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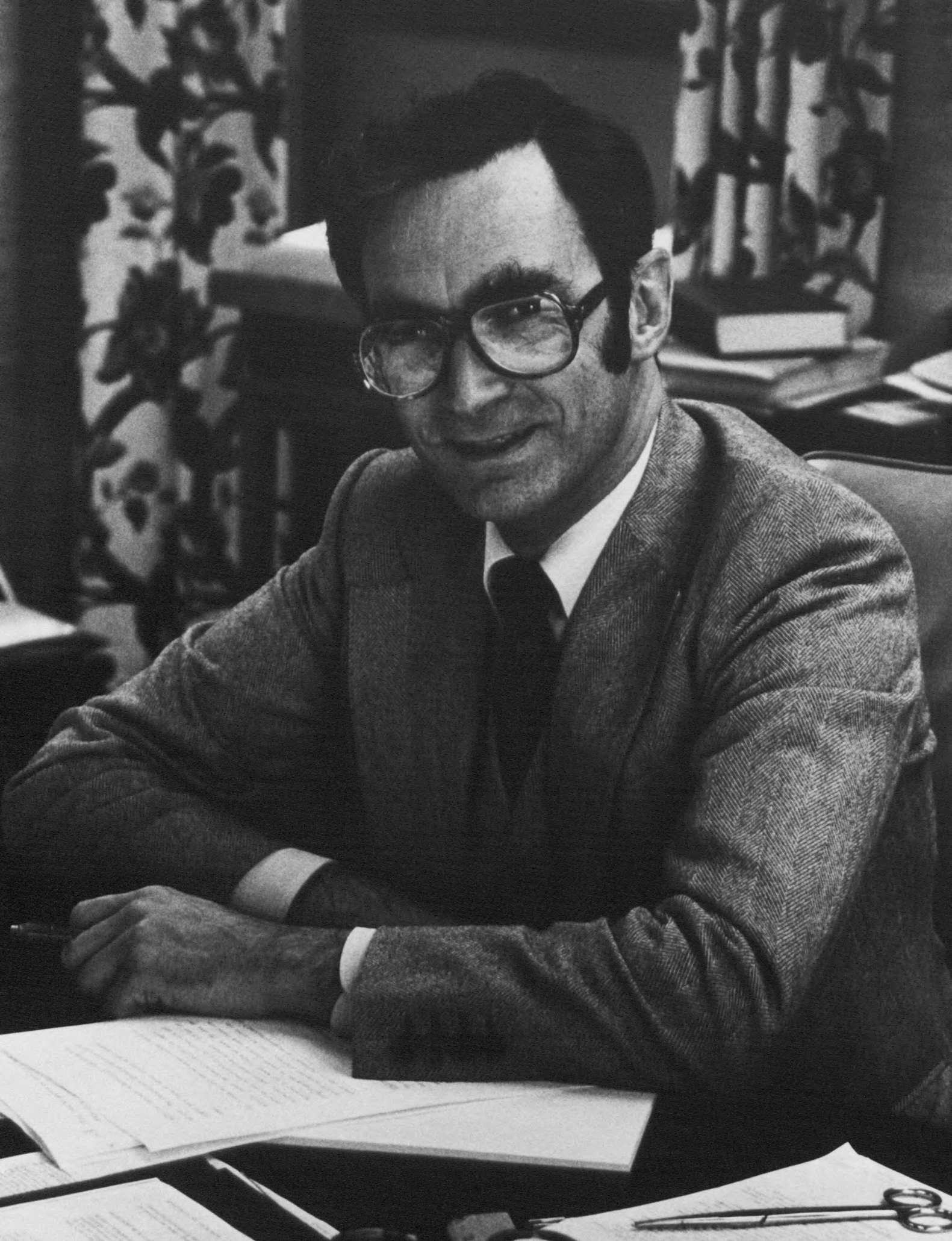


A Message from the Director

The Baker Institute celebrated its thirtieth anniversary by setting new records in productivity and by completing an ambitious program for improving facilities. The record level of sponsored research, and the many published reports by our staff in 1981, attest to the Institute's continuing vitality and commitment to excellence.

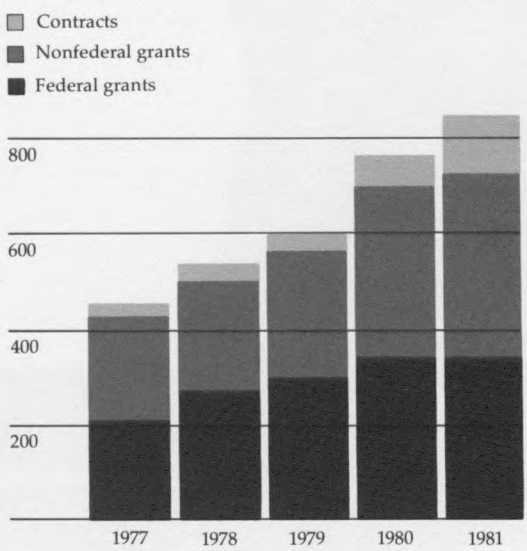
The accompanying figure indicates that much of the Institute's operating income derives from research grants from the federal government. Income from this source has increased steadily in recent years and is now more than double the level of just five years ago. The steady increase in government support is impressive, because there has been only a modest increase in federal resources, and our staff scientists must compete for these funds with biomedical scientists in institutions throughout the country.

The results of research undertaken at the Institute were reported in scientific journals and at professional meetings in this country and abroad. Reports on canine nutrition, hip dysplasia, and parvoviral disease were also distributed to kennel clubs, veterinarians, and friends of the Institute. Copies of these reports can be obtained by writing to the Institute or telephoning Mrs. Florence Huth (607/277-3044).

The Institute was honored this year when three of its members received national awards. Professor Leland Carmichael received the Gaines Award for his pioneering work on canine parvovirus. The Ralston Purina Award was shared by Professors Carmichael, Max Appel, and Roy Pollock. Purina Awards are made each year in recognition of outstanding achievements in small-animal medicine.

Significant advances in our research were made in 1981. These achievements are described elsewhere in this report. But one project merits special attention, because it illustrates the spirit of innovation that has sustained the Baker Institute for three decades. With aid from the Geraldine R. Dodge Foundation, a cell hybridization facility was created. Here Professor Antczak and his associates are generating continuously growing cell lines that secrete antibodies of a single molecular species. Such "monoclonal antibodies" have an exceptional capacity to discriminate infectious agents, cellular antigens, and other biologically important molecules. Not only are monoclonal antibodies powerful research tools, but they are also useful in diagnosing disease.

