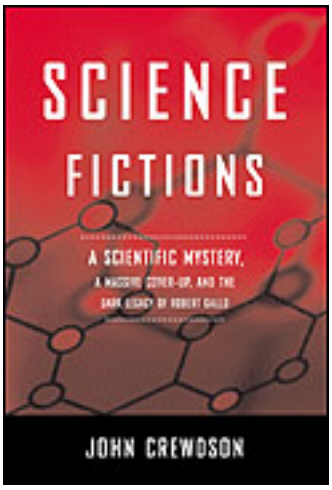
Science Fictions

by John Crewdson



1

"Too Lucky to Be True"

In the spring of 1981, a handful of young men began turning up in the emergency room at the UCLA hospital in West Los Angeles with the same mysterious complaint: a prolonged fever and swollen lymph glands, followed by a rare type of pneumonia previously seen only in the elderly or in malnourished children.

The most puzzling feature of their illnesses was what Michael Gottlieb, the UCLA clinician who examined them, described as an "acquired T-cell defect." Something was causing the men to lose large numbers of T-cells, the class of white blood cells that orchestrate the immune system's response to infections. Presumably, it was this T-cell depletion that had allowed the pneumonia-causing protozoan *Pneumocystis carinii* to take hold in the lining of their lungs. But Gottlieb's patients weren't elderly or malnourished. The thing they seemed to have in common was that they were all homosexuals.

Within months, the same immune-system disorder was being reported in gay men in New York, San Francisco, Miami, and Washington, D.C., as well as in a few hemophiliacs and recent immigrants from Haiti. By the summer of 1982, 500 cases of what would come to be called *acquired immune deficiency syndrome* had been recorded in the United States.

Few events capture the attention of doctors more readily than a

new disease, and in August 1982 several hundred specialists in cancer, immunology, and infectious diseases convened at Mount Sinai Medical Center in New York to share their ideas about its origins. It seemed that everybody had a different theory. Perhaps AIDS was the result of stress imposed on the immune system by an overload of different viruses, a syndrome often seen in developing countries. Perhaps it was the immune-suppressing properties of semen, or the chemical stimulants some gay men inhaled during sex. One researcher, noting that no cases were being reported in populous countries like China or India, suggested that AIDS might have a genetic component. The more realistic proposals included some kind of transmissible agent, probably a virus. Many viruses were on the suspect list, most of them known to be blood-borne or sexually transmitted. According to the National Cancer Institute's Dr. Robert Gallo, the most "reasonable candidate" was his own laboratory's recent discovery, a human retrovirus called HTLV.

Gallo's quest for HTLV had begun a dozen years before, with what had appeared to be the first detection of reverse transcriptase in human leukemia cells. Gallo wasn't claiming to have found a leukemia-causing human virus, only to have seen its biochemical footprints. But *Time* heralded Gallo's discovery as "a crucial clue" to the eventual isolation of the first human cancer virus.

When another NCI laboratory, headed by a virus hunter named George Todaro, tried to reproduce Gallo's data, it failed. Using Gallo's methods, Todaro's lab found it was possible to detect what *appeared* to be reverse transcriptase in normal human cells as well as leukemic cells. Since healthy cells weren't infected by retroviruses, Todaro reasoned that Gallo's test for reverse transcriptase must be detecting some other kind of enzyme.

For the virus hunters, it was the first false start of many. Six months later, a team at the M. D. Anderson Cancer Center in Houston announced the isolation of the first human cancer virus. Named ESP-1, after Elizabeth S. Priori, the virologist who discovered it, the new virus had come from a five-year-old boy with a blood-cell cancer called lymphoma. According to the *New York Times,* ESP-1 looked like the real thing. Experts in Houston and New York, the *Times* said, had "proved to their satisfaction that it is not a contaminating animal virus and that it is not any virus hitherto discovered." Soon Gallo had confirmed the presence of reverse transcriptase in the cultures where ESP-1 was growing.

It was George Todaro's lab that proved Priori was wrong. ESP-1

wasn't a new human virus, merely the same mouse leukemia virus in which David Baltimore had found reverse transcriptase, and which had somehow contaminated Priori's cultures. Scarcely had the ink dried on Todaro's paper than Columbia's Sol Spiegelman announced the detection of a retrovirus in breast cancer cells. But there wasn't any breast cancer virus in Spiegelman's lab, only another animal virus contamination. More unfounded virus sightings followed, but rather than provoke a reassessment of the cancer virus program the false alarms seemed only to spur the cancer virus hunters on.

It was a rare weekend that didn't find Todaro or Gallo in their labs, but Gallo seemed to have a determination that even the other virus hunters lacked. "Gallo's a student of Roman history," recalled Dave Gillespie, who had left Spiegelman to become Gallo's chief molecular biologist, "and he ran his lab like a battlefield. Sometimes he would put two people on the same subject independently, knowing that one of them was going to get screwed and the other one would publish." On a visit to India, Gallo had even taken the trouble to seek out the briefly famous fifteen-year-old guru, Maharaj Ji, to ask where he might find the origin of cancer. "Look deep within the mysteries of life itself," the Perfect Master had replied, not a bad piece of advice.

In newspaper and magazine articles, Gallo's single-mindedness was frequently attributed to the death of his five-year-old sister Judith from childhood leukemia, an event Gallo recalled as the most traumatic of his young life, and which had transformed the Gallo household into a grim and joyless place without music or laughter, where Thanksgiving and Christmas were no longer observed. For all the grief it had doubtless caused, his sister's tragic illness brought the teenaged Gallo into contact with a succession of doctors and hospitals, ultimately including Boston's famed Children's Hospital and Sidney Farber, who pioneered the treatment of childhood leukemia with the chemotherapeutic drugs. What followed was an adolescent fascination with biology, then medicine and cancer—and, according to Gallo, an abiding resolve to track down and vanquish his sister's killer.

As spurious detections of reverse transcriptase continued to mount, those researchers who were not finding reverse transcriptase in every cancer cell were beginning to ask the obvious question: Where's the virus? RT, as reverse transcriptase was called for short, was unarguably the product of a retrovirus. But finding RT in a succession of human cancer cells without finding the virus was like following a trail of peanut shells without

finding an elephant.

Gallo explained that testing for RT was easy, requiring a relatively small number of malignant cells, whereas actually isolating the virus that was producing the enzyme required many more cells than could be grown in the laboratory with existing methods. Leukemic blood cells multiplied ferociously in the human body, but not so well in a laboratory flask, where they seemed to be missing some stimulus the body provided. If the mysterious growth-enhancer could be identified, perhaps enough cells could be grown in a flask to isolate a leukemia-causing virus.

