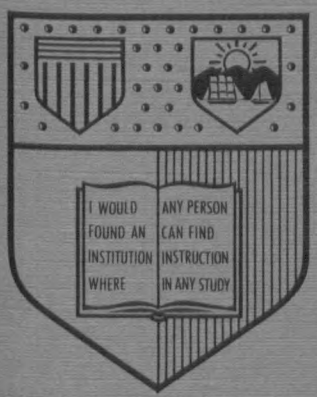
James A. Baker Institute for Animal Health

Annual Report 1980



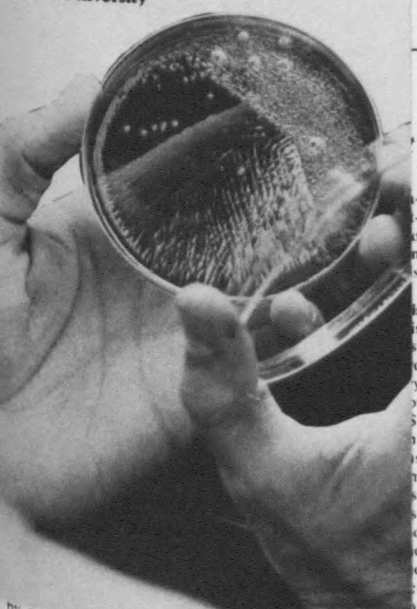
Cornell University

New York State College of Veterinary Medicine
Volume 30

This report honors those
whose generosity sustains the Institute's independence
and commitment to excellence and the pursuit of truth.

Canine Brucellosis: The Silent Threat

James A. Baker Institute
for Animal Health
Cornell University



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Cornell Research Laboratory for Diseas

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FOR ANIMAL HEALTH
Cornell University
Ithaca, New York 14853

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Cornell Research Laboratory for Diseases of Dogs

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FOR ANIMAL HEALTH
Cornell University
Ithaca, New York 14853

Laboratory Report
Series 3, Number 2
September, 1980

Feeding For Health and Long Life in the 80's

We are told—"Never has the opportunity for supplying good nutrition for dogs been better than it is today." TODAY, however, the average dog competes directly with other members of his family for a share of the household budget.
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Weight of Dogs		*Ad*
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15	6.8	
30	13.6	
50	22.7	
75	34.1	
100	45.5	

1980 modification by Cor
NAS-NRC Publication No.

or Diseases of Dogs

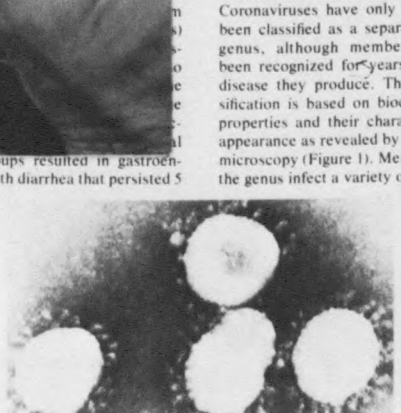
Laboratory Report
Series 2, Number 9
July, 1978

by a Corona-like Virus:
for Information

to 6 days. Contact animals developed the disease within 4 days, proving its contagiousness. Deaths were not reported. In a second study by the same group of investigators (Am. Jour. Vet. Res. *37*: 247–256, March 1976), virus was found to persist in feces for 6 to 9 days, although it could be recovered from intestinal tissue for 10 days. The infection was not fatal in pups, and atrophy of the villi of the intestine healed within 7 to 10 days. There was rapid clinical recovery.

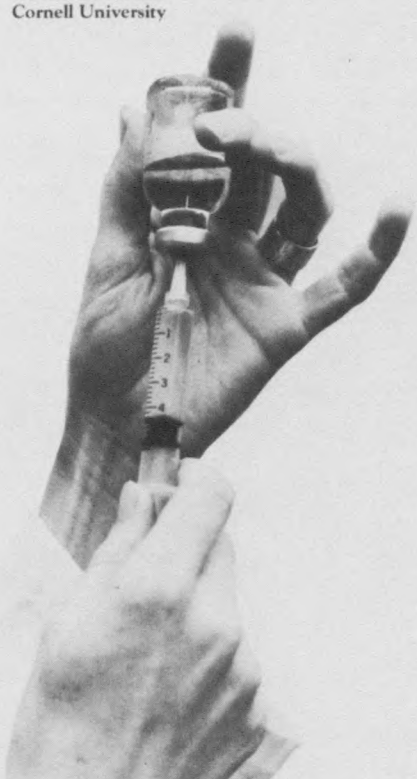
What are coronaviruses? Coronaviruses have only recently been classified as a separate viral genus, although members have been recognized for years by the disease they produce. Their classification is based on biochemical properties and their characteristic appearance as revealed by electron microscopy (Figure 1). Members of the genus infect a variety of animal

beagle pups resulted in gastroenteritis with diarrhea that persisted 5



Canine Parvovirus

James A. Baker Institute
for Animal Health
Cornell University



standing of this debilitating condition has been a long sought objective of owners, breeders, and veterinarians alike.

Why has progress been erratic and slow? For a number of years dog breeders have attempted to eliminate hip dysplasia from the population through selective breeding programs. The essential doctrine upon which this endeavor has been based was to breed only those dogs which have normal hip joints. While progress has been made in reducing the incidence of hip dysplasia, systematic improvement has been slower than anticipated. The reasons for this have not been determined and will re-

tion — An Emergin

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Founded for the Future

James A. Baker Institute
for Animal Health
Cornell University

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phasized extensively. It is a developmental deformity since abnormalities in hip joints have not been detected at birth, but appear later in the life of dogs at risk. A number of events that precipitate hip dysplasia in dogs or contribute to its severity have been proposed, but its etiology and pathogenesis have not yet been defined. Several observations associated with the disease have been difficult to explain; for example, the disease is found mainly in the large breeds of

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A Message from the Director

The year 1980 was a strong and productive one for the James A. Baker Institute for Animal Health. The optimistic expectations expressed in last year's report were realized in significant advances in our research, improvements in our facilities, and growth of our public relations and continuing education programs.

