placement assistance for each unemployed member who seeks help (most of the money goes for an ACS job clearinghouse and free ads in *Chemical and Engineering News*); and the society has formed a committee on economic status to seek out opinion on the most effective means for using the society's multitude of expensive manpower and salary surveys. These and similar moves run along the lines of programs advanced by Nixon and his supporters but are considerably less aggressive than the professionalists' proposals. Nevertheless, Nixon's success as a gadfly candidate has earned him the reputation, in the words of one ACS staffer, as the "George Wallace of the American Chemical Society."

Kindlier acquaintances called him an "unquenchable idealist," and Alan Nixon is that, if nothing else. Although he is having a tangible effect on the policies of organized chemistry—to the extent that chemistry is organized—any drastic transformation of the ACS is about as remotely probable a feat as radicalizing the National Academy of Sciences.

The entrenched leadership and the paid executives of the society see themselves as severely limited in their role as advocates for chemists—partly by the necessity of preserving the society's pristine image as a scientific body and partly by the more mundane necessity of preserving its tax status as a purely educational institution. And right now the tax status is of utmost importance: Even though dues were increased 2 years ago, the ACS is facing the prospect of a worrisome deficit next year. This year its publications—the primary source of income—barely broke even, and the society's main news publication, *Chemical and Engineering News*, is in deep financial trouble. Frederick T. Wall, the executive director of the ACS, told the society's governing council last week that *C&EN* will probably lose $650,000 this year, despite a 30 percent cut in staff and a 20 percent reduction in content. Wall said that the loss was due to severely reduced advertising. To offset its deficit next year, the society plans to raise the nonmember subscription rates of its journals by 50 percent.

Such stringencies augur poorly for any aggressive new campaigns by the society to shore up the sagging economic status of the chemical profession—even if the society were so inclined.

Ironically though, it may well be that those who stand to gain the most from new professional activism on the part of the ACS—the recent graduates—really care the least.

Otis Rothenberger, an articulate young chemist who received his doctorate from the University of Delaware last June, took part in two symposiums on employment arranged by the society, and he told them both that he and a lot of other young Ph.D.'s just aren't interested in the affairs of large scientific organizations. Rothenberger, who said he spent a year before and after graduation looking for a job, told one manpower panel:

"I respect the ACS as a publishing organization, but as a 'people' organization I don't care if it lives or dies.

"The society doesn't owe me or anyone else a job," he said. "I think we are just going to have to accept the fact that some of us aren't going to be able to play chemistry as we'd hoped."

—ROBERT GILLETTE

Race for Human Cancer Virus: Odds against Houston Team Lengthen

Immunology is part science, part black art, and contributions from the latter source underlie the controversy that now swirls round a promising claim to have discovered a human cancer virus. Early in July, scientists at the M.D. Anderson Hospital and Tumor Institute in Houston announced they had found a virus in a culture of cells derived from a cancer patient. Just as other laboratories were gearing up to study the role of the Houston virus in causing human cancer, a research team based at the National Cancer Institute undermined their hopes with the suggestion that the virus was simply a contaminant of the cell culture, since it appeared to be of mouse, not human, origin.

The argument about the virus's origin, which is still far from closed, hinges on the interpretation of advanced immunological techniques that are being stretched to the limits of their range. The development of the controversy also reflects the pressure of the present political maneuverings to legislate a cure for cancer, under which the already brisk pace of competition in molecular biology has been accelerated to near breakneck speed.

Although Peyton Rous first showed some 60 years ago that a kind of cancer in chickens is caused by viruses, and many other animal cancers have since been proved to be viral in nature, the only type of uncontrolled growth in humans that has so far been nailed down to a virus is the common wart. Two categories of virus, herpes-type viruses and C-type particles, have been found in association with a number of human cancers—last month brought news of the discovery of herpes-type viruses in the cells of patients with Hodgkin's disease. But chiefly for lack of plentiful supplies of the virus, the association has never been raised to causal status. This is why considerable excitement attended the news that the cancer cell line established at Houston was producing virus in quantities sufficient to interest virologists.