To search for the elusive "growth factor" Gallo teamed a talented virologist named Bob Gallagher with a Pakistani technician, Zaki Salahuddin, who had been visiting friends in Washington when war broke out between his country and India. After the first few days of fighting, news arrived that Salahuddin's father had been killed and one of his brothers thrown in jail. Salahuddin, who had already concluded that serious biological research was impossible in a country where issues of *Nature* arrived six months late, promptly answered Gallo's ad for a laboratory technician in the *Washington Post.*

Gallagher and Salahuddin began by screening white blood cells extracted from fetal tissue. If a cellular "growth factor" existed, Gallagher reasoned, it would be most plentiful during the fetal period, when many kinds of cells grow prodigiously. In what seemed a surprisingly short time, the pair had isolated a substance that seemed to keep one type of leukemic blood cells alive in the test tube. The cells, designated HL-23, had come from a Texas woman with myeloid leukemia, and they had previously tested positive for reverse transcriptase. With the addition of the new growth factor, the cells continued to multiply until there were enough to put beneath an electron microscope, which uses beams of electrons rather than rays of light to illuminate objects too small to be seen with an optical lens.

At the edge of one cell, magnified thousands of times, was a small, round object that vaguely resembled a weather balloon on the distant horizon of a pockmarked planet. Upon closer examination, the balloon became a particle with a dinnerplate-shaped core resembling a retrovirus. All Gallo needed to nail down the discovery was a demonstration that the particles could transform normal blood cells into leukemic ones, the *sine qua non* of cancer virology. But before that crucial experiment could be attempted, the freezer containing the laboratory's minuscule supply of growth

factor somehow became unplugged.

It happened over a long holiday weekend, and by the time Gallagher and Salahuddin returned to work everything inside the freezer had thawed. Perhaps a janitor had knocked the plug from its socket. Perhaps a child had done it; people often brought their children to the lab on weekends. But neither possibility would explain why the controls on Salahuddin's incubator, a table-top oven that holds cultured cells at the approximate temperature of the human body, had been turned up to roasting levels. "Around that time a lot of weird things were happening," recalled a technician in Gallo's lab. "You'd go in on the weekends and somebody had changed all the dials on the counters. People were unplugging incubators. You'd come back on a Monday morning, and clearly something weird was going on. Because always, an attempt had been made to destroy something."

However the freezer had come unplugged, without new infusions of growth factor the HL-23 cells were soon dead, and with them whatever cancer-causing virus they might have harbored. Not only was the critical transmission experiment no longer possible, there wouldn't be any growth factor to supply other researchers who wished to confirm Gallo's results. But the original HL-23 cells remained, and Gallo could put things on hold while Gallagher and Salahuddin searched for a new source of growth factor. Or he could publish the data they already had collected, suggesting the presence of a virus that no longer existed.

Gallo chose to publish, not in Nature but in its American counterpart, the journal *Science.* Even before the HL-23 paper appeared in print, Gallo's discovery was in the news. The *New York Times,* chastened by too many false virus sightings, was no longer reporting cancer-virus discoveries, but the *Washington Post* still thought they were front-page material. While a half-dozen other human cancer virus claims hadn't panned out, said the *Post,* Gallo's newest virus had passed all the scientific hurdles. "I got up one morning and read the headlines with no forewarning whatsoever," Bob Gallagher recalled. "I was shocked to find that he'd been talking to the press. The paper was not out yet."

Behind the scenes, Gallo was mounting a frantic effort to reisolate the HL-23 virus, enlisting the help of a respected British retrovirologist, Robin Weiss from the Chester Beatty Laboratories in London. Weiss, who had first encountered Gallo at a cancer meeting a few years before, had initially been put off. "He was so loud," Weiss recalled. "He was charismatic, and yet he was loud.

He was complaining about others, and bragging that he was doing the greatest work. And I thought, 'Here's a guy to avoid.' I just didn't like his manner." By the time he arrived in Bethesda, Weiss had come to know Gallo better. "I saw that his aggressiveness and vanity was partly because he wore his emotions on his sleeve," Weiss recalled. "To my mind he wasn't covering things up. He was a raw, ambitious person. You knew where you were."

Like most European biologists, Weiss had been disdainful of the American virus hunters. "There was a certain amount of snobbishness about them taking the easy route," he said. "We real molecular biologists were working things out with chicken and mouse viruses. Sure there may be human viruses, but that became tainted with the excessive number of papers that never hardened up the claims. There was a monthly paper from Spiegelman's lab, bimonthly from Gallo."

Once he had seen it close up, Weiss was agog at the scope of the American cancer-virus program. Although British science has been spectacularly successful, it has traditionally been done with big intellects and small laboratories. But even Gallo's sprawling setup in Bethesda seemed modest compared to George Todaro's lab, recalled by Weiss as "a huge factory with a dozen postdocs and twenty-four technicians."

It took Weiss and Natalie Teich, a close colleague who accompanied him from London, only a few attempts before they hit pay dirt. Weiss and Teich hadn't found the missing growth factor, but they had somehow managed to re-culture the HL-23 cells without it, and to isolate once again what looked like a human retrovirus. Indeed, the virus that emerged from the new culture—Weiss named it HL23V, for HL-23 *variant*—grew even better than the original.

Carrying a supply of HL-23V-infected cells packed in dry ice, Weiss and Teich flew home to write up their dramatic rescue for *Nature.* Samples of HL-23V had begun making the rounds of the other cancer-virus labs, where they were put to every conceivable test. Once again, it was George Todaro's lab from which the bad news emerged: HL-23V wasn't a human virus after all, but a mélange of three animal viruses—a woolly monkey virus, a gibbon ape virus, and a baboon virus—jumbled together in a retroviral cocktail.

Gallo and Todaro shared some common roots. Both had grown up in Italian-American families within a few dozen miles of one another, Todaro in New York City and Gallo in Waterbury,

Connecticut, the Brass Capital of the World. Both had graduated from medical school in the spring of 1963, Todaro from New York University and Gallo from the Jefferson Medical College in Philadelphia. Both wanted nothing more than to find the first human cancer virus, but there the commonalities ended. Todaro's deceptively laid-back style and faintly melancholy demeanor contrasted sharply with Gallo's own hyperkinesis. When Todaro spoke, it sometimes seemed as if his brain was struggling to catch up with his mouth. Gallo's conversations often sounded as though a tape recording were being played back at faster than normal speed, and his syntax frequently lent the impression of someone whose first language was not English.

By all accounts, Todaro's seeming befuddlement disguised a Machiavellian brilliance. "Todaro totally intimidated Gallo," Dave Gillespie said. "If he had a meeting with Todaro, Gallo would be petrified going into the room." On this occasion it was Todaro who delivered the verdict that, once again, something had gone terribly wrong in Gallo's lab. "There was, ah, a meeting," Todaro recalled, "where some of us, uh, did try to talk to Bob and tell him, 'You have a contaminant.' He, er, resisted for quite a while, but the evidence became overwhelming." Gallo's capitulation came during a day-long cancer-virus symposium in Hershey, Pennsylvania, where one speaker after another presented data showing that HL-23V was not a human virus. "I certainly took a lot of punches," Gallo said, years later. "It wasn't one, it wasn't two—how about *three* primate retroviruses mixed together?"