Grant and Contract Income
(in Thousands of Dollars)



The Institute's cell hybridization facility is the first of its kind in veterinary medicine and is the only laboratory on the Cornell campus dedicated exclusively to research using cell hybridization methods. University scientists who require monoclonal antibodies in their own research are pursuing collaborative projects with the Institute's staff. We welcome this interaction, because it broadens our perspective, creates new opportunities for the study of disease, and enhances the intellectual environment of the Baker Institute.

As we look to the future, we expect that the competition for resources will increase; yet we are confident that we will prosper. We have an organization of dedicated men and women and a growing constituency of concerned benefactors, and we are served by an advisory council that is resolute in its commitment to excellence. This spirit is our most cherished resource and will serve us well in the years ahead.

Douglas D. McGregor
Director

Neil H. McLain, the Institute's administrative manager, responded to many inquiries about canine parvovirus. Nancy D. Combs's responsibilities include purchasing, grants management, and inventory control.

Staff of the Baker Institute

Administration

Douglas D. McGregor, director: B.A., M.D., University of Western Ontario; D.Phil., Oxford University
Neil H. McLain, administrative manager: A.B., Cornell
Nancy D. Combs, administrative aide
Ann W. Signore, administrative aide: Cornell
Ok Chon Allison, secretary: A.S., Canal Zone College; A.A., Dong-a University
Kim T. Arcangeli, secretary
Florence C. Huth, administrative aide
Douglas S. Robson, consultant in statistics: B.S., M.A., Iowa State University; Ph.D., Cornell

Laboratories

Cornell Research Laboratory for Diseases of Dogs

Leland E. Carmichael, John M. Olin Professor of Virology: A.B., D.V.M., University of California; Ph.D., Cornell
Susan P. Montgomery, graduate research assistant: B.A., Harvard
Colin R. Parrish, graduate research assistant: B.Sc., Massey University
Jean C. Joubert, research technician
Priscilla H. Dunham, laboratory technician

Giralda Laboratory for Canine Infectious Diseases

Roy V. H. Pollock, assistant professor of microbiology: B.A., Williams College; D.V.M., Ph.D., Cornell

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George Lust, associate professor of biochemistry: B.S., University of Massachusetts; Ph.D., Cornell
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Hadley C. Stephenson Laboratory for Study of Canine Diseases

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Donnelley Laboratory of Immunochemistry

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Oswald R. Jones Laboratory of Immunology

Robin G. Bell, assistant professor of immunology: B.Sc., Australian National University; Ph.D., John Curtin School of Medical Research
Ching-hua Wang, graduate research assistant: M.S., Peking Medical School
Lincoln S. Adams, research technician: B.S., Hobart College; AALAS accreditation
Ralph W. Ogden, laboratory technician: B.S., University of Maine

Immunogenetics Laboratory

Douglas F. Antczak, assistant professor of immunology: B.A., Cornell; V.M.D., University of Pennsylvania; Ph.D., Cambridge University
Mark J. Newman, research associate: B.Sc., M.Sc., Ohio State University; Ph.D., Australian National University
Kim H. Beegle, laboratory technician: B.S., Cornell
Jane M. Miller, laboratory technician: B.S., Cornell

Richard King Mellon Laboratory for Electron Microscopy

Helen A. Greisen, research associate: B.S., M.S., Ph.D., Cornell

Colgate Division for Tissue Culture

Kim H. Beegle, laboratory technician: B.S., Cornell

Glassware Department

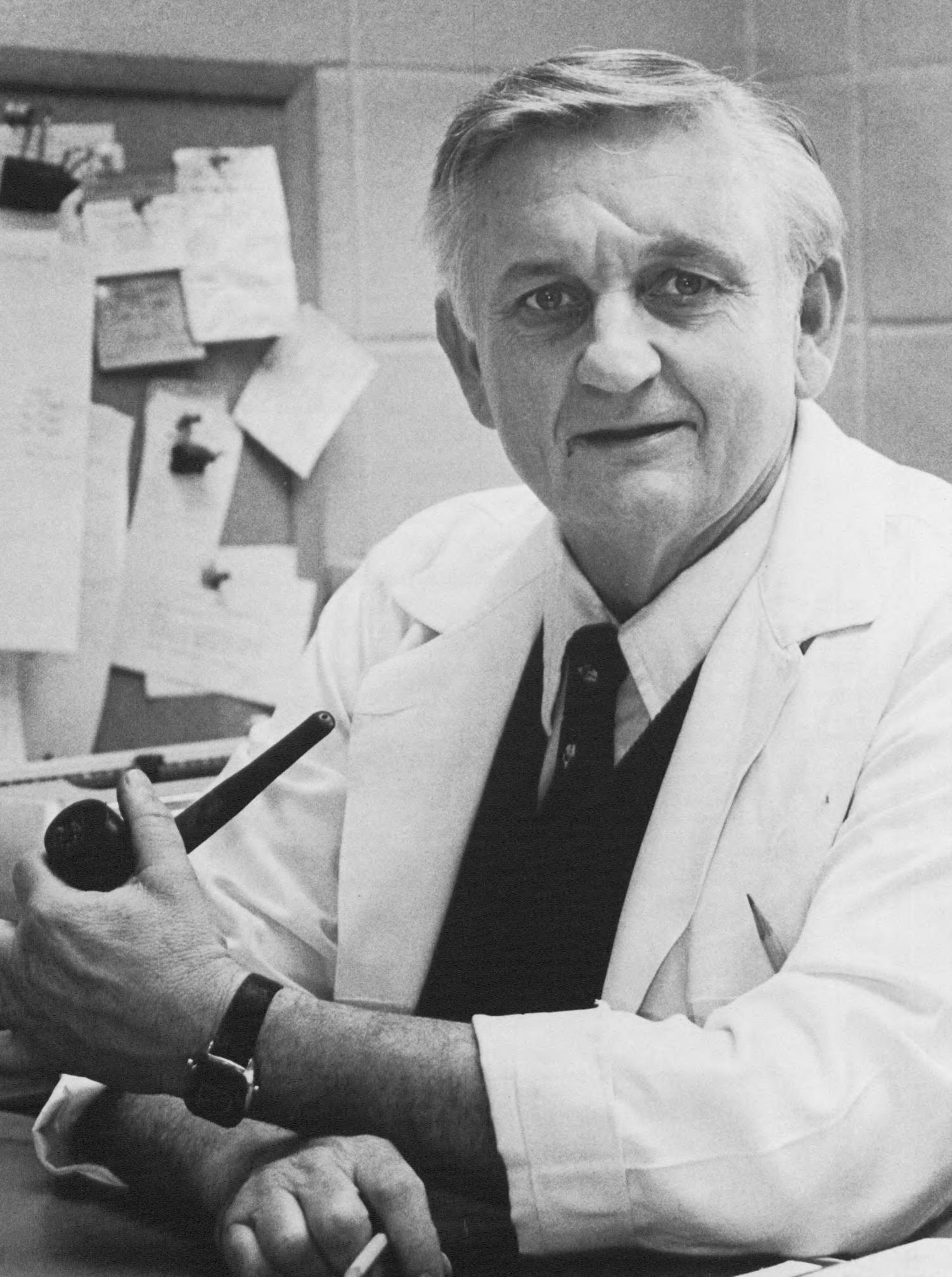
Jeannette R. Kniffen, laboratory attendant
Elizabeth C. Wheeler, laboratory attendant