The Houston team is led by Elisabeth S. Priori and her former supervisor Leon Dmochowski, both of whom have devoted many years work to the search for human cancer viruses. Their cell line—designated ESP-1 after Priori's initials—was established from the tissue of a 5-year-old child suffering from the type of cancer known as Burkitt's lymphoma. (The child died, in fact, from chicken pox in June 1970.) C-type particles began to appear in the ESP-1 cells 4 months after the culture had been established.

The Houston team delayed announcement of their finding in order to check that the virus was a genuine component of the ESP-1 cells and not a

contaminant from outside—elsewhere in the laboratory a culture of human embryo cells infected with a mouse leukemia virus was being maintained. In addition to their own tests, the Houston team sent a sample of ESP-1 cells for analysis by Lloyd J. Old at the Sloan-Kettering Institute in New York. It was Old who pioneered the technique of distinguishing immunologically between the family of C-type viruses that cause leukemia in the mouse, rat, cat, and other animals.

Old and his colleagues informed the Houston group that they had been unable to detect in the ESP-1 cells the characteristic antigens of any of the known animal leukemia viruses, including that of mouse. On the strength of this finding, Priori and Dmochowski were confident that the virus in the ESP-1 cells was of human origin and not a contaminant. Their initial paper, published on 14 July,* does not claim or even explicitly suggest a human origin for the virus; but the group's hopes were made more explicit in a press release issued on 2 July. (The press release, it has been observed, broke the 14 July embargo date of the paper's publication in *Nature* and thus happened to precede, and be mentioned in, the 7 July debate in the Senate on the proposal to set up an independent cancer agency, from which all cancer institutes stand to benefit in varying degrees. But the press office at the M. D. Anderson Institute explains that the press release was brought forward because of a leak that had appeared in the 28 June issue of *Newsweek*.) The press release stated that the virus was of human origin, not a contaminant, and quoted the head of the M. D. Anderson Institute, R. Lee Clark, as saying that the research, coupled with H. Temin's discovery of the reverse transcriptase enzyme† might hasten the day when the role of viruses in human cancer is defined.

Optimism about the virus was more strongly expressed in a second article, published on 4 August, the title of which described the ESP-1 agent as a "Type C virus of human origin."‡ The article, written by R. C. Gallo and P. S. Sarin of the National Cancer Institute together with members of the Houston team, reported that the ESP-1 virus has the characteristic reverse transcriptase enzyme that has been discovered in every RNA tumor virus so far investigated.

The case for the ESP-1 virus being of human origin rested largely on the negative finding, made in Old's laboratory, that the virus was not that of mouse. The case was therefore weakened quite severely by news that the ESP-1 agent did after all appear to contain the antigens typical of mouse leukemia virus. This was the conclusion announced early last month by Raymond V. Gilden, of Flow Laboratories in Rockville, Maryland, and by Wade P. Parks, Robert J. Huebner, and George J. Todaro of the National Cancer Institute.§ Despite this blow, Priori, Dmochowski, and their colleagues are fighting a skillful rearguard action which, in the opinion of neutral observers, may yet win them the day. The Houston team argues that the ESP-1 virus differs sufficiently from mouse leukemia viruses in its immunology to suggest that it is a human virus, or at least unique.

Doubts about Mouse Antigen

The three laboratories that are party to the debate, those of Old, Gilden, and Parks, are among the few in the country that possess the necessary reagents for studying the immunology of mammalian C-type particles. It was Old and his colleagues who discovered in 1966 that certain antigens are common to all mouse leukemia viruses. The antigens were named gs-1, gs-2, and gs-3 (for group specific), after the number of precipitation lines they formed with their antibodies on agar plates. Antigen gs-2 has since dropped from history. The gs-1 antigen has been shown to be species specific, since the leukemia viruses of mouse, rat, cat, and hamster each have their own, unique gs-1 antigen. The gs-3 antigen has turned out to be interspecific, in that the leukemia viruses of all these species share the same gs-3 antigen. Early this year Gilden, Huebner, and S. Oroszlan demonstrated that the gs-1 and gs-3 antigens form part of the same molecule, a protein of about 30,000 molecular weight (depending on the species), which lies on the interior surface of the virus.