Within a few months, *Nature* had published a retraction of HL-23V. "You could look for faults and say someone should have been more cautious," said Robin Weiss, who regretted having gotten involved at all. "But I don't remember thinking that it didn't hold up, or that it didn't feel right at the time we got the paper submitted. I may have been over-influenced by Gallo as a pushy, charismatic fellow. I think we should have started wondering earlier. Gallo never quite forgave Natalie and myself for growing up that virus to the stage where it could be shown to be a contamination."

Nobody who worked with viruses, including Weiss, believed Gallo's lab had been the victim of an accidental contamination. "What else could it have been but deliberate," Weiss said, "if it was three viruses instead of one? Or at least two? If it was a rival group at NIH looking for human retroviruses, one could certainly make an enormous fool of Gallo." As Weiss thought back over his time in Bethesda, he remembered the weekend that George Todaro turned up unexpectedly at Gallo's lab. "He got me to let him in," Weiss

said, "and he chose a time when Gallo and Gallagher and everyone else was out of town. He marched in and opened every incubator and looked at the cultures. He said, 'What are you growing there?' I distinctly remember this over-curiosity about details, that one quiet afternoon or evening."

"Totally untrue," Todaro says. "Absolutely not true. Never happened. I distinctly remember never having gone there on a weekend. You can get three viruses into a virus preparation easily just by being sloppy, and Gallo had plenty of sloppy people."

Courtesy of the War on Cancer, the National Cancer Institute's budget had more than doubled in four years, from $377 million in 1972 to $815 million in 1976, an extraordinary increase for any federal agency in peacetime. But even before HL-23, many of the cancer virus labs had been closed down in the face of protests from university scientists, who viewed the spectacularly unsuccessful cancer-virus program as a waste of precious research dollars that should have been flowing to other laboratories, preferably their own.

The debacle of HL-23 represented the final straw. What remained of the cancer-virus program was placed in serious jeopardy, and Gallo's career as well. "When the virus disappeared, the NIH would have preferred Gallo to disappear along with it," recalled Vincent DeVita, who then headed the NCI's Division of Cancer Treatment. "The only reason he didn't was me. He's a handful. He cost me a lot of time. I spent hours sometimes to calm him down. But there are two kinds of investigators in this world. There are people who discover things, and there are people who build a brick at a time. You need both. Discoverers are rarer, and I think Gallo, frankly, is one of those. That's how I saw him, his personality problems notwithstanding."

It would be said later that Doris Morgan was a technician who got lucky. But that would have been unfair. After years of culturing cells, it occurred to Morgan that she was at least as bright as some of the scientists who were designing the experiments she was carrying out. A divorce propelled Morgan into graduate school, from which she emerged with a Ph.D. in hematology and a postdoctoral fellowship at M. D. Anderson, followed by a job offer from Gallo as the HL-23 affair was coming to a head. Morgan leapt at the chance to work at the National Cancer Institute, but she was a bit apprehensive about her new boss. "A lot of the new people coming in had difficulty," she recalled. "It was not an atmosphere

that really generated a lot of confidence for a young scientist."

Morgan's first assignment was to join the search for the erstwhile growth factor that could reignite the HL-23 culture. "My directive from Gallo," Morgan recalled, "was 'We have to grow myeloid leukemic cells, so we can reproduce HL-23.'" After many months in cold storage, the HL-23 cells were not in good shape. Morgan thawed them anyway, rinsed them with water, and put them in a bath of nutrients and amino acids, the molecules from which proteins are constructed. When the cells failed to grow, Morgan concluded that HL-23 was history, and turned her attention to culturing cells from other cancer patients.

With one group of cells Morgan had better luck. "If you kept these cells too far apart they didn't do well," she recalled. "They appeared to need contact and interaction with each other." Each morning Morgan went to her incubator, took out her test tubes, and studied the visible clumps of cells at the bottom. When the microscope confirmed that one batch was indeed multiplying, Morgan split it in two, feeding half the cells with a bean-plant extract called PHA. "I wasn't sure which way they would be happiest," she said. Eventually Morgan concluded that the PHA-stimulated cells were producing some kind of biochemical that encouraged them to grow. Moreover, the fact that the surviving cells were all of the same kind suggested that the growth factor was specific for one type of cell. But what kind of cell that was, Morgan had no idea.

Once a week, usually on Monday afternoons, Gallo summoned his staff to join in a roundtable critique of each other's work. A few researchers would be designated to speak about their current experiments, then subjected to questions that were not always polite. When Morgan's turn came to talk, she reported that she might have found a factor that induced white blood cells to grow in culture. When someone asked what kind of white blood cells, Morgan admitted she hadn't a clue. The response was far from enthusiastic. "There was a total indifference," Morgan recalled. "'This is not what we were after. This is a failure.'" The only encouragement came as the meeting was breaking up. "Bob Gallagher came up to me and said, 'I think you've got something really interesting. Take it to the immunologists and find out what it is.'"

The mid-1970s were the Dark Ages of cell biology, a time when it was barely possible to distinguish one type of blood cell from another. The immunologists told Morgan what she already

suspected, that she hadn't been growing myeloid cells. They were, nevertheless, white blood cells, which meant they were probably lymphoid cells, or lymphocytes, then considered the dross of hematology. Myeloid cells were complex, while lymphocytes were simplistic. Myeloid cells were involved in interesting leukemic diseases. When lymphocytes became malignant, they caused uninteresting lymphomas. Soon a new joke was circulating at NIH: the good news is that Gallo has finally found a cellular growth factor. The bad news is that it's for growing lymphocytes.

In those years, lymphocytes were classified into two broad categories: B-lymphocytes, thought to come from the bone marrow, and T-lymphocytes, believed to originate in the thymus. According to the immunologists, Morgan's cells appeared to be mostly T-cells—not leukemic T-cells, but *normal* T-cells. Discovering something that induced healthy T-cells to grow was like finding a better way to grow weeds. "I was on the fringe," Morgan said, "and no one was paying that much attention to what I was doing. Gallo had no interest in this. It almost got discarded."

Morgan's self-appointed savior was Frank Ruscetti, who had joined the lab after encountering Gallo at a reception during a medical meeting in Atlanta. "I was hired at a cocktail party," recalled Ruscetti. "Gallo said, 'Don't you want to come to the lab that's going to win the Nobel Prize?'" Ruscetti accepted Gallo's invitation, only to discover that he, like Doris Morgan, was expected to find the erst-while HL-23 growth factor.