Animal Care

Charles B. Bailor, animal technician
Roy L. Barriere, animal technician: AALAS accreditation
Bernard L. Clark, animal technician
Raymond M. Combs, animal technician
Raymond J. Corey, animal technician: A.A.S., State University of New York Agricultural and Technical College
James Hardy, animal technician: B.S., Cornell
Kurt W. Richau, animal technician: A.A.S., State University of New York Agricultural and Technical College; AALAS accreditation

Maintenance

Edson Wheeler, maintenance supervisor
Arthur D. Howser, maintenance mechanic
Gerald G. Rice, vehicle mechanic
John C. Howe, custodian



The development of veterinary biologicals should be pursued as rigorously as that of vaccines for human use. Our commitment to this principle motivated our research on a modified living homologous (canine) parvovirus vaccine that was developed at the Institute. Safety, without sacrifice of efficacy, has been our foremost concern. The Baker Institute strain of canine parvovirus (CPV) fulfilled this requirement in laboratory and field studies. After about eighty serial passages in canine cell cultures, the vaccinal CPV strain did not cause illness in pups but retained its capacity to immunize at low viral doses.

The vaccinal strain is characterized by a large-plaque marker in cell culture. This characteristic permits identification of the vaccinal strain and distinguishes it from the virulent small-plaque virus. The vaccinal strain of CPV is shed in the feces of vaccinated dogs for a few days, but the virus does not revert to virulence and retains its large-plaque characteristics even after five serial "back passages" in dogs.

This vaccinal strain of CPV induced the greatest and earliest antibody response of any parvovirus vaccine yet studied. The responses were uniformly vigorous in susceptible seronegative pups. Moreover, the vaccine induced long-lived immunity, lasting at least a year and a half.

Only after these studies were completed was the vaccinal strain released to selected producers of biologics for development of commercial vaccines.

The immune response of dogs to the modified living CPV vaccine was inhibited by preexisting maternal antibody and by antibodies engendered by other parvovirus vaccines. Its behavior in this regard was similar to that of killed vaccines and the vaccinal strain of feline panleukopenia virus (FPV), as described in the report on the Giralda Laboratory. Some dogs with low levels of serum antibody (titers of 1:10 to 1:20) were successfully immunized with the modified living CPV vaccine, whereas dogs with similar titers failed to respond to killed vaccines or to living FPV vaccines. Although the vaccine strain of CPV was superior in this regard, the responses to the modified canine vaccine were inconsistent when the antibody titers of the test subjects were more than 1:10. Field studies confirmed our laboratory observations: more pups with low levels of maternal antibody were successfully vaccinated when the modified CPV vaccine was used.

Individuals in Europe and Australia have expressed the concern that continued growth of CPV in noncanine cell cultures might favor the genetic selection of mutants that if shed by vaccinated animals, could involve other species, even human beings! This hypothesis has led to highly speculative claims, some of which have been prompted by commercial interests. Such statements have no basis in fact. If such an event occurred, it would be unique in nature.

Recent studies have revealed that the canine cell–passaged vaccinal strain of CPV becomes highly attenuated when passaged serially in feline cells. After passage in feline cells, the virus replicates preferentially in feline cells, and its capacity to immunize dogs is greatly diminished. This finding was surprising but not unexpected in view of similar experience with other viruses. The results underscore the need for periodic evaluation of vaccinal viruses manipulated in this way.

Another important area of research is being addressed by Colin Parrish. It concerns the biological relationships between CPV, FPV, and mink enteritis virus (MEV). The

antigenic relationships of these three viruses are being examined using both conventional antisera and monoclonal antibodies (mAbs). The latter were prepared by a form of genetic engineering discussed elsewhere in this report. Several serologic methods demonstrated that six out of sixteen mAbs reacted only with CPV; the remainder reacted equally well with CPV, FPV, and MEV. Using the agar gel diffusion test, we demonstrated antigenic differences between the viruses. The discovery of antigenic differences between two strains of FPV was an additional and unexpected finding.

The goal of our continuing research is to determine the precise composition of CPV, FPV, and MEV, and to identify the viral proteins that react with the various mAbs at our disposal. Another objective is to devise a test for the diagnosis of CPV infection that is both accurate and sensitive. The insights gained from these studies may also shed light on the origin of CPV, which emerged suddenly as a new pathogen of dogs just four years ago.

Our work with canine brucellosis continued. It was shown that the cultural conditions under which *Brucella canis* is propagated greatly influence the colonial characteristics of the organism. Physicochemical differences were also demonstrated between the surface antigens of *B. canis* and other "rough" brucella. The practical outcome of these studies is that we now know how to prepare stable *B. canis* antigens for serodiagnostic tests.

A study of eight dogs that encompassed the period of initial infection at four days of age through recovery at five and a half years was recently completed. Serologic profiles were constructed from a panel of sera obtained at weekly intervals throughout the observation period. Seven different serological procedures were used, and the results were related to bacteriological findings in the same animals. A potentially important new finding was the discovery that an immunodiffusion test using protein antigens obtained from cytoplasmic extracts of *B. canis* cells ("internal antigens") was a diagnostically reliable method for detecting *B. canis* infection in dogs, particularly when the test was performed with one using cell wall antigens. The internal antigens detected antibodies to *B. canis* shortly after the onset of bacteremia, and for at least six months after bacteria could no longer be isolated from the blood. Other serologic tests gave negative or equivocal results during this period.

The common sites of persistent brucella infection were the spleen and lymph nodes and, in males, the prostate gland and epididymides. The presence of radiographic lesions in three chronically infected dogs provided further evidence for a causal role of *B. canis* in discospondylosis.

Leland E. Carmichael

Giralda Laboratory for Canine Infectious Diseases

Research was concerned with controlling canine parvovirus (CPV) infection. Studies of inactivated vaccines revealed that protection against systemic infection lasts at least six months if the vaccines contain enough virus. Protection against asymptomatic infection of the intestinal tract was shorter, only three or four months. Dogs inoculated with killed vaccines five months earlier were exposed to CPV and became infected, but viral growth was confined to the intestinal tract and its associated lymphatic tissues. Viral growth in these tissues was greatly reduced. While these dogs could have served as a source of infection for other dogs, they did not themselves become ill after challenge with virulent virus. They developed antibody titers similar to those of challenged but nonvaccinated dogs. These observations probably account for the general success of inactivated vaccines in the field.