The Institute has experienced many important events in its thirty-year history, but none more significant than the sudden emergence of canine parvovirus (CPV) in the dog population two years ago. CPV is an entirely new pathogen of dogs. The development by Drs. Leland Carmichael, Roy Pollock, and Max Appel of practical strategies for diagnosing and controlling CPV infection represents a landmark advance in veterinary medicine.

While these accomplishments have been widely acclaimed, they have placed a heavy burden on our staff. This year alone we received more than fourteen thousand telephone calls and letters requesting information and assistance. All were answered promptly and efficiently while our research staff went about the business of developing reliable methods for diagnosing CPV infection and producing safe, effective vaccines for disease prevention. Reports on CPV, as well as canine brucellosis and the nutritional requirements of dogs, were prepared and distributed throughout the country. Copies of these reports can be obtained by writing to the Institute or telephoning Mrs. Florence Huth (607/277-3044).

CPV dominated the headlines; however, we also made significant advances in other areas. Our research findings are described elsewhere in this report, in the various pamphlets and reports prepared by the Institute, and in articles printed in scientific journals. In 1980 a record number of such reports were published.

Several projects in the program to improve our facilities were completed this year. A new wing, opened in November, houses laboratory animals in a rigorously controlled environment. The second floor of the Institute was extensively renovated as part of a project that will create a new laboratory of immunochemistry. This laboratory will have the equipment needed to isolate and purify microbial antigens, antibodies, and other factors that contribute to the host's defense against infection. Also, improvements were made to our canine isolation units, and a new central storeroom was opened.

The Dorothy Havermyer Foundation this year appointed Dr. Douglas Antczak as a foundation investigator. This award will enable him to continue his studies of the major histocompatibility complex of the horse.

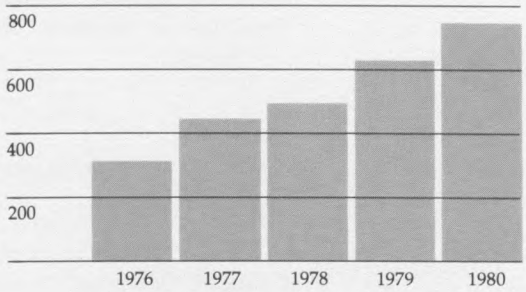
The Institute sponsors training programs and encourages its staff to participate in work-related seminars and courses offered elsewhere on the Cornell campus. In 1980 Messrs. Raymond Corey, James Hardy, David Watkins, and James Young received advanced-level certification by the American Association for Laboratory Animal Science. They join other certified members of the staff who have demonstrated their commitment to high-quality animal care.

Expenditures for research reached a record level this year. The monies needed to sustain our operations were provided by research grants and contracts from the federal and state governments, foundations, and industry and by gifts from veterinarians, kennel clubs, and individuals. The accompanying figure shows that the

level of research support has increased each year since 1976 and has doubled in the last four years.

We have entered a decade of political uncertainty, continuing inflation, and diminishing resources. Yet we can meet this challenge and maintain the Institute's record of innovation, productivity, and service. Our confidence springs from the knowledge that we have a skilled and dedicated staff of men and women, an advisory council made up of leaders of the scientific and business communities, and a growing constituency of concerned benefactors. In an uncertain environment there can be no greater asset for an institution.

Research Support
(in Thousands of Dollars)



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
Kim T. Arcangeli, secretary
Florence C. Huth, secretary
Audrey E. Lowes, secretary: A.A., Paul Smith's College
Ann W. Signore, secretary: Cornell
Douglas S. Robson, consultant in statistics: B.S., M.A., Iowa State University; Ph.D., Cornell

Laboratories

Giralda Laboratories for Canine Infectious Diseases

Leland E. Carmichael, John M. Olin Professor of Virology: A.B., D.V.M., University of California; Ph.D., Cornell
Colin R. Parrish, graduate research assistant: B.Sc., Massey University
Roy V. H. Pollock, graduate research assistant: B.A., Williams College; D.V.M., Cornell
Steven J. Zoha, postdoctoral associate: B.S., Lehigh University; Ph.D., Cornell
Jean C. Joubert, research technician
Priscilla H. Dunham, laboratory technician
Susan P. Montgomery, laboratory attendant: B.A., Harvard

Daynemouth Laboratory for Canine Nutrition

Ben E. Sheffy, Caspary Professor of Nutrition: B.S., M.S., Ph.D., University of Wisconsin
Alma J. Williams, laboratory technician: B.A., University of Pennsylvania; M.S., Cornell

Biochemistry Laboratory for the Study of Canine Hip Dysplasia

George Lust, associate professor of biochemistry: B.S., University of Massachusetts; Ph.D., Cornell
John M. Olsewski, graduate research assistant: B.A., Cornell
Susan J. Harter, laboratory technician: B.S., Lock Haven State College

Hadley C. Stephenson Laboratory for Study of Canine Diseases

Max J. G. Appel, professor of virology: Dr.med.vet., University of Hannover; Ph.D., Cornell
Joseph M. Friedlander, graduate research assistant: B.S., Cornell
Stuart G. Mendelson, laboratory technician: B.A., Cornell
Mary Beth Metzgar, laboratory technician: University of Evansville

Microbiology Laboratory

Douglas D. McGregor, professor of immunology: B.A., M.D., D.Phil.
Melissa C. Woan, postdoctoral associate: B.Ed., Taiwan Normal University; M.S., Ph.D., University of Illinois
Laura M. Stenzler, laboratory technician: A.A.S., State University of New York Agricultural and Technical College; B.S., Cornell

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
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
Linda H. Remick, research technician: A.A.S., State University of New York Agricultural and Technical College; B.S., Cornell
Beverley E. Bauman, laboratory technician: B.S., Rochester Institute of Technology
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

Linda H. Remick, research technician: A.A.S., B.S.