Ruscetti had begun by searching established cell lines one at a time, but without any luck. When he sensed that Doris Morgan had found something interesting, his antennae began to shift in her direction. "Doris basically didn't know what to do with it," Ruscetti recalled. "So we sort of pooled our resources and our energies and did it together. She was constantly in trouble with Gallo, because she would only do things her own way. She could only grow these cells in test tubes, and not in flasks. I was basically trying to help her, because it was obvious that if she didn't change her ways she'd get fired."

It was help Morgan thought later she might have done without. When her discovery of what Gallo dubbed T-Cell Growth Factor was published in Science, in the fall of 1976, the authors of the article were Morgan, Ruscetti, and Gallo. A follow-up paper a few months later was by Ruscetti, Morgan, and Gallo. Not long after that, Doris Morgan was gone from Gallo's lab. "I was, I think the expression is, canned," Morgan said. "When I got fired, someone in the lab

drew a silhouette of a person rejected, with a tear dropping. And under it he wrote, 'Don't cry, Doris. There's more to T-cell biology than any of us realizes.'"

The laboratory Doris Morgan left behind was no longer so close to the center of the scientific universe. Still, a postdoc with an interest in T-cell leukemias could find a less interesting place to work. Bernie Poiesz had spent the first year of a three-year NIH fellowship at the Veterans Administration Hospital in Washington, where he had seen a number of patients with relatively rare T-cell skin cancers. Poiesz had become curious about the origins of those cancers, and he wanted to use the remaining two years of his fellowship to do some laboratory research. He had chosen Gallo's lab, Poiesz said later, mainly because "I liked Ruscetti's spiel."

When Poiesz and Ruscetti drew up a proposal to study malignant T-cells, they decided, almost as an afterthought, to test the cells for reverse transcriptase as well. But the proposal hadn't met with much enthusiasm from Gallo and his staff, who were no longer looking for human cancer viruses. "They had kind of given up on it," Poiesz recalled, "but we told Bob that this was our project, and he told us to work on it for a few months." When Doris Morgan, who had found a new job in Daniel Zagury's lab at the University of Paris, heard that Frank Ruscetti was again growing T-cells, she was surprised. "After I left Gallo I thought the whole thing would be abandoned," Morgan said. "I said to myself, 'Why is Frank growing T-cells?'"

Some of the T-cells in Poiesz and Ruscetti's incubators had come from Charles Robinson, a twenty-seven-year-old patient at the VA hospital diagnosed with a T-cell skin cancer called mycosis fungoides. When one of Robinson's lymph nodes was biopsied, it had been Poiesz who authorized the surgery. He hadn't thought more about the man until he got a call from Adi Gazdar, an NCI pathologist who was interested in Robinson's case. Using a variety of laboratory tricks and a dash of T-Cell Growth Factor supplied by Ruscetti, Gazdar had succeeded in growing Robinson's leukemic T-cells in a flask. As long as the cells were fed with nutrients, they went on reproducing, transforming themselves into a self-perpetuating culture known as continuous cell line that produced an endless quantity of malignant blood cells to study.

In return for the T-Cell Growth Factor Gazdar promised to share with Ruscetti any continuous T-cell lines he was able to establish. In addition to the Robinson line, which Gazdar named HUT-102

(for Human T-cell line number 102), Gazdar had thrown in another line, HUT-78, established without T-Cell Growth Factor from the cells of a patient suffering from another kind of T-cell cancer called Sezary's syndrome. When HUT-78 proved negative for reverse transcriptase— an indication that it didn't contain a retrovirus— Ruscetti stuck the cells in his freezer and forgot about them.

It was nearly Christmas of 1978 by the time Bernie Poiesz got around to testing Charlie Robinson's T-cells for RT. The cells were positive, but at such a low level that, according to the protocol previously established by Gallo's lab, further testing would have been pointless. "In order for something to be considered interesting," Poiesz said, "there had to be an extraordinary amount of reverse transcriptase. That would have required that the cell line be spewing out retrovirus. But I had done enough reverse transcriptase assays on normal T-cells to recognize that if there was a little bit of production, that's all you needed."

When Robinson's cells were compressed into a pellet, thinly sliced and put beneath the electron microscope, Poiesz could see what Gallagher and Salahuddin thought they had seen five years before —retrovirus-like particles escaping from the surface of a cell. But plenty of people claimed to have detected "virus-like particles" in human cancer cells without ever isolating a virus.

After a decade of animal virus contaminations HUT-102 would have to be checked against every available animal retrovirus. Poiesz and Ruscetti agreed to keep Gallo in the dark until they were certain of what they had found. "Gallo wasn't even aware of its existence for something like six months," recalled Ruscetti's postdoc, Tina Eastment. "Because of the previous faux pas, they wanted to make sure to do all the right tests and everything. They knew that Bob would go and make a big deal out of it right away, and they wanted to make sure that neither they nor he were going to be made fools of."

Gallo, who had been on a visit to Europe when the cross-checking was finished, learned about the discovery of the first human retrovirus over cocktails in his backyard. "Gallo came back and gave a party," Poiesz recalled, "to which he invited John Minna, who was Adi Gazdar's boss and lived down the street," and who had known about the discovery for two or three weeks. "The last word I said to him," Poiesz recalled, "was 'Please don't tell Bob until after we're sure. And second of all, we should tell him, you shouldn't.' " Minna evidently hadn't been able to resist. "Gallo called me up in the middle of the night," Ruscetti said, "and gave

me hell."

Even then, Gallo was skeptical that Poiesz and Ruscetti had succeeded where he had so often failed. "Gallo basically didn't believe it," recalled Ruscetti's technician, Andrea Woods. "He didn't believe Bernie's work, but Bernie pursued it." Once the importance of the discovery had registered, Gallo "was a bit astounded that this had happened," Poiesz recalled. "Because they had tried so long to find a retrovirus. Some very skilled people had tried it, and here was a junior member of the team coming in, and in the first months of working he finds the first human retrovirus. That seemed too lucky to be true."

To make sure he and Ruscetti were really first, Poiesz began a search of the scientific literature. In an obscure European journal, he found what looked like a close call. The year before, a group in the Netherlands had reported observing virus-like particles and detecting reverse transcriptase in cells from seven patients with mycosis fungoides and two more with Sezary's syndrome. The article contained several pictures, electron micrographs of particles that looked very much like those around Charlie Robinson's T-cells.

Most of the Dutch data was the work of a twenty-four-year-old graduate student, Elizabeth van der Loo, who had been finishing a degree in dermatology when she stumbled across what looked like a retrovirus in a mycosis patient. "It was like a hobby, you know, to look everywhere for retroviruses," van der Loo recalled. "I found them after searching a very, very long time." Not being a virologist, and not knowing how to isolate the virus she had seen, van der Loo had simply published her findings and continued her dermatology studies.