The increasing prevalence of immune dogs in the population helped reduce even further the number of new CPV cases this year. Nevertheless, infection frequently occurred in eight- to sixteen-week-old puppies. Infections were reported even among repeatedly vaccinated subjects. Maternal antibody was shown to be the principal cause of such vaccine failures. Antibodies are transferred from the dam to her pups through the placenta and colostrum. Since most adult dogs are now immune to CPV as a consequence of inapparent infection or vaccination, most pups receive substantial, but variable, amounts of antibody to CPV. This antibody is protective during the first few weeks of life, but it is slowly degraded, with half the remaining amount lost each nine to ten days.

Although maternal antibody protects pups against infection, it also suppresses the animal's immune response to vaccination. This effect is also observed with distemper and hepatitis. The period of suppression is related to the amount of antibody the pups received from their dam; this is proportional to the antibody titer of the dam. When large amounts are transferred, pups may not respond to immunization until they are sixteen weeks old. Since litters vary in the amount of antibody they receive, and hence in the age at which they can be successfully immunized, they must be repeatedly vaccinated to assure protection. Vaccination should continue at two- to three-week intervals for at least sixteen weeks. Laboratory studies confirmed our suspicion that there is a critical period during which maternal antibody is low enough so that pups can become infected if exposed to CPV but still high enough to impede the immunization process.

Experiments were performed in which litters of pups were divided into two groups, one of which was vaccinated at intervals with inactivated or modified living heterologous (feline panleukopenia) virus and the other of which received oral-nasal challenge inoculations with virulent virus. All pups with antibody titers less than 1:40 became infected, but with few exceptions, vaccinated pups did not develop an active immune response until more than two weeks after antibody titers had declined below 1:10. Infection during this period of unpredictable susceptibility would be especially common where the risk of exposure is great, as in commercial breeding colonies or pet shops. A goal for the coming year is to shorten this period of susceptibility. In recent experiments most pups with antibody levels high enough to block the response to inactivated or modified living heterologous (feline) virus responded one to two weeks earlier to an experimental modified living homologous (canine) parvovirus vaccine.

Roy V. H. Pollock

Nutrition studies concentrated on the special needs of old dogs. Beagles nine to ten years of age fed a diet designed for old dogs were active and healthy and remained metabolically normal during the first year's observations.

A brisk ten-minute daily jog at a rate of three miles per hour and a slope rise of fifteen degrees visibly improved the attitude and spirit of the old dogs. It also improved their ability to utilize energy from their diet. Other physiological and metabolic parameters measured were not significantly altered as a result of exercise.

Analysis of urine, blood chemistry, hematology, enzyme activity, and liver and kidney function failed to reveal differences due to the age of dogs, the diet, or exercise. Thyroid function tests, however, did indicate that old dogs are less responsive than year-old dogs. There was no evidence that aging compromises the capability of dogs to synthesize vitamin C.

Nutrient balance studies supported the earlier observations of metabolic normality. In fact, the old dogs consistently demonstrated a better ability to digest nutrients than year-old dogs, although the recorded difference was not statistically significant. Unlike humans, old dogs digested and retained minerals, particularly calcium and phosphorus, as well as, or better than, young dogs. There was no difference between dogs and bitches. In old dogs the major portion of excreted minerals was found in the feces, although more sodium and potassium was excreted in the urine.

Old and young dogs had the same immune responses to vaccination with canine parvovirus vaccines. However, in vitro cellular immunity responses (T-cell function) were consistently lower for old dogs. T-cell function was measured by recording in vitro lymphocyte proliferation after stimulation by the mitogen concanavalin A. These studies will be expanded in the coming year to determine the significance of the observed suppression and whether it can be corrected by diet.

In summary, old dogs thrived on regularity, both in management and in diet.

Last year we reported new roles for vitamin E, namely in maintaining retinal integrity and healthy skin. These studies have now been expanded to include studies of interactions of vitamin A and E in these phenomena.

Ben E. Sheffy


PICKER
PICKER

John M. Olin Laboratory for the Study of Canine Hip Dysplasia

Our research is seeking answers to the questions of how and why dogs develop hip dysplasia and what can be done to treat and ultimately prevent this crippling disease. The research is proceeding along two lines. First, we are analyzing tissues in the region of the hip joint in an effort to identify factors that favor the expression of hip dysplasia. Second, we are continuing our biochemical studies of the articular cartilage from diseased joints.

Excessive movement of the femoral head within the hip joint socket, which occurs in dogs with hip dysplasia, prevents normal apposition of the bone surfaces. This creates mechanical stresses that may contribute to degeneration of the articular cartilage and stretching of the round ligament that holds the femoral head within the joint. Precisely why this displacement, or subluxation, occurs is unknown. Last year we reported differences in the degree of torsion, or twisting, of the femurs in normal dogs and dogs with hip dysplasia. But careful radiographic measurements in a larger number of subjects failed to substantiate our earlier findings, in which inferior methods were used. We are therefore led to the conclusion that excessive torsion of the femur is not the important factor in hip dysplasia that we had initially suspected. Further measurements of this kind are being made in disease-prone dogs to ascertain whether subtle changes in the shape of the acetabulum might contribute to subluxation of the femoral head.

At the biochemical level we are pursuing our investigation of a metabolic abnormality in the articular cartilage of diseased joints. The abnormality involves collagen, an important constituent of the cartilage matrix. Our investigations so far have disclosed a diminution in the rate of collagen synthesis in foci of cartilage degeneration. The defect results in the depletion of collagen, a change that might be expected to render the tissue more vulnerable to injury. We are now turning our attention to the question of whether collagen is also broken down more rapidly within these areas.

Last year we reported that the metabolic abnormality in the articular cartilage of diseased hip joints can also be demonstrated in cartilage taken from the shoulder, knee, and elbow joints of dysplastic dogs. It is possible that hip dysplasia is only the most conspicuous manifestation of a disease affecting many joints. We will vigorously pursue this lead, because it opens new avenues to the investigation of hip dysplasia and the arthritis associated with the disease.

George Lust

Canine distemper virus (CDV) usually causes an acute infection; most dogs either die or recover. Occasionally, however, dogs become persistently infected and develop chronic brain disease. A similar situation occurs in human beings, where infection with measles virus sometimes causes subacute sclerosing panencephalitis.