Glassware Department

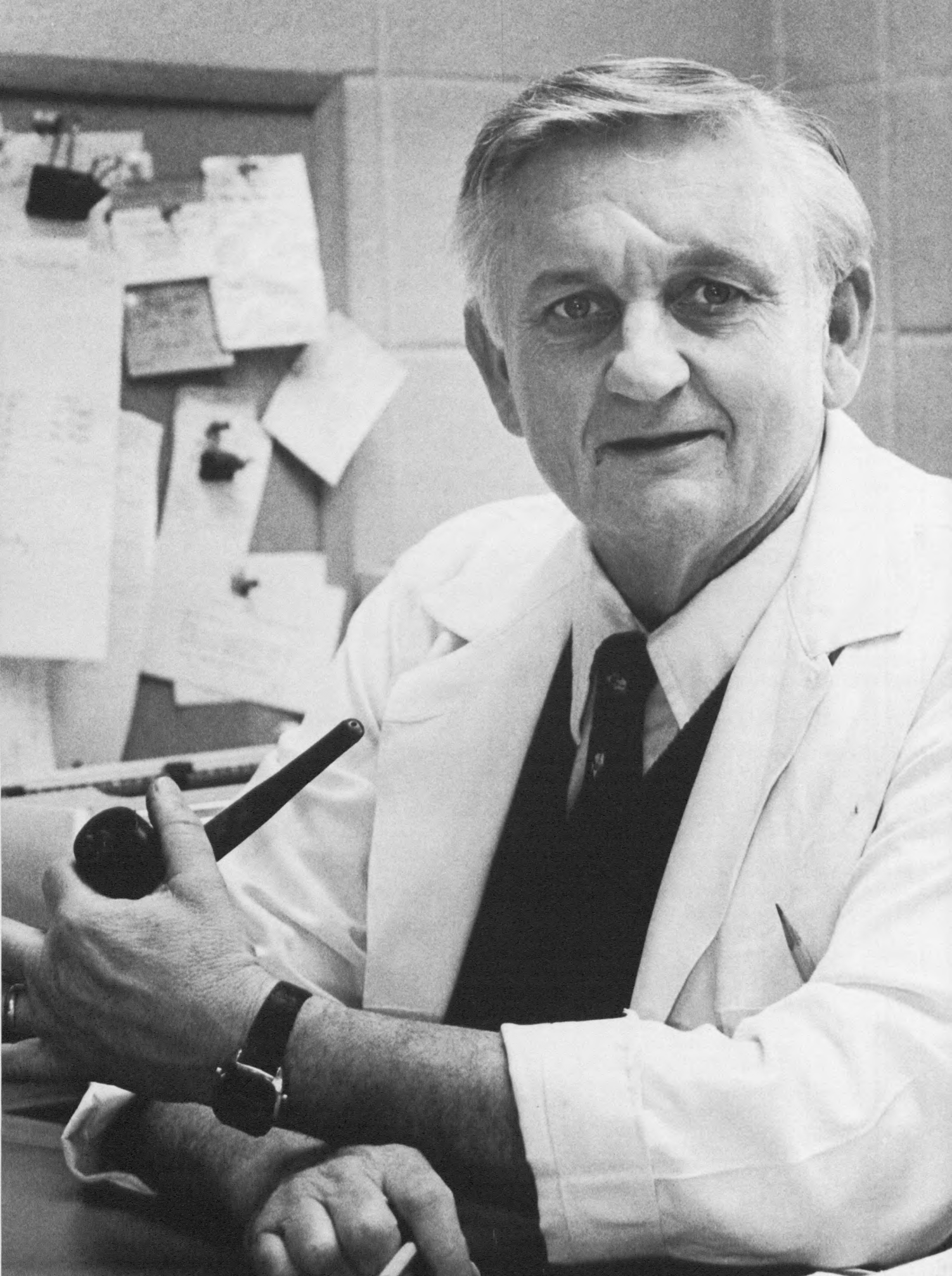
Elizabeth C. Wheeler, supervisor
Jeannette R. Kniffen, laboratory attendant

Animal Care

Bernard L. Clark, research technician
James C. Hardy, research technician: B.S., Cornell
Charles B. Bailor, animal technician
Roy L. Barriere, animal technician: AALAS accreditation
Raymond J. Corey, animal technician: A.A.S., State University of New York Agricultural and Technical College
James M. Ebel, animal technician
Gerald W. Hiller, animal technician: AALAS accreditation
Ronald L. McKinney, animal technician
David L. Watkins, animal technician: A.A.S., State University of New York Agricultural and Technical College; AALAS accreditation

Maintenance

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



A new parvovirus appeared in the canine population in 1978. It is now a major cause of disease in dogs throughout the world. The origin of canine parvovirus (CPV) is unknown, but its similarity to feline panleukopenia virus (FPV) and mink enteritis virus (MEV) indicates that these viruses are related. This similarity accounts for cross-reactions between antibodies formed in response to these viruses and the ability of the feline virus to protect dogs against canine parvovirus infection. Mr. Colin Parrish is examining the precise relationship of CPV and FPV/MEV; his findings may reveal how CPV suddenly emerged as a new pathogen of dogs.

Whatever its origin, CPV is now a cause for concern. Two forms of the disease are recognized: (1) myocarditis in young pups and (2) systemic illness, with the principal manifestations being enteritis and profound immunodepression due to viral growth in lymphatic tissue. The current status of CPV is summarized in a recent Institute bulletin *(Canine Parvovirus)*.