Poiesz made a copy of the van der Loo paper and gave it to Ruscetti, along with another article he had stumbled across, a three-year-old report from Japan of a previously unrecognized disease called Adult T-Cell Leukemia. As Poiesz read about the Japanese leukemia patients, he thought the article could have been describing Charlie Robinson. The new disease involved T-cells. Once people began to exhibit symptoms, they didn't live very long. The Japanese patients had extraordinarily high levels of calcium in their blood. So had Robinson, and there were other similarities.

The one thing that set Robinson apart from the Japanese patients was that he had been diagnosed with mycosis fungoides. Nearly all the Japanese leukemia patients resided in the same area, Kyushu

province on the southwestern tip of Japan, which implied that ATL might be caused by a transmissible agent. The Japanese doctors who had discovered the disease weren't ready to rule out genetic factors, but they urged that the possibility of a cancer-causing virus also be explored.

The tradition of naming viruses after their discoverers ended with Tony Barr and Yvonne Epstein, discoverers of the Epstein-Barr virus, a member of the herpesvirus family that causes infectious mononucleosis and may play some role in Burkitt's lymphoma. As the number of virus discoveries mounted it seemed less confusing to identify new viruses according to what they did or where they had been discovered, rather than who had found them. The name Gallo proposed for Poiesz and Ruscetti's discovery was Human T-Cell Leukemia Virus, or HTLV. Poiesz objected that Robinson had died of lymphoma, not leukemia. "Gallo wasn't real happy," recalled Andrea Woods. "He wanted a leukemia virus. He kept saying, 'Couldn't we call it a T-cell leukemia virus?' But the people around him said no."

Gallo's next suggestion, Human T-Cell Lymphoma Virus, still overstated the case. Poiesz had found the virus in a single patient — proof of nothing, certainly not that the virus had been the cause of Robinson's lymphoma. Nor did Poiesz have any conclusive evidence that the virus was human, only that it wasn't one of the animal retroviruses against which it had been compared. "We hadn't proven that it had caused disease," Poiesz said, "nor did we have any idea about where it was in the world. But HTLV is the name that Gallo wanted it to be."

Only the year before, human cancer viruses had been dismissed as heresy by no less than the University of California's J. Michael Bishop, who declared in a widely reprinted lecture that "few investigators would now argue that infection with a retrovirus is the sole cause of any malignant process in human beings." The only justification for putting so much time and money into the futile search for human cancer viruses, Bishop said, was that the virus hunt had aided science's understanding of human cancer genes, "not the vague hope that human cancer will turn out to be a virus disease."

With the scientific winds blowing so strongly against him, Gallo's chances of announcing the discovery of the first human retrovirus in Nature or Science were nil. But there was one journal where the paper might have a chance—the *Proceedings of the National Academy of Sciences,* to which prospective articles are

"communicated" by a member of that august body, who thereby attaches his or her reputation to the significance of the data therein. If *PNAS* had more lenient standards than *Nature and Science,* it also had less prestige and visibility, but what mattered was getting HTLV into print.

In search of sponsors, Gallo approached David Baltimore and Henry Kaplan, the Stanford radiologist whose pioneering treatment had made Hodgkin's disease a survivable cancer. Kaplan had gotten to know Gallo after being appointed by Vince DeVita, in the wake of HL-23, to provide an independent assessment of Gallo's research. Initially skeptical, Kaplan had come away impressed by Gallo's persistence. But Kaplan, like Baltimore, was wary of Gallo's latest claim. To allay their concerns, Gallo invited the two to meet with Poiesz and Ruscetti. "We presented our data to them," Poiesz recalled, "so they could tell us if they saw any flaws in it. And they couldn't find any flaws."

Scientific convention holds that first authorship of a scientific article be reserved for the person who has done the most to produce the data, with the second author having made the next-most-important contribution, and so on down to the last author, a slot traditionally reserved for the principal investigator of the study that produced the discovery or the chief of the laboratory in which it occurred. Shortly before the HTLV article was submitted to *PNAS,* Gallo took Frank Ruscetti aside to suggest that he, Gallo, should be the paper's first author. "He told me, 'You know, there's no reason that the person who did most of the work should be first author,' " Ruscetti said. When Poiesz learned what Gallo had planned, "I advised him that it would probably be best that he be the last author," Poiesz recalled. "I told him I felt fairly strongly about that."

When "Detection and Isolation of Type C Retrovirus Particles from Fresh and Cultured Lymphocytes of a Patient with Cutaneous T-cell Lymphoma,"d communicated by Henry Kaplan, appeared in December 1980, the first author was Bernie Poiesz, followed by Frank Ruscetti and Adi Gazdar, with Gallo bringing up the rear. Five years before, the discovery of HTLV would have caused a scientific sensation. But with memories of HL-23 still fresh, HTLV was ignored by the scientific journals and the popular press. A follow-up article, communicated to PNAS by David Baltimore, also sank without a trace. The only interest in Gallo's new virus seemed to be in Japan.

Three months after the publication of Poiesz and Ruscetti's first

paper, Gallo found himself at a former Buddhist monastery on Lake Biwa, in the mountains outside Kyoto, a place remembered by the French virologist Guy de Th? as "very beautiful and quiet, with lots of grass." The ostensible reason for the Lake Biwa symposium was to discuss recent advances in human tumor virology. The real topic of the meeting was the mystery of what caused Adult T-Cell Leukemia.

At first the Japanese thought ATL might be an aftereffect of the atomic bombing of Nagasaki, the capital of Kyushu prefecture, where the disease was most prevalent. That theory was discarded when ATL patients began showing up who were born after the bomb was dropped. Southwestern Japan is warm and humid, a place where insects thrive, and insects are responsible for the transmission of many exotic diseases. But if ATL were caused by an insect-borne microbe, why was it being seen only in adults and not children? As the possibilities were considered and eliminated, the explanation that made the most sense was a slow-acting virus.

At the time of Lake Biwa, HTLV was a virus in search of a disease. Despite Charlie Robinson's diagnosis, when Poiesz and Ruscetti had tested blood cells from other mycosis patients they yielded no trace of HTLV. Nor did HTLV seem to be present in people with ordinary T-cell lymphomas. Indeed, the only other patients in whom Poiesz and Ruscetti reported any evidence of HTLV were a sixty-four-year-old Trinidad woman, M.B., and a merchant seaman from Boston, M.J., both of whom suffered from Sezary's syndrome. But when fifty other Sezary patients were screened for HTLV, every last one was negative. "We were striking out," Poiesz said.