We are studying the mechanisms by which CDV causes a persistent infection in dogs. To determine whether the virus mutates or in some other way becomes adapted in lymphocytes, Mr. Friedlander has been passaging virulent CDV in lymphoblastoid cells. Dogs inoculated with the infected cells developed persistent infection, whereas the native virus produced acute disease. Also, virulent CDV rapidly lost its disease-producing capacity when passaged serially in canine epithelial cells or fibroblasts rather than in (human) lymphoblastoid cells. We are now trying to determine how CDV variants are selected and why these variants differ in their capacity to infect canine cells.

In collaboration with colleagues in Stockholm, Sweden, we pursued an entirely different approach to the problem of CDV persistence. Can a defect in the animal's cellular or humoral immune capacity influence the pattern of disease? Although only a few experiments have been performed, the results have demonstrated the importance of cell-mediated immunity in resistance to infection. Dogs immunized with inactivated measles virus, inactivated CDV, or protein constituents of CDV showed a specific antibody response to the virus but did not develop cell-mediated immunity. When subsequently challenged with virulent CDV, such immunized dogs developed acute disease. Those that survived the infection showed a vigorous cellular response and were protected from further challenge.

Cell-mediated immunity to CDV seems to be important in the acquired resistance to CDV. However, circulating antibodies may influence the expression of disease. Evidence was obtained by adding CDV antibodies to canine cells in culture. Antibodies of the immunoglobulin G (IgG) class fixed complement and protected macrophages against CDV. When the segment of the IgG molecule that reacts with complement was removed enzymatically, the remaining portion of the molecule retained its capacity to bind CDV. However, the fragmented molecule could no longer protect macrophages against infection. This finding suggests that distemper antibodies of the IgG class bind CDV and that the complexes so formed attach to complement-dependent receptors on the macrophage membrane. Attachment at this location seems to be important in directing CDV into the vacuolar system of the macrophage, where the virus is inactivated. When the complement receptor is not engaged—as occurs when the receptor is enzymatically cleaved—CDV penetrates the macrophage in a different way and, by avoiding the vacuolar system, grows as it does in epithelial cells. Additional experiments are planned to explain this phenomenon and to ascertain whether the pattern of disease is influenced by the properties of individual CDV isolates and the type of immune responses the virus induces.

A modified living canine parvovirus (CPV) vaccine was evaluated in a large commercial kennel. Use of inactivated vaccines and modified living feline panleukopenia virus over a two-year period had failed to control CPV enteritis. The

mortality rate in eight- to twelve-week-old pups was about 7 percent despite repeated vaccinations. Within a month of the initiation of a controlled vaccination program with living CPV, deaths ceased and the number of enteritis cases was greatly reduced. Mortality rates and the frequency of CPV-associated enteritis were unchanged in the control groups that continued to receive inactivated or living heterologous (feline) vaccines. We are now attempting to determine the optimal spacing of vaccinations.

Another study was prompted by reports of CPV-like enteritis outbreaks in raccoons. To determine whether raccoons are susceptible to CPV, seronegative animals were inoculated oral-nasally with virulent CPV. Also, a CPV-like parvovirus isolated from a raccoon with fatal enteritis was inoculated into susceptible pups. The CPV did not infect raccoons, and the raccoon parvovirus did not infect dogs. Preliminary studies suggest that the raccoon virus, like the mink enteritis parvovirus, is more like feline panleukopenia than like CPV in its biological properties.

Max J. G. Appel

Donnelley Laboratory of Immunochemistry

The research undertaken in this laboratory is concerned with immune responses mediated by thymus-derived lymphocytes, or T cells. These account for most of the lymphocytes in the blood, lymph nodes, and spleen and for many lymphocytes in other tissues. While T cells appear to be very similar when viewed under the microscope, we now know that they belong to several families of cells, each of which has its own life history and function. Some T cells operate as "helper cells," promoting the differentiation of bone marrow–derived lymphocytes, or B cells, into antibody-forming cells. Other T cells have an immunoregulatory function, while still others cooperate with macrophages in delayed-type hypersensitivity (DTH) reactions to a variety of microorganisms, foreign cells, and tumors.

Our research is concerned mainly with the role of T cells in DTH and acquired resistance to infection. We are studying these responses in tissue culture and in rats infected with the *Listeria monocytogenes* (LM), a bacterium known for its capacity to induce a T cell–mediated response. Last year we reported that when the T cells of LM-immune rats are stimulated in culture by antigens of this organism, they rapidly acquire the capacity to kill other cells. We have substantiated this observation and demonstrated that a variety of target cells are vulnerable to attack. This finding suggests that these cytolytic T cells are involved in some way in the regulation of the immune response to infection, or possibly in the inflammation that develops at sites of microbial invasion.

This year we concentrated our efforts on defining the antigenic characteristics of LM-dependent cytotoxic lymphocytes. Using monoclonal antibodies that recognize antigens on the surface of membranes of some, but not all, T cells, we showed that LM-dependent cytotoxic T cells have the same antigenic profile as T cells that have suppressor activity in other systems. This observation encourages the notion that LM-dependent cytotoxic T cells have an immunoregulatory function.

We also examined the conditions required for T-cell activation in the *Listeria* system. It was discovered that at least two classes of T cells are required for the activation process. We will extend our observations in this area, for we expect that the results of this investigation will provide new insights into the manner in which T cells cooperate with one another, and with other cells, in the initiation, expression, and control of DTH and cellular resistance to infection.

Melissa C. Woan


RAPI

We investigated the genetic basis for the variation in immunity to intestinal parasites. Mice were used because animals of defined genetic composition were required, and many inbred strains of mice have been developed for studies of this sort. We showed that mice, generally considered weak responders to *Trichinella spiralis,* could respond just as strongly as rats, but this capacity was expressed in only two out of ten inbred strains.