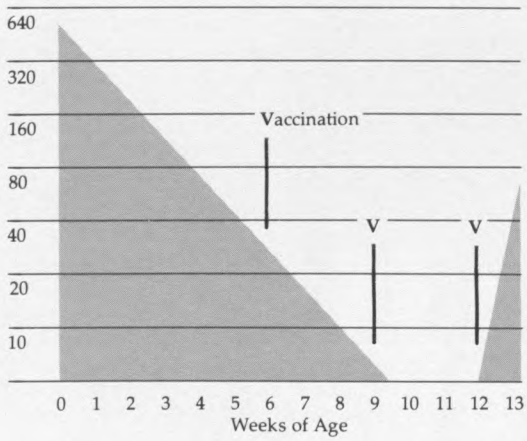
Dr. Roy Pollock continued to investigate the response of dogs to various parvoviral vaccines. This work initially involved the development and testing of inactivated FPV and CPV vaccines. Betapropriolactone (BPL) was shown to inactivate CPV while preserving its capacity to stimulate the formation of antibodies. In this regard both BPL and various aziridine compounds were superior to formaldehyde.

Inactivated CPV, FPV, and MEV vaccines all produced immunity to CPV. In each case the antibody response was proportional to the amount of inactivated virus in the vaccine. Antibody levels increased rapidly after vaccination, to a maximum at two weeks, and then declined. A more sustained response was achieved when two doses of inactivated vaccine were given three to four weeks apart. But even after two injections the antibody response declined over a period of nine to twelve weeks, at which time dogs challenged with virulent CPV shed virus in their feces. It is unlikely that such vaccines can interrupt virus spread.

The response of dogs to living FPV was also found to be related to the dose of virus. An experimental vaccine containing a large amount of FPV resulted in all cases in high and durable (greater than six months) antibody levels. This vaccine contained about ten times the amount of FPV in commercial vaccines. A single injection of the latter immunizes about 60 percent of dogs; two injections, about 80 percent.

Critical levels of antibody, either passively acquired or generated in response to injections of inactivated vaccines or live FPV, can block the response to subsequent injections of parvovirus vaccine. In dogs successfully immunized with FPV, antibody

Effect of Maternal Antibody on Response to Inactivated FPV
(Mean Serum Titer)

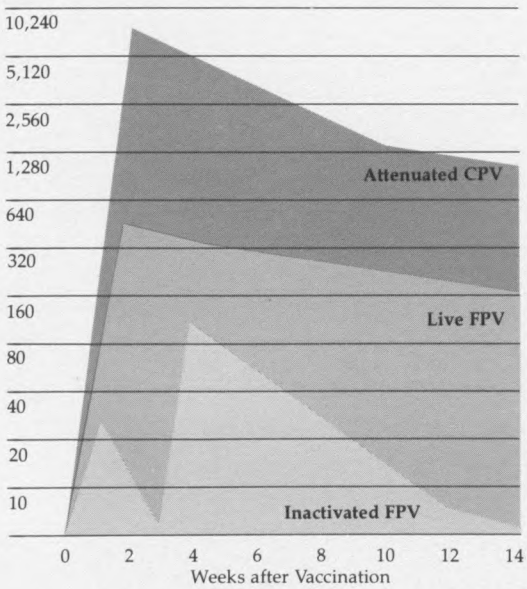


concentrations are maintained at a protective level for at least six months. Since animals challenged with CPV during this period do not shed virus, there is a greater prospect of interrupting the spread of CPV through the use of living FPV vaccines than inactivated virus vaccines.

The development and testing of an attenuated homologous CPV vaccine that is safe and effective and provokes long-lived immunity has been a major objective of our research. A large-plaque (LP) variant was developed, and this variant was found to be less virulent than the naturally occurring small-plaque type. Further studies established the permanent attenuation of the LP variant. Laboratory and field studies in more than twelve hundred dogs have further established that this virus can safely be used in vaccines for dogs of all ages, including specific pathogen–free pups inoculated during the critical newborn period. The antibody levels in vaccinated dogs are high, indicating a vigorous immune response. The duration of immunity is not yet known; however, studies in progress indicate that it lasts at least eighteen months.

Antibody Responses to Parvovirus Vaccines

(Mean Serum Titer)



Our work on canine brucellosis was continued by Dr. Steven Zoha. Several cell wall antigens and internal antigens of *Brucella canis* were isolated and purified. One of the cell wall antigens and three internal antigens seem to be specific for *B. canis*. These antigens are being evaluated as reagents for the serodiagnosis of canine brucellosis. Also, a variant strain of *B. canis* that is nonpathogenic for the reproductive tract was found to be physically, chemically, and antigenically different from virulent *B. canis*. The attenuated variant, which can immunize dogs against virulent *B. canis*, is being evaluated as a potential vaccine strain.

Leland E. Carmichael

The role of vitamin E (E) and selenium (Se) in the immune response and maintenance of vision were evaluated, and new studies were undertaken to define the nutritional requirements of dogs.

Last year we reported on a substance in the serum of dogs deficient in vitamin E and Se that inhibits the in vitro proliferation of T lymphocytes after stimulation by polyclonal mitogens. The inhibitory factor has not yet been identified, but we now know that it also inhibits the lymphocytes of humans, cattle, pigs, and cats. Supplementation of the dog's diet with vitamin E in the amount normally recommended (50 mg per kg of diet) eliminates the inhibitory effect.

Supplementing the diet of nondeficient puppies with higher than the recommended levels of E and Se (0.1 mg per kg of diet) before, during, or after vaccination with canine distemper and canine adenovirus biologicals did not increase the rate of specific antibody production or the level of immunity three weeks later. These findings indicate that the vitamin E and Se content of commercial dog foods is sufficient to support an optimum immune response to vaccination.

Last year we described a severe dermatosis resulting from a deficiency of both vitamin E and Se in dogs fed diets high in polyunsaturated fatty acids (corn oil). Recent studies have shown that another nutrient, zinc, is involved in this syndrome. Zinc requirements are increased when dogs are fed diets high in unsaturated fat and marginal in vitamin E and Se. It may be that this combination results in abnormal prostaglandin metabolism; formal proof will require direct measurements of prostaglandins and prostaglandin intermediates in the skin.