If HTLV didn't cause mycosis fungoides or Sezary's syndrome, Poiesz reasoned, perhaps it caused this new disease from Japan, and he recalled suggesting to Gallo that the Adult T-Cell Leukemia patients be tested for HTLV. The summer before Lake Biwa, Gallo had gotten in touch with the chairman of the microbiology department at Kyoto University, Yohei Ito, to ask if Ito could spare a few blood samples from his ATL patients.

The subject of Gallo's own talk at Lake Biwa was T-Cell Growth Factor, which Gallo now counted as a historic breakthrough in the annals of virology.f But the presentation most vividly recalled by Guy de Thé had come from one of Gallo's assistants, Marjorie Guroff, who reported that some of the ATL samples had indeed contained anti-bodies that reacted with HTLV—an indication that the patients from which they had come were infected with HTLV, or a virus very much like it.

As Guroff spoke, Japan's most prominent virologist, Yorio Hinuma, the director of Kyoto University's virological institute, listened in imperious silence. Hinuma had intended to speak about his work with the Epstein-Barr virus. But as he recognized that the Americans were trying to link HTLV to ATL, and thereby gain the credit for discovering the cause of a Japanese disease, Hinuma informed Ito that he was changing the topic of his talk.

Like Marjorie Guroff, Hinuma had been testing ATL blood samples for evidence of a human retrovirus, and he had found a positive reaction in twenty-five cases out of twenty-five. The Japanese hadn't yet isolated the virus in question, which put them behind the Americans, but they had one crucial thing that Gallo lacked: incontrovertible evidence of virus-like transmission from one patient to another.

Around the time Bernie Poiesz was joining Gallo's lab, a young Japanese virologist, Isao Miyoshi, had drawn ten milliliters of blood from a farmer with ATL. When the farmer's cells were cultured in Miyoshi's lab they continued to grow, leading Miyoshi to suspect, correctly, that they must be making their own growth factor. Miyoshi named the cells MT-1, thereby christening the world's first human leukemic T-cell line.

Miyoshi's most inspired experiment came with a second cell line, MT-2, established by mixing the cells of a forty-five-year-old female ATL patient with those from a healthy newborn baby. It was Miyoshi's choice of a male baby which allowed him to distinguish, via chromosomes, the baby's previously normal blood cells from the woman's leukemic ones. When the baby's cells became leukemic, the only explanation was that the woman's cells contained a transmissible cancer-causing agent.

Judging from Hinuma's electron micrographs, that agent was a retrovirus. But the Japanese hadn't been able to fish their virus out, and over dinner Gallo offered to help, proposing that the Japanese provide him with more blood samples from ATL patients as well as from healthy Japanese living in the region where ATL was endemic. If Gallo could isolate a virus from the Japanese patients using the methods that had produced HTLV, that virus could be compared with HTLV. Perhaps the virus Hinuma and Miyoshi were chasing had already been discovered by Poiesz and Ruscetti.

Yohei Ito welcomed Gallo's proposal. But Guy de Th? remembered

Hinuma declaring that no collaboration with the Americans would be necessary, that Japanese science could solve the riddle of ATL without the National Cancer Institute. What had begun as a collegial dinner in a celestial setting ended with what Guy de Thé recalled as "a silence that I will never forget."

"Gallo was far enough ahead on the virology that he thought he could scare the Japanese into being docile collaborators," said Frank Ruscetti, whose attendance at Lake Biwa had been canceled by Gallo at the last minute. "But he was stunned, because the Japanese had been working with typical Japanese enthusiasm and already had some data and some epidemiology themselves."

In the end, it was the Japanese who convinced the National Cancer Institute that HTLV was the genuine article. Because Miyoshi's report of MT-2 had appeared in a Japanese-language journal, Bernie Poiesz's library search had missed it. As punishment for Miyoshi's collaboration with Hinuma, Yohei Ito hadn't invited Miyoshi to present his data at Lake Biwa. For most Western scientists, the first chance to see the Japanese data came ten months after HTLV, in the autumn of 1981, when Hinuma's pictures of a "Type-C retrovirus" appeared in PNAS, followed a few months later by Miyoshi's MT-2 data in *Nature.*

Gallo rushed to tell Vince DeVita, now the NCI's director, about the reports from Japan, according to Gallo the first "clear-cut unambiguous and noncontroversial" association between a retrovirus and a human cancer, albeit a rare and exotic cancer. "The story," Gallo assured DeVita, "is really breaking very nicely." DeVita's memories of HL-23 were too recent to permit much enthusiasm over Gallo's initial accounts of HTLV. Now that the Japanese had weighed in, DeVita allowed himself to believe that Gallo might have finally snared a human cancer virus. "This time it looks real!" DeVita wrote across the margin of Gallo's note.

It had been a decade since David Baltimore's NIH lecture had launched Gallo on his quest, five years since Gallo's public evisceration at Hershey. At last he seemed to have prevailed, and some of his colleagues applauded his perseverance.

"We stopped looking, but he didn't," said Stu Aaronson, one of George Todaro's postdocs. "I have a lot of admiration for that kind of constant drive. Rather than sort of going and hiding in a corner and giving up, he kept persisting. And that drive, it's unbelievable to me, the degree of that drive to find something. He's a very, very determined guy, and he finally found one. I've never seen a

fighter like him, in terms of being willing to take on almost anybody."

Vince DeVita was persuaded that HTLV was real, but many of the questions raised by Bernie Poiesz's discovery had yet to be resolved. In addition to causing T-cell leukemia, was HTLV also the cause of Charlie Robinson's mycosis fungoides? What about M.B.'s Sezary syndrome? Three different diseases seemed a lot to ask of one virus. To answer such questions, the Japanese were eager to compare their Adult T-Cell Leukemia virus with Gallo's HTLV. All they needed was some of Gallo's virus. Most major scientific journals require that, once an author publishes the discovery of a virus or a cell line, he is obliged to share it with other researchers. The NIH has a similar rule of its own. But Gallo's seeming reluctance to allow HTLV to leave his lab led to what George Todaro recalled as "a feeling around NIH that there was something, ah, *wrong* with HTLV."

With Vince DeVita's ascendancy to the NCI director's office Gallo's future was assured, but Todaro's days were numbered. "They were constantly at war with each other," DeVita recalled. "The tension they created was very negative, and clearly they both couldn't stay. Todaro is the Mozart of schemers. He was absolutely brilliant at it. Very, very smart guy. He certainly had a capability of doing good science, and he did good science. He also had far-flung facilities, unbelievable money squirreled away, I think $13 million a year. We cut him back to about $3 million, at which point he decided to leave. Basically, we opted for Gallo, and Todaro left. They hate each other."

"Gallo was like a son to DeVita," Todaro said later. "At least I was Mozart, not Salieri."