The antigens are difficult to work with because, in order to raise antibodies of the necessary specificity, the protein containing the antigen must be highly purified before it is injected into the animal. A technique for purifying the protein by isoelectric focusing has been exploited by Old and Gilden. Using this technique, both Gilden and Parks in their separate laboratories showed that antibodies raised against the gs-1 antigen of mouse leukemia virus reacted with an antigen from the ESP-1 virus.

There are several possible interpretations of this result. The most likely, by some margin, is that the ESP-1 virus is a mouse virus. A second interpretation, noted by Gilden and colleagues in their paper, is that the gs-1 antigens of mouse and man are immunologically identical. Among the five or six species so far studied, gs-1 antigens are not known to cross species barriers, but it is certainly possible that they could do so. (One straw in the wind blowing this way is the unpublished finding by L. R. Sibal of the National Cancer Institute that both normal and breast cancer patients may possess circulating antibody that reacts with the mammary tumor agent of mice.)

A third explanation is that the reaction is nonspecific in that the antibodies against mouse gs-1 may be reacting not with the gs-1 determinant of the ESP-1 virus but with some other determinant that the two gs proteins hold in common.

Why did the Gilden and Parks teams come up with a different result from that of Old and his colleagues? It seems that the concentration of virus in the ESP-1 samples first made available to Old was too low to show up the mouse antigen found by Gilden and Parks, although high enough to detect mouse leukemia virus in its usual form. Old (who could not be reached for comment this week) is understood to have repeated the determination and to have confirmed Gilden's finding of a presumptively mouse gs-1 antigen in the ESP-1 cells. But according to Gayla Geering, a member of Old's team, the ESP-1 virus behaves sufficiently differently from ordinary mouse leukemia virus for substantial doubt still to exist. "We are still very open about it," Geering says. "We would be willing to believe that it is mouse but it is not yet showing all the components of mouse behavior."

To prove incontrovertibly that ESP-1 is mouse in nature requires performance of the reciprocal experiment of that already done by Gilden and by Old. The test involves raising antibodies to the gs-1 antigen of the ESP-1 virus and seeing whether they react with the gs-1 antigen of mouse leukemia virus.

* E. S. Priori, L. Dmochowski, B. Myers, J. R. Wilbur, *Nature New Biol.* **232**, 62 (1971).
† See B. J. Culliton, *Science* **172**, 926 (1971).
‡ R. C. Gallo, P. S. Sarin, P. T. Allen, W. A. Newton, E. S. Priori, J. M. Bowen, L. Dmochowski, *Nature New Biol.* **232**, 140 (1971).
§ R. V. Gilden, W. P. Parks, R. J. Huebner, G. J. Todaro, *Nature* **233**, 102 (1971).

NEWS & NOTES

• **ROGERS REVIEWS CANCER PLAN**: It appears now that the Administration-backed bill calling for an independent Conquest of Cancer Agency, which swept the Senate 79–1, is in for a rougher ride through Congress than had been anticipated. Representative Paul Rogers (D–Fla.), chairman of the House health subcommittee, says his subcommittee has started 3 weeks of hearings on the bill, during which a wide range of scientists who oppose the Senate-passed measure will testify. Rogers and five members of his subcommittee have introduced an alternate bill that would strengthen cancer research within NIH's National Cancer Institute. Those who oppose the separate authority, which would be nominally within NIH but would report directly to the President, believe that such an arrangement would fragment NIH research efforts and merely create a new bureaucracy (or "monstrosity," as Rogers termed it). Many legislators have supported the Senate measure for fear that opposing it would brand them as "pro-cancer"; further airing of the matter may permit second thoughts.

• **KNOWLES TO ROCKEFELLER FOUNDATION**: John H. Knowles, whose politics cost him the nation's top health post 2 years ago, has been named president of the Rockefeller Foundation. He will succeed J. George Harrar, an early leader in the "green revolution," who will retire in December. Knowles, an outspoken liberal on public issues as well as medicine, has been general director of Massachusetts General Hospital since 1962. Knowles was nominated as HEW's assistant secretary for health and scientific affairs in 1969, but pressure from conservative Republicans and the American Medical Association caused the nomination to be dropped. He will assume leadership of the nation's second-largest foundation next July.