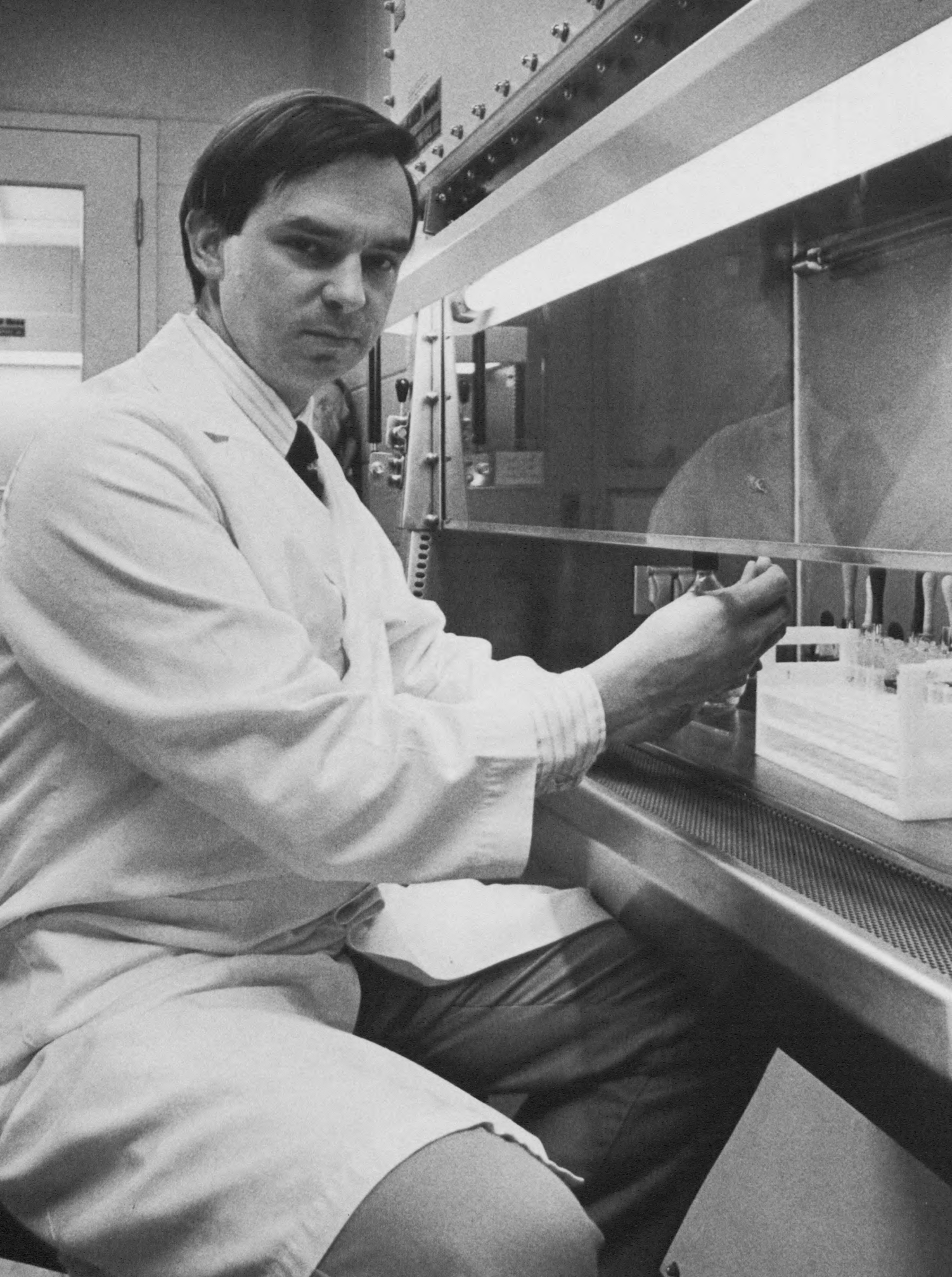
We also demonstrated that the strength of the response is determined by genes that influence discrete, stage-specific immune responses to *T. spiralis.* Each response is governed by a particular set of genes. For example, the rapid expulsion response is expressed in an all-or-none fashion in every mouse strain tested. Each strain either has or does not have the gene for rapid expulsion. The rapid expulsion gene is dominant, it is not located on the chromosome that determines sex, and it is not linked to the major histocompatibility complex, a well-defined genetic region on chromosome seventeen.

While rapid expulsion appears to be under the control of a single gene, other responses, such as anti–adult immunity or antifecundity, are not. Both of these responses are controlled by at least two different sets of genes, some of which appear to be linked to the major histocompatibility complex. Overall, it is likely that between six and twenty genes are involved in the response to *T. spiralis* in mice.

These studies have defined the genetic and immunological response pattern of the mouse and have provided genetic tools for examining the protective process. We are now investigating whether responses to other parasites are governed in a similar way. Parallel studies of the genetics of resistance of mice to *Nematospiroides dubius (Heligmosomoides polygyrus),* a hookwormlike intestinal nematode, have begun. Although these studies are still at an early stage, it is clear that there are significant differences in the response of mice to *T. spiralis* and *N. dubius.* While immunity to *T. spiralis* and *N. dubius* are subject to gene control in mice, there is some evidence that different genes govern the responses to these parasites. In studies of two mouse strains, both of which are strong responders to *T. spiralis,* one strain responded much more strongly to *N. dubius* than the other strain.

By conducting detailed genetic studies of this sort, we expect to gain new insights into the host-parasite relationships and to learn more about how immunity is expressed in the intestine.

Robin G. Bell



Immunogenetics Laboratory

During the past three years we have established a program for research on genetic systems in domestic animals. Our efforts have thus far concentrated on studies of the equine leukocyte antigen (ELA) system. Through the use of genetic, serological, and biochemical techniques, we have demonstrated that the ELA system is the major histocompatibility complex (MHC) of the horse. The MHC is a highly variable genetic region that has been identified in several mammalian species, including human beings, mice, cattle, and dogs. Although the complete structural and functional characteristics of the MHC are not yet known, it has been established that the MHC is involved in many types of intercellular communication. This communication is important in many aspects of the immune response to infectious agents and tumors and in allergic diseases. There is also evidence that the MHC has a significant role in maternal-fetal interactions in pregnancy.

Using antibody and cell culture techniques, we have identified twenty-three different ELAs. With the initiation of a parentage verification service for horse breeders in New York State, we have begun to apply our knowledge of the ELA system. We are also collaborating with Dr. W. R. Allen of the British Thoroughbred Breeders' Equine Fertility Unit in Cambridge, England, to study the relationship between the ELAs and equine pregnancy. It is our goal to use our knowledge of equine genetics to investigate the genetic basis of equine diseases. This year we were instrumental in organizing an international conference on ELAs, conducted by the Dorothy Russell Havemeyer Foundation.

In 1980 we undertook a pilot project to determine if a new technique in genetic engineering, the production of monoclonal antibodies by cell hybridization, could be applied to problems of animal health. The project was an overwhelming success, and we have dedicated much of our efforts to the application of this form of genetic engineering to research programs at the Baker Institute, the College of Veterinary Medicine, and elsewhere on the Cornell campus.

The technique involves the fusion of single antibody–forming cells (lymphocytes) with tumor cells to create hybrid cells with properties of both parent cells. The hybrids have an unlimited capacity to grow, like the tumor cell parent, and they produce the single type of antibody molecule that their normal lymphocyte parent produced. Such hybrid cells can produce unlimited quantities of exquisitely specific antibodies. By appropriate selection techniques, it is possible to produce monoclonal antibodies to virtually any antigen.