Any comparisons between HTLV and ATLV would have to be done in Bethesda, and Miyoshi agreed to send Gallo the ATLV-infected cell line. To perform the comparison Gallo chose a forty-year- old Czechoslovakian virologist, Mikulas Popovic, whom he had met over a drink at a conference in Norway and invited to spend a year in his lab.38 After Popovic reported in Nature that ATLV from Japan appeared identical to HTLV 39 Gallo began distributing HTLV to a few laboratories i - although not before changing its name from Human T-Cell *Lymphoma* virus to Human T-Cell Leukemia virus.j At last Gallo had his human cancer virus.

Only a supremely suspicious person—in Gallo's view, only a

paranoid person—could seriously imagine that Gallo, having belatedly discovered HTLV to be yet another animal virus contamination, had induced the Japanese to send him their virus, then switched test tubes and passed the Japanese virus off to the outside world as the virus discovered by Poiesz and Ruscetti.

It was an impossible proposition to prove—or disprove. But that mattered not to Abraham Karpas, a Lithuanian émigré who had begun his career as a veterinarian tending Swiss milk cows. Karpas claimed an impressive succession of jobs: the Pasteur Institute in Paris, the Boston Children's Hospital lab of the legendary virologist John Enders, and with Albert Sabin, the co-inventor of the polio vaccine. "I must tell you," Karpas said of Sabin, "he's a terrible man. He treated his people terribly. That's one of the reasons he never got the Nobel Prize."

Abe Karpas presided over a small lab at the Molecular Biology Laboratory in Cambridge, England, the successor to the hallowed Cavendish Laboratory where Crick and Watson had started it all. Karpas was a talented grower of cells, although not quite the sort of scientist one might have expected to find sharing a table at lunch with Sir Max Perutz or Professor Sir Aaron Klug, and there was some truth in the suggestions that Karpas's career owed much to his rapier wit and his talent for ingratiating himself with the mandarins of molecular biology.

Karpas considered Robin Weiss his archrival, and Weiss was convinced that Karpas had been carrying on a whispering campaign with the high priests of Cambridge to deny Weiss the thing he wanted most: the three initials after his name, "F.R.S.," that denote membership in the Royal Society, the British equivalent of the American National Academy of Sciences.

Karpas cheerfully acknowledged Weiss's suspicions. "I haven't exactly been whispering," he said, and Weiss was only slightly more charitable toward Karpas. "Abe was claiming he had viruses in this and that leukemia," Weiss said, "and he wouldn't collaborate with any molecular biologists. In the floors above him, in the same building, there were bunches of them. There were Nobel prizes sort of hanging off the windows."

But Weiss thought anyone who wrote Karpas off as a poseur was making a mistake. "Although he's difficult and truculent," Weiss said, and "does half-a-dozen half-baked things instead of one thing decently, he actually has some flashes of intuition which most of us don't get. I've always thought he's remarkably similar to Gallo,

and that's why they hate each other. They think everyone else is out to get them, so it ends up that people are. They claim things in retrospect that never happened that way. They're very similar personalities, a mixture of paranoia and ambition, but with some real scientific imagination."

Like Gallo, Karpas had spent years looking for a human cancer virus, and he had watched with glee as Gallo's various discoveries turned to dust. "His level of biological intuition is very low," Karpas said, nevertheless insisting that his feud with Gallo wasn't personal. "He sets a very bad example for young people," Karpas said. To anyone who would listen, Karpas pointed out what seemed to him telling discrepancies in the HTLV story, including the absence of any evidence that HTLV was a human retrovirus, and the genetic differences between different isolates of HTLV.

Karpas, who thought two isolations of the same virus from different patients should be much more alike than not, was particularly fond of observing that the virus from Charlie Robinson bore only a superficial resemblance to the one from the Trinidadian woman, M.B.k Karpas seized on these and other disparities, such as the statement in one of Gallo's articles that an upcoming article by Poiesz would report the detection of HTLV DNA in leukemic cells from two new patients. If true, that would make it far more likely that HTLV was indeed a human virus and not an animal contaminant. The fact that one of the two infected patients was described as "a child with acute lymphatic leukemia" was even more interesting, since HTLV-induced leukemia typically takes decades to show up. But when Poiesz's article appeared, it hadn't contained the promised data.

In Yorio Hinuma, Abraham Karpas found a kindred spirit. Convinced that Gallo hadn't discovered what the Japanese had found, Karpas and Hinuma launched a search for ATLV in patients with mycosis fungoides and Sezary's syndrome. When no virus materialized from either group, Karpas and Hinuma declared that HTLV couldn't possibly be the same as ATLV, and they weren't alone in suspecting something amiss with HTLV.

"When the American papers are closely scrutinized," another Cambridge scientist wrote to the British medical journal The Lancet, "they turn out to depend a great deal on cross linking and sometimes it is well nigh impossible to extract the evidence, but in order to disentangle them one has to spend many hours trying to unravel the threads. Conversely the Japanese work is impeccable, straightforward and highly regarded....Quite a number of workers,

including myself, are beginning to wonder what the situation really is."

The principal interest in HTLV was not among clinicians, who except for those in Japan had never seen a case of Adult T-Cell Leukemia, but among molecular biologists, who wanted to know what made the first human retrovirus tick. Did it resemble the animal tumor viruses? Or was there some genetic oddity that distinguished HTLV from its animal counterparts?

The answers hadn't been forthcoming from Bethesda, because nobody in Gallo's lab was capable of cracking HTLV's genetic code. But the Japanese had the capability, and they were literally taking ATLV apart. Using a technique invented by the legendary Cambridge scientist, Fred Sanger, that made it possible to "see" DNA as smudges on a piece of X-ray film,m the Japanese had assembled the complete DNA sequence of ATLV, a string of 9,032 nucleic acids, or nucleotides— adenine, thymine, guanine, and cytosine -laid end-to-end. "The Japanese worked very fast and very hard," conceded Bernie Poiesz. "They beat us to the punch."

Not all the Japanese had accepted Mika Popovic's conclusion that ATLV and HTLV were the same virus by different names, and in their publications many Japanese had continued to use the term ATLV. Now that Gallo at last was willing to share HTLV, the Japanese could perform the same comparison on their own turf. Although there were some minor genetic differences, the Japanese agreed that ATLV and HTLV represented "the same species of retrovirus," and, therefore, that both were the cause of Adult T-Cell Leukemia. Because Poiesz and Ruscetti had published first, with the exception of Yorio Hinuma the Japanese agreed to drop the term ATLV in favor of HTLV.

In public, Gallo repaid their deference. Addressing the biggest Japanese cancer meeting of the year, the Princess Takamatsu symposium in Tokyo, Gallo remarked how honored he was to be in Japan, "where so much of the important ground-breaking research in this field has been conducted." In the scientific literature, where it counted, it seemed to pain Gallo to give the Japanese any credit at all. In one article recapping the discovery of HTLV, Gallo accorded the Japanese two footnotes out of eighty-five. When a colleague asked Gallo for a complete list of published papers on HTLV, he got back the titles of Gallo's own publications and the caveat that "much of the additional work, most of it from Japan, is really not terribly relevant."