The pivotal roles of vitamins A and E in the maintenance of vision are being investigated in collaboration with Dr. E. R. Loew. The influence of dietary unsaturated fatty acids, Se, and sulphur-containing amino acids are of special interest. These studies, sponsored by the National Eye Institute, will be a major aspect of our research in 1981.

The number of aged in the dog population, as in the human population, is increasing. Many questions have been raised about their nutritional needs and about the effects of individual nutrients on the course of degenerative diseases of the aged. We are undertaking a study of the comparative needs of one-year-old dogs and nine- and ten-year-old dogs.

The dog is basically a carnivore with a short and simple digestive tract. Nevertheless, it is adapted to being omnivorous both in habit and in nutrient utilization. In the metabolism unit of the Daynemouth Laboratory we are determining the differences in alimentation and nutrient utilization due to the particle size and degree of cooking of corn fed to dogs as part of a balanced diet. We have determined that cooking has no beneficial effect on either alimentation or total nutrient digestibility when the corn (52 percent of the diet) is ground to a fine consistency. These experiments, however, do not take into consideration palatability of the diet.

Ben E. Sheffy


PICKER

Biochemistry Laboratory for the Study of Canine Hip Dysplasia

The major goals of our research on canine hip dysplasia are to understand how and why the disease occurs.

A common misconception is that the disease affects the hip joint only. This year we obtained clear evidence that the same disease process occurs in other joints, including the shoulders, knees, and elbows. Hip dysplasia may thus be simply the most conspicuous manifestation of a disease affecting many joints.

To find out why the hip joint is a prominent site of disease and why only some dogs are affected, we need to know more about the development and function of tissues in the region of the hip joint. This year we analyzed these tissues in Labrador Retrievers. We discovered that both body weight and the length of the femurs (thighbones) increase during the first five months of life. After that, the femurs stop growing in length while body weight continues to increase. We also found that the growth plates of the acetabulum (joint socket) usually close at six months of age, whereas the growth plates of the femoral head remain open several months longer. It may be significant that the initial X-ray diagnosis of hip dysplasia is made most often when dogs are five to six months old.

We also examined the relationship between torsion, or twisting, of the femur and the development of hip dysplasia. Femoral torsion angles were five to fifteen degrees in dogs with normal hips, and thirteen to twenty-three degrees in dogs with hip dysplasia. Thus there is an abnormal configuration of the femur in dogs with hip dysplasia. We will undertake further studies to find out whether the degree of femoral torsion puts stress on the opposing bones of the hip joint.

It has been known for many years that genetic factors influence the development of hip dysplasia, and that the prevalence of hip dysplasia can be reduced through selective breeding. It may be that the process of selecting animals for breeding can be improved. We have observed that the development of hip dysplasia is delayed when the growth rate of puppies during the first three months of life is restricted. The reverse is also true: the development of hip dysplasia is accelerated when the growth rate is increased. These observations suggest a practical strategy for selecting breeding stock. In a population of dogs being considered, the expression of hip dysplasia would be forced through manipulation of the diet and other management practices. Only animals with normal hip joints would be chosen from this group for breeding purposes. By forcing the expression of hip dysplasia in young pups, one would disclose abnormalties of the hip joint that might otherwise escape detection. The procedure would also reveal the potential for transmitting the disease in apparently normal parents.

By excluding from the breeding population dysplastic animals and those who have the potential to transmit disease, one could increase the frequency of dysplasia-free offspring. A program of this kind requires a strong commitment by breeders, because the initial result would be to produce more dysplastic dogs. The reward is the prospect of reducing the prevalence of hip dysplasia in subsequent generations.

George Lust

Hadley C. Stephenson Laboratory for Study of Canine Diseases

We continued our studies of canine parvovirus (CPV) infection. Parvoviral infections of several species are associated with reproductive failure and developmental abnormalities, particularly cerebellar hypoplasia. To determine whether aftereffects of this kind occur in dogs that have had CPV, we undertook an epidemiological study in a large kennel, with the help of Drs. Larry Glickman and Paul Meunier. The kennel was virtually free of CPV at the beginning of the study, whereas nearly all dogs showed evidence of infection two years later. There was no significant change in the proportion of barren bitches, prevalence of stillborn pups, average litter size, number of pups weaned per litter, or prevalence of cerebellar hypoplasia. The results suggest that breeding failure and fetal abnormalities are infrequent complications of CPV infection. This conclusion is supported by experimental evidence indicating that fetal infections are uncommon even when pregnant bitches are infected with CPV.

To demonstrate differences in the expression of CPV infection in individual animals, studies in specific pathogen–free dogs and conventionally reared dogs continued. The early growth of CPV in lymphatic tissue and viremia were found to correlate with the severity of enteritis later in the infection. Since CPV replicates only in dividing cells, perhaps the virus grows to high titer in lymphocytes that have been activated by environmental stimuli. Animals in this situation might be especially vulnerable to infection.

We continued our work with canine distemper virus (CDV) for two reasons. First, CDV is an important disease of dogs, and second, information gained in the study of CDV infection may give insight into certain complications of measles virus (MV) infection in humans. CDV and MV have similar molecular compositions and antigenic properties, and they cause similar diseases in dogs and humans. A better understanding of CDV infection may explain the development of an uncommon but potentially dangerous complication of infection with MV: encephalitis. It has long been suspected that encephalitis, and possibly certain chronic brain diseases such as multiple sclerosis, result from infection with an unusual strain of MV that persists. This possibility is being tested in experiments using CDV. In collaboration with Dr. Barry Bloom (Albert Einstein College of Medicine) we demonstrated that cell lines derived from human lymphocytes can be infected with different strains of CDV. Temperature-sensitive mutants have emerged from these cultures. These mutants are being tested for their capacity to induce persistent brain infections in dogs.