Our own research has concentrated on producing hybrid cell lines that secrete monoclonal antibodies to antigens of horse red and white blood cells. We have worked with Dr. Leland Carmichael and Mr. Colin Parrish to produce monoclonal antibodies to canine parvovirus, and we are collaborating with Dr. Robin Bell to produce monoclonal antibodies to the parasite *Trichinella spiralis*.

Collaborative projects have been initiated with several other researchers on the Cornell campus in the Departments of Chemistry, Genetics, Biochemistry, Nutrition, and Veterinary Microbiology. We are trying to produce monoclonal antibodies to a wide range of antigens, including protein molecules, free-living single-cell organisms, and tumor viruses.

Douglas F. Antczak

Richard King Mellon Laboratory for Electron Microscopy

The electron microscopy laboratory provides a diagnostic service for the Baker Institute by searching for and identifying viruses in tissue cultures and tissue samples from sick animals. We also use the electron microscope for research. This year we concentrated on two projects: (1) a structural analysis of the articular cartilage from dogs with hip dysplasia and (2) a comparison of different strains of canine distemper virus (CDV) and the cells in which they grow.

With the high resolution made possible by the electron microscope, we observed changes in the joint tissues of dogs with hip dysplasia. Damage to the articular cartilage was found to be a conspicuous early feature of the osteoarthritis associated with hip dysplasia. High-magnification electron micrographs demonstrated that the articular surface of normal cartilage is covered by fibrous material whose composition is unknown (fig. 1). In dogs with hip dysplasia and osteoarthritis the surface of the cartilage is eroded and the underlying collagen fibrils are more widely spaced (fig. 2).

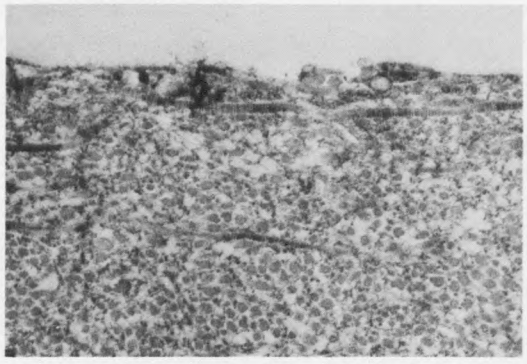


Fig. 1. Normal cartilage from a young dog, × 36,500. Collagen fibrils, seen mostly in cross section, are tightly packed. Several fibrils are cut longitudinally, showing their characteristic banding structure. The matrix appears as fibrous material among the collagen fibrils. The cartilage surface is covered by a thin layer of fibrous material.

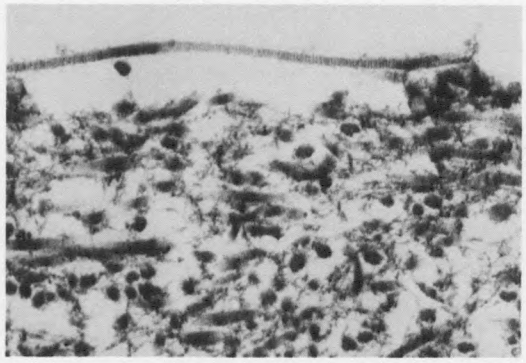


Fig. 2. Cartilage in early osteoarthritis in a young dog, × 36,500. The fibrous surface layer is missing, the collagen fibrils are more widely spaced, and the matrix is thinner. One collagen fibril is fraying into the joint space.

The hip joint, and other movable joints, are surrounded by a tough, collagenous capsule lined by a thin layer of cells, the synovial membrane. Inflammation of the synovial membrane, or synovitis, is a common feature of osteoarthritis and has been cited as a causal factor in the disease process. However, our electron microscopic studies demonstrated that damage to the articular cartilage often occurs before any change can be detected in the synovial membrane. This finding suggests that synovitis may be a reaction to injury rather than a causal factor in the disease process.

A traditional aspect of our research has been to locate and identify viruses in infected cells. Studies of this sort are revealing, because viruses often infect and grow in some, but not all, cells. Determining the range of cells infected by a particular virus can help explain the pattern of disease. We are engaged in such an investigation at the moment. Strains of CDV with different biological characteristics are being examined. Aside from structural differences in the viruses themselves, we are seeking differences in their infectivity for various cell types in the tissues that are the seat of infection.

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The Baker Institute in Perspective

The Baker Institute was founded in 1950 as a permanent facility for research on animal diseases and the teaching of all that pertains to such diseases. Its mission is to seek measures for controlling disease and to provide advanced training for scientists in comparative medicine, thereby ensuring that the Institute's work will be perpetuated.

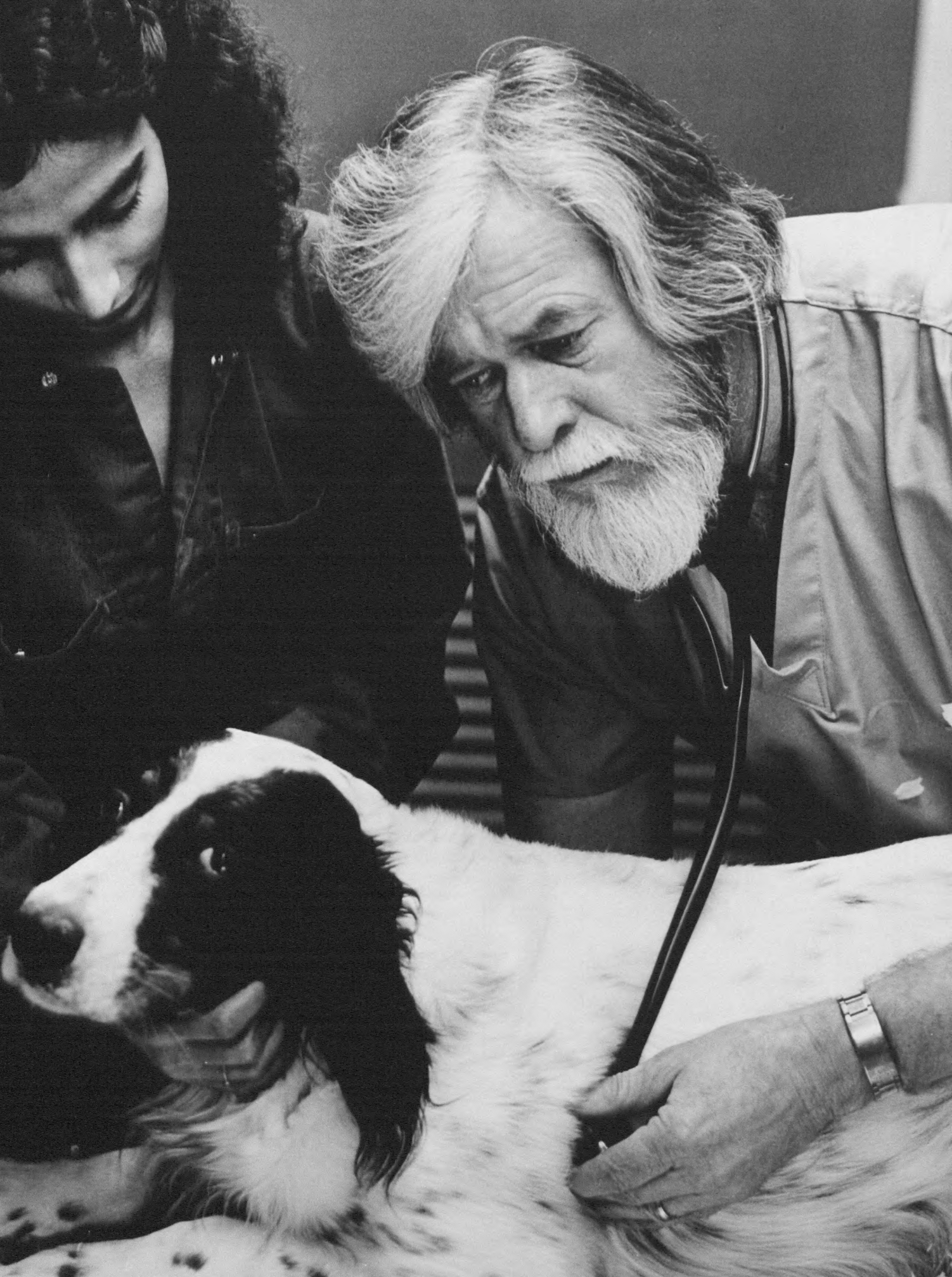
The Institute has made some giant steps to fulfill the promise implicit in its charter. From a modest beginning it has grown into a modern, well-equipped research enterprise that operates at the forefront of veterinary medicine. Its history is one of innovation, productivity, and service. Many pathogenic viruses were first isolated at the Baker Institute; the diseases caused by these agents in dogs and domestic animals were characterized, and measures were developed for disease control. Research with the same objectives continues.

