an experimental treatment that has shown promise in an Australian study.

Now 52, Harvey is finally starting to work again. Although he still suffers from muscle pain and persistent bladder infections, he has regained his vision and his clarity of mind. His frightening experience in the cockpit prompted him to write an article that is aimed at flight surgeons who pass judgment on pilots. It warns them about what the disease can do. He has also served as an expert witness in cases where CFS may have contributed to an accident.

TAUNTED TEEN: MISSOURI

"We get less sympathy than AIDS patients," says Chris Spalding, a 42-year-old single mother who worked as a union carpenter until she and her son developed CFS two years ago. The trouble started when Spalding and her two kids, Hoben (now 13) and Emily (now 6), came down with an intestinal flu. Emily was the only one who got better. "Hoben would fall asleep 20 times a night doing his homework, or he would fall asleep over an open drawer, looking for a T shirt," says Spalding, who now makes ends meet through telephone work and a part-time restaurant job. "Once he even fell asleep petting our dog."
Reducing the child's school hours seemed only sensible to Spalding. But the school system didn't understand. Truant officers dropped by regularly to lecture him on responsibility. His teachers thanked him sarcastically when he made it to class. The other kids taunted him in the hallways, calling out, "He has AIDS." At one point last year, Hoben talked about suicide. But nowadays he feels physically better. "I never feel energetic and ready to conquer the world," he says. "I just know I can survive." This year, as he entered the eighth grade, school officials flatly refused to let him attend half days. They said that unless he learned to handle full days he would never succeed in high school or a job. Eventually they relented. But, says Hoben's mother, "they still believe it's an attitude, not an illness."

NOT JUST THE BLUES

Chronic fatigue syndrome is sometimes dismissed as depression, but the two illnesses have very different effects on people's mental abilities.

Most people, whether healthy or depressed, can improve their performance on a memorization test by using cues. But cues are largely worthless to CFS patients, suggesting a diminished ability to form new memories.

A depressed person can handle a brief interruption when trying to memorize something. CFS patients' performance suffers six times as much from the interruption.

When asked to predict how they'll do on a cognitive test, depressed people tend to underestimate their abilities, but CFS patients tend to overestimate theirs.

A DOG'S LIFE: CALIFORNIA

Many patients in the CFS movement know the story of Murphy. Murphy was a seven-pound white Maltese. At the age of 5 1/2, the dog started to gain weight and turned into a picky eater. Over a period of two years, Murphy became terribly lethargic and his lymph nodes got so tender that he winced when he was touched. Eventually his owners, Reed and Gloria Baker of Riverside, Calif., had him put to sleep.

Gloria Baker suffered from chronic fatigue syndrome. Murphy got sick about two years after she did. And because routine tests showed that she and the dog shared several blood abnormalities, researchers performed a full immunologic analysis once the dog was dead. Oddly enough, Murphy tested positive for the newly identified HHV-6, a herpes virus that Baker was also fighting.

There may be nothing to such stories, but so many CFS patients mention having sick pets that one researcher, Dr. Paul Cheney, has conducted a survey to gauge the phenomenon. He will discuss his findings this month at a CFS conference in Charlotte, N.C. In Britain, a wave of bovine spongiform encephalopathy, better known as mad-cow disease, has led patients to wonder whether CFS is somehow related to that phenomenon. Most experts doubt that a single infectious agent is making people and animals sick; viruses tend to be highly adapted to one species or another. But because the illness is still such an enigma, the
possibility can't yet be ruled out.

**SPREADING THE NEWS: NORTH CAROLINA**

After 11 years, Marc Iverson is used to being sick. No longer able to hold a regular job, even to plan a day in advance, he does what he can to help other sufferers. As president of the 10,000-member CFIDS Association, a national patients' group based in Charlotte, N.C., Iverson often works by phone, from his bed. "The original script for my life was shredded," he says, "but the new one isn't so bad."

At 27, Iverson was a Yuppie superachiever. He had sailed through Colgate, Wharton and Harvard and become a vice president at BarclaysAmerican. Suddenly, in the fall of 1979, his energy evaporated. He got vertigo. He couldn't remember things. All that came of four trips to the Mayo Clinic and a month in a psychiatric ward was a mountain of medical bills. The CFIDS Association was born in 1987, when Iverson started meeting with other local sufferers. The association and its sister organization, the San Francisco-based CFIDS Foundation, have since found a global constituency. The association publishes the quarterly CFIDS Chronicle, and members have funded $400,000 in research. Says Iverson, "They have no other way of fighting back."

*WITH MARY HAGER AND NADINE JOSEPH*

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