Several years ago a vaccine containing MV protein was administered to many people. Because the vaccine induced only partial protection, some vaccinated subjects later developed an unusual disease when infected with MV. A similar process might account for persistent infection and the development of chronic brain disease in dogs infected with CDV. This proposition is being tested in dogs by immunizing the animals with CDV glycoproteins provided by our collaborator, Dr. Erling Norrby (Karolinska Institute in Sweden). Our initial studies were inconclusive, because dogs injected with CDV proteins were protected against a challenge infection with virulent CDV. Additional experiments are planned using purified CDV proteins and different vaccination protocols to determine whether dogs immunized in this manner develop a persistent infection after viral challenge.

Max J. G. Appel

We continued our research on mechanisms of acquired resistance to pathogenic bacteria. We are particularly concerned with organisms that can survive and grow in macrophages. Such organisms cause chronic infections such as tuberculosis, brucellosis, tularemia, typhoid fever, and plague. Because many of these organisms have fastidious growth requirements, they can be studied only in laboratories equipped with the proper facilities for disease containment. Even then there is a risk of accidental infection. For these reasons we have chosen to study the infection caused by *Listeria monocytogenes* in laboratory rats. *L. monocytogenes* can be grown readily in standard culture media; the organism can be safely handled, and the disease caused by this agent is an acceptable model of more exotic infections.

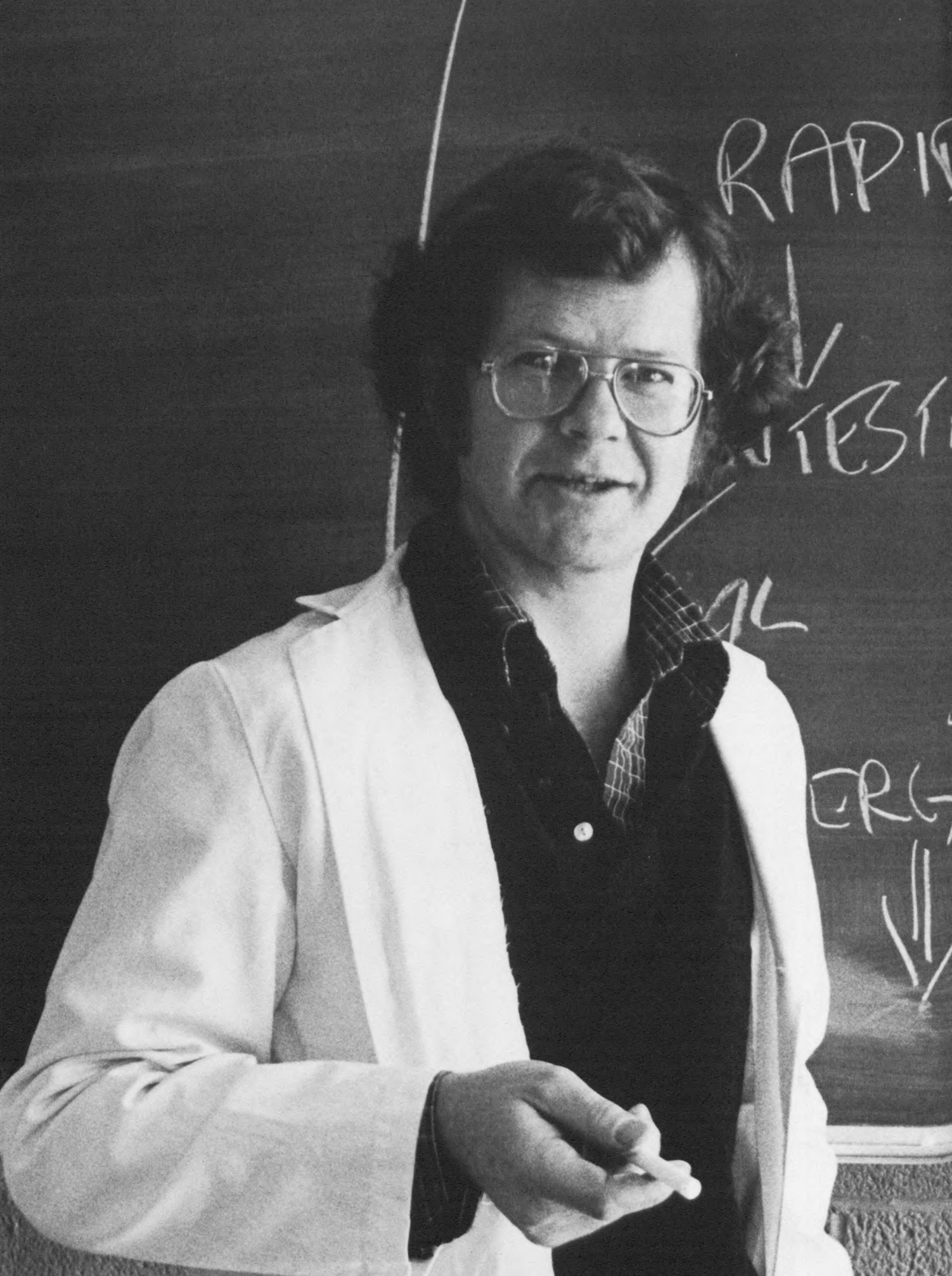
It has long been known that antibodies provide little if any protection against *listeriosis*. Immunity against the causal organism depends instead on the activity of a particular class of lymphocytes formed in the thymus. These thymus-derived (T) lymphocytes are exported to the spleen, lymph nodes, and other lymphoid organs and make up a substantial portion of the lymphocytes in the blood.

We have been studying how T lymphocytes recognize *Listeria* antigens and how the responses of these cells are regulated. We determined that T lymphocytes from rats convalescing from a recent *Listeria* infection can be maintained in tissue culture. When stimulated by *Listeria* antigens, some of these lymphocytes divide, while others acquire the capacity to kill a wide variety of the cells with which they come in contact. Our attention has focused on the nature and function of these killer lymphocytes. We now know that *Listeria*-dependent cytotoxic cells derive from T lymphocytes, although they have a far broader lytic capability than T cells activated by viruses, simple chemicals (haptens), or the antigens of foreign tissues.