When *Science* returned a Gallo manuscript on HTLV with the suggestion that Gallo might include some reference to "other workers in the field," Gallo replied that he was "astonished by this request in view of the history of the field, the novelty of this work, and the incredible failure of the most obvious other laboratory in Japan to reference anything." On a few occasions Gallo went so far as to declare that his lab, not Hinuma's, had forged the causal link between HTLV and Adult T-Cell Leukemia, including the first detection of HTLV antibodies in healthy Kyushu residents.o Only later, Gallo said, were "subsequent results obtained by investigators in several laboratories in Japan."

As an editor of the definitive textbook on RNA viruses Robin Weiss tried to present a more balanced view, noting that HTLV had first been observed by Elizabeth van der Loo, and that the cell line in which HTLV was found had been established by Adi Gazdar. Weiss also accorded Hinuma the credit for linking Adult T-Cell Leukemia with HTLV. "Hinuma did some very nice work, once the penny dropped," Weiss said. "I think he deserves real credit for that. The Japanese work, never mind who deserves most of the credit within Japan, was driven by studying the disease and the curious pattern of the disease. Gallo's work was driven by coming across this virus and being unaware of Japanese ATL."

Gallo didn't think the lack of awareness was his fault. "The Japanese epidemiologists never knew there was an increase in leukemia in the southern part of Japan," Gallo said years later. "They missed it totally. If I lived in Japan, I would have discovered HTLV in the early seventies. It wouldn't have taken until the end of the seventies."

HTLV was interesting to virologists, but its discovery hadn't done much to advance the National Cancer Institute's primary mission, the treatment of cancer. Indeed, the link between HTLV and cancer was tenuous at best. Not only was Adult T-Cell Leukemia among the rarest of cancers, hardly seen in the Western Hemisphere, of every 200 Japanese infected with HTLV only one ever actually got Adult T-Cell Leukemia.

Pushing such details aside, the NCI ranked the discovery of HTLV as "one of the most exciting stories of 20th Century biology," which put it on roughly the same plane as antibiotics and the double helix. When the NIH nominated Gallo for the National Science Medal, it lauded him not only for the "discovery and isolation of the first human retrovirus," but for "obtaining evidence

that this virus, HTLV, is specifically associated with Adult T-cell Leukemia." When they were mentioned at all, the Japanese were viewed as "adding weight to Gallo's new evidence."

The Lasker Prize is American medicine's highest honor, often described as the "American Nobel," although much of its prestige has resulted from the careful selection of recipients who are only a few years away from winning the Nobel itself. When the Lasker jury met to choose the winners for 1982, there was general agreement that the list should be headed by Mike Bishop and his University of California colleague Harold Varmus, whose work with the Rous Sarcoma Virus had changed the course of cancer research.

Vince DeVita was among that year's Lasker jurors, and DeVita didn't see how the jury could give the prize to Bishop and Varmus without including the discoverer of the first human cancer virus. "Gallo was tagged on at the last minute," Robin Weiss said. "They said, 'Oh, let's have the human as well.'" Gallo had missed the plane that carried Temin and Baltimore to Stockholm five years before. But as the recipient of the Lasker Prize for the "revolutionary discovery of the first retrovirus known to be associated with a human malignancy," he might make the trip with Bishop and Varmus.

Not included in the Lasker citation was Frank Ruscetti, whose run-ins with Gallo had put him on the endangered list. "You don't actually get fired at NIH," explained Ruscetti's technician, Andrea Woods. "You lose your space and lose your technical help. When blood samples come in you aren't on the list to get them, you aren't invited to staff meetings. You basically dry up and blow away." Ted Breitman, Gallo's old boss, remembered a Saturday afternoon in the lab when "Gallo came back from Europe, and he was seething. Someone had approached him and asked him if he was 'still collaborating with Ruscetti.' I don't know whether it was coincidence or not, but Frank was out not long after that."

Bernie Poiesz, his cancer institute fellowship having come to an end, had departed Bethesda to set up a small research lab at the VA hospital in Syracuse, taking with him the rare satisfaction of having made a significant biological discovery. Poiesz needed more than memories to fund his research, but after arriving in Syracuse unsettling things began to happen. Applying for a grant to continue his work with HTLV, Poiesz asked Gallo for a letter of recommendation. What he got in return was far from an enthusiastic endorsement. "He basically wanted to make sure that

he was the major player as the theme of HTLV worked its way out into the world," Poiesz said. "I think his competitive nature was such that, for some reason, there was a false sense of concern that I might do well with it."

Poiesz thought that calling Gallo the discoverer of HTLV was "like saying that Queen Isabella discovered America after Columbus came home and told her about it. I'm the discoverer of HTLV," Poiesz said, "working in Bob Gallo's laboratory. The moment of discovery was mine."

"I think I've forgotten more than Poiesz and Ruscetti ever knew," Gallo says. "Times ten."

sci fi movies list â¤ Science Fiction full movies. KissTube â¤. KissTube â¤.Â Star Odyssey âœªFREE FULL MOVIEâœª Science Fiction Alien Invasion. nousheen iftikhar. nousheen iftikhar. Science Fiction Movies for 2021: Most Popular Sci-Fi Feature Films Released in 2021. Please note: This list excludes animated and superhero movies. Refine See titles to watch instantly, titles you haven't rated, etc.Â Several women enter a science fiction author's life over the course of a few years, after the author has lost the woman he considers his one true love. Director: Kar-Wai Wong | Stars: Tony Chiu-Wai Leung, Ziyi Zhang, Faye Wong, Gong Li. Votes: 50 933 | Gross: $1.44M. The literary genre of science fiction is diverse, and its exact definition remains a contested question among both scholars and devotees. This lack of consensus is reflected in debates about the genre's history, particularly over determining its exact origins. There are two broad camps of thought, one that identifies the genre's roots in early fantastical works such as the Sumerian Epic of Gilgamesh (earliest Sumerian text versions c. 2150â€“2000 BCE). A second approach argues that science fiction only Definition & Examples. When & How to Use Science Fiction. Quiz. I. What is Science Fiction? Science fiction, often called â€œsci-fi,â€ is a genre of fiction literature whose content is imaginative, but based in science. It relies heavily on scientific facts, theories, and principles as support for its settings, characters, themes, and plot-lines, which is what makes it different from fantasy. So, while the storylines and elements of science fiction stories are imaginary, they are usually possible according to scienceâ€”or at least plausible. Daily updates on science fiction and fantasy books, movies, comics, television shows and graphic novels.Â Ben Bova, revered in the science fiction community for his work as an author, an editor, and a scientist, passed away this pastâ€¦ Comic Books. Movies.