• **OFF-SEASON AT WOODS HOLE**: The Marine Biological Laboratory at Woods Hole has announced the completion of a dormitory and dining complex that will enable it to accommodate conferences of about 200 persons, as well as winter visitors who would like to make use of the library facilities. For details, scientists may write Homer P. Smith, General Manager, MBL, Woods Hole, Massachusetts 02543.

Both Gilden and Old have this experiment under way. Old's laboratory is also trying to find out whether the surface antigens of the ESP-1 virus resemble those of mouse leukemia virus.

The new data obtained by Old, together with other new results, were presented on 31 August at a round table conference of the scientists involved in the debate. One participant, Wade P. Parks, believes that the advocates of a mouse origin for the virus carried the day. But Leon Dmochowski, speaking last week from Padua (where many of the principals in the ESP-1 debate are attending a symposium on leukemia), noted that the outcome of the round table conference was a decision to continue the work on the ESP-1 virus. Gilden's results were interesting, Dmochowski says, but in order to demonstrate the antibody-antigen reaction the Gilden and Parks teams had to resort to "drastic methods" and even then were able to find, by using extremely high concentrations of virus, only a trace of gs-1 antigen. "In our opinion such a reaction may be entirely nonspecific," Dmochowski says.

He and Priori have accumulated several new results suggesting that ESP-1 virus differs importantly from the mouse leukemia virus. For example, serums from tumor patients give an immunological reaction with ESP-1 cells, but not with human cells infected with mouse leukemia virus. Also antiserums against mouse gs-1 react with human cells infected with mouse virus but fails to coat ESP-1 cells.

Another result suggesting that ESP-1 virus differs from mouse leukemia virus is the finding by Gallo and Sarin, announced last week at the Padua conference, that the reverse transcriptase enzyme of ESP-1 virus differs from that of the mouse leukemia viruses. Gallo's evidence is that antibody raised against the latter class of enzymes fails to inactivate the enzyme of ESP-1 virus.

The debate on the origin of ESP-1 virus thus remains unresolved; it is still on the cards that the Houston group has uncovered a human virus, or at least a new kind of C-type particle, although for the time being the most likely hypothesis must be that of contamination. An obvious issue is why the Houston group did not delay the announcement of their results until the issue raised by Gilden had been clarified. "That is the $64,000 question," says Dmochowski, who feels that the Gilden group was unreasonably reluctant to show him their results. (The results seem to have become available some time between the publication of the two papers by the Houston group.) Having studied the ESP-1 cell line for more than a year and with other laboratories already at work on the ESP-1 virus, the Houston group was doubtless anxious to establish its priority, and after the negative results from Old's laboratory the reasons for delay must have seemed less compelling than the need to announce a finding of potentially great importance to cancer workers and cancer sufferers. Nevertheless, the initial hopes raised by the announcement are now not so high as they were. Will science be harmed in the public eye by such oscillations of confidence? "It ought to be, if Congress is watching," says Wallace P. Rowe, virology chief at the National Institute of Allergy and Infectious Diseases; "The field is moving at such a fast clip that everyone is forced into this quick draw type of publication." But other scientists, such as Sol Spiegelman of the Columbia University Institute of Cancer Research, believe that little if any harm has been done. Spiegelman, who considers the ESP-1 issue still unresolved, says that rapid publication is justified in cancer research because of the urgency of finding a causal agent.

Dmochowski, for his part, is unbowed in adversity. In 1956 he detected C-type particles in a leukemia patient but was unable to repeat the observation for another 6 years. "That was a long time to stand up like a sore thumb," Dmochowski says. "Maybe history is now repeating itself but I'm not yet sure."—NICHOLAS WADE

APPOINTMENTS

Albert W. Cook, associate professor of neurosurgery, Downstate Medical Center, State University of New York, to chairman, neurosurgery department at the center. **Thomas G. Baffes**, clinical professor of surgery, University of Health Sciences/The Chicago Medical School, to chairman, surgery department at the institutions. . . . **Martin R. Baron**, professor of psychology, Kent State University, to chairman, psychology department, University of Louisville. . . . **William V. D'Antonio**, chairman, sociology department, University of Notre Dame, to chairman, sociology department, University of Connecticut.