Listeria-dependent killer cells are activated only in cultures containing specifically sensitized lymphocytes and *Listeria* antigens. We have shown that the responder lymphocytes in such cultures are efficiently activated only when the antigens concerned are displayed on the surface membranes of other (accessory) cells. Stimulation occurs only when the responder lymphocytes and accessory cells derive from rats of the same inbred strain. While the conditions for activation are thus shown to be antigen-specific and dependent on the cooperative interplay of histocompatible cells, the fully activated killer cells are aggressive toward a variety of cellular targets, including cells derived from different strains of rats and even some strains of mice.

We are now growing these cytotoxic lymphocytes in culture to find out how they realize their cytotoxic capability. We will also test the proposition that such cytotoxic cells have an immunoregulatory function and a defensive capability in the animal.

Melissa C. Woan


RAPI

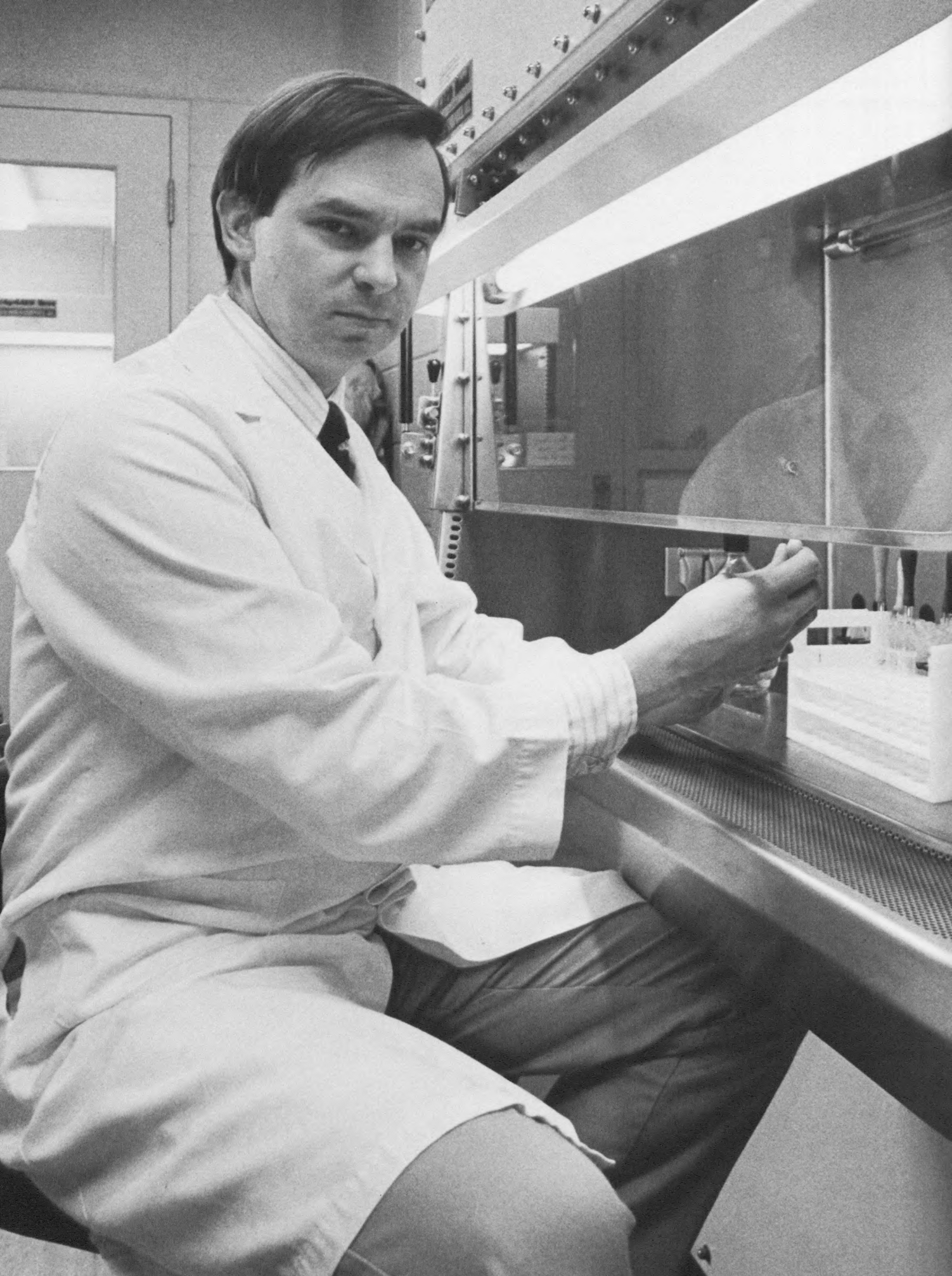
Our investigations of the host-parasite relationship have centered around a description and analysis of stage-specific host responses to the intestinal parasite *Trichinella spiralis*. The concept behind these investigations is that the ordered differentiation of the parasite in the intestine presents the host with a changing spectrum of antigens to which the animal can mount an immune response. The course of infection depends on the rapidity and strength of this response. Some antigens are expressed only briefly by infectious larvae. By the time the host responds to such antigens, they are no longer present on the mature worm. The response these antigens engender has little influence on a primary infection, but some antigens of this type can immunize when used in vaccines that are administered *before* infection.

Our studies in rats have demonstrated the existence of stage-specific immunity to *T. spiralis*. This year we extended these observations by showing stage-specific immunity to another parasite, *Strongyloides ratti*. We also demonstrated that mice respond to the antigens of *T. spiralis* in a manner similar to that observed in rats. Our experiments indicate that effective vaccines against this parasite will require a mixture of antigens from several stages: larvae, preadults, and adults. These studies provide an experimental basis for the development of vaccines against a variety of intestinal helminths that infect dogs, cats, domestic animals, and humans.