The year 1981 marked the start of a significant new phase in the Institute's development. A three-year capital improvement program was completed. The improvements have nearly doubled the useful working space at the Institute. New facilities have been created to meet the exacting standards required for the study of infectious diseases and for the sheltering of animals, who are the ultimate beneficiaries of the Institute's research.

The Baker Institute is breaking new ground in studies of the immune response to parasites and in the analysis of genetic factors that influence the susceptibility of animals to disease. The application of genetic engineering methods to the production of monoclonal antibodies has been given prominence. The technique for producing such antibodies, and their applications in research and in the diagnosis of disease, are described elsewhere in this report.

An additional benefit derives from the Institute's pioneering work with these "modern miracles of science." Research undertaken in the Institute's cell fusion facility has fostered collaboration with Cornell University scientists in the Departments of Chemistry, Biochemistry, Genetics, Nutrition, and Veterinary Microbiology. This collaboration has had a favorable impact on the Institute's own programs and can be expected to open new avenues to the improvement of animal health. Commenting on the significance of these programs, Cornell University's president, Frank Rhodes, emphasized "the spirit of innovation and commitment to excellence that have guided the Institute's efforts to improve the health of animals and of man himself."

Dorothy R. Donnelley



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Your interest in the James A. Baker Institute for Animal Health, expressed by your gift, enables us to carry out our day-to-day mission. With your support we can respond swiftly to opportunities as they arise and improve the quality of animal health. Your gift earns the Institute's deepest thanks.

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*Gave $100–$499 since January 1, 1981.
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Willamette Valley Kennel Club
William Penn West Highland White Terrier Club
Wolverine Dog Training Club, Inc.
Wyoming Valley Beagle Club
York Kennel Club, Inc.
(In honor of Dr. Harold Neibert)
Yorkshire Terrier Club of the Nation's Capital
(In honor of Mrs. Betty Munden)
Yucca German Shorthaired Pointer Club

Veterinary Associations

Central Alabama Veterinary Medical Association
Finger Lakes Veterinary Medical Association
Northwest Jersey Veterinary Society
Quad Cities Veterinary Medical Association
Western New York Veterinary Medical Association, Inc.
Women's Auxiliary Long Island Veterinary Medical Association
Women's Auxiliary to the New York State Veterinary Medical Society

Foundations and Trusts

Allegheny Foundation
C.A.L. Foundation
Geraldine Rockefeller Dodge Foundation, Inc.
Gaylord Donnelley Foundation
Gilbert W. and Louise Ireland Humphrey Foundation
Ireland Foundation
Walter Kendall Trust
Robert J. Kleberg, Jr., and Helen C. Kleberg Foundation
Kroc Foundation
James A. Macdonald Foundation
Richard King Mellon Foundation
Dr. J. E. Salsbury Foundation
Shepard Foundation
Trebor Foundation, Inc.
Westminster Kennel Foundation
Robert C. Whitehouse Estate
Robert Winthrop Trust

Companies

Acmaro Securities Corporation
American Boarding Kennel Association Region 2
Animal Inn, Inc.
Biotron Laboratories, Inc.
Burns-Biotec Laboratories Division
Central Kentucky Small Animal Hospital Association
Chemical Bank and Trust Company
Cleveland Animal Protective League
Docktor Pet Centers, Inc.
Dog Owners Educational League, Inc. (In honor of Dr. Arthur North, Jr.)
Gaines Dog Research Center
General Foods Corporation
Gibbs and Hill, Inc.
Hoffmann-LaRoche, Inc.
Marble Hill Crematory for Pet Animals, Inc.
Mony Trust
Oaks Plantation, Inc.
Professional Medical Supply Company, Inc.
Ralston Purina Company
SmithKline Corporation
Sterling-Winthrop Research Institute
Veterinaria AG, Zurich

In Memoriam

Mr. Steven Averill III
Mr. Joseph Binder
Mrs. Andrew G. Carey
Mrs. Alta Clark
Dr. David B. H. Dalrymple
Mrs. Lorraine L. Donahue
Mrs. Charles Forrest Dowe
Dr. Myron G. Fincher
Dr. Lendall K. Firth
Dr. Herbert R. French
Mrs. Mary Gannon
Ms. Gretchen Greiner
Ms. Greta Haight
Mrs. Charles Lamphere
Mrs. Pierce Onthank
Ms. L. Grace Proskey
Mr. George S. Russell
Col. Louis L. Shook
Mr. Vernon Lee Smith
Mr. Peter Spytz
Mrs. Rhea Struble
Mr. Charles Vota

Publications

Publications listed as *in press* in last year's report are repeated this year, with their original numbers, to record their full bibliographic details.

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