Other investigations were undertaken to define genetic factors governing the immune response to parasites. These studies were performed in inbred strains of mice infected with *T. spiralis*. We found that each inbred strain responds in a distinctive way: some strains respond slowly; others respond rapidly; while still others show an intermediate response. By breeding slow-responder to rapid-responder mice, we demonstrated that the pattern of responsiveness to *T. spiralis* is genetically controlled. Analysis of the response exhibited by certain slow-responder strains revealed that such mice lack the capacity to recognize one or more stage-specific *Trichinella* antigens.

A similar pattern of responsiveness has been observed in humans and domestic animals, where certain individuals and breeds (e.g., of sheep) are especially vulnerable to parasitic infections. Studies of the kind we are pursuing in mice may lead to the development of novel strategies for improving resistance; for example, by transferring the genes for resistance from responders to nonresponders. The successful development of such a method would have enormous economic significance by rendering susceptible animals resistant to disease.

Robin G. Bell



Immunogenetics Laboratory

We are developing laboratory tests for use in identifying horses in cases of disputed parentage. The test we use most frequently is a sensitive microcytotoxicity assay in which antibodies to equine leukocyte antigens kill peripheral blood lymphocytes. The antigens are polymorphic; that is, they exist in several forms, called alleles. These alleles are determined by genes within a particular chromosome region, the major histocompatibility complex. In the horse this genetic region has been designated the equine leukocyte antigen (ELA) region.

Eight new ELA specificities were identified in 1980. In addition, four other genetic markers within the ELA region were discovered, using a lymphocyte culture technique called the mixed lymphocyte interaction. These genetic markers make the ELA region the most polymorphic genetic region known in the horse, and there are still several markers in the ELA region that we have not identified. Because of this extreme variability, markers for genes in the ELA region will likely be an extremely useful system for determining parentage in the horse.

We are beginning to use our knowledge of the ELA system to study certain aspects of reproductive physiology in the horse. We have found that mares in their first pregnancy make a strong cytotoxic antibody response against the foreign tissues of their fetuses by day sixty of pregnancy. It is possible that these antibody responses are responsible for some cases of unexplained abortion. However, we have also made seemingly paradoxical observations suggesting that incompatibility between dam and fetus is beneficial to pregnancy. We are collaborating with Dr. W. R. Allen of the British Thoroughbred Breeders Association Equine Fertility Unit in Cambridge, England, to study this problem.

During 1980 we undertook experiments using a powerful new immunological tool to study immunogenetic systems of the horse. The technique involves the fusion of single antibody-forming cells with tumor cells to create hybrid cells with properties of both parent cells: the hybrids have an unlimited capacity to grow like the tumor parent, and they produce the single type of antibody molecule that they inherited from their normal antibody-forming cell parent. 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. Because monoclonal antibodies are homogeneous, their use in laboratory tests incurs fewer problems with cross-reactivity than are observed with conventional antisera.

This year we developed eleven hybrid cell lines producing monoclonal antibodies to several different horse red blood cell and lymphocyte antigens. We are now characterizing the cell surface molecules recognized by these antibodies. We want to expand our library of monoclonal antibodies next year and begin to use them in our investigation of the genetic basis of the immune capacity of horses.

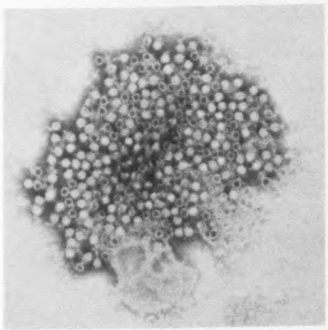
Douglas A. Antczak

Richard King Mellon Laboratory for Electron Microscopy

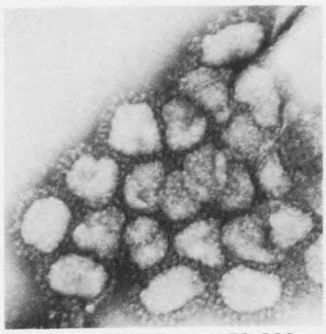
This laboratory houses an instrument that can magnify objects more than 200,000 times. With this instrument, a Phillips electron microscope, one can locate and identify viruses too small to be seen through the conventional light microscope. Furthermore, one can look inside cells to discover the effects of infection and other disease processes.

Tissues from both dogs and goats were examined to pinpoint the sites of initial growth of virus and spread of virus in the brain. The object of this research is to understand the disease processes caused by these neurotropic agents.

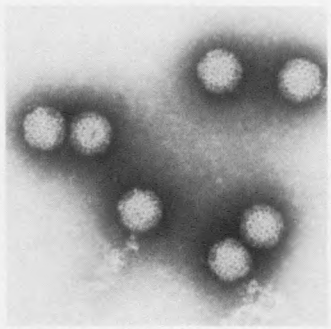
We also continued to provide diagnostic support for other laboratories where virus vaccines are being developed. Electron microscopy provides an accurate and rapid method for identifying viruses in tissue cultures and field specimens. We examined a large number of fecal specimens from dogs with enteritis. Canine parvovirus was the most common agent associated with disease, but other specimens contained canine coronavirus, rotavirus, and reo viruses.



Canine parvovirus, ×52,000



Canine coronavirus, ×73,000



Rotavirus, ×76,000

We also used the electron microscope to study early changes in the joint tissues of dogs with hip dysplasia. We demonstrated changes at the surface of the articular cartilage and in the synovial membrane, or joint lining, before disease could be detected in pelvic radiographs (X-rays) or by examining joint tissues in the light microscope. The early development of these changes may provide a clue to the underlying cause of this common and crippling malady of dogs.

Helen A. Greisen

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The Institute in Perspective: A Personal View

This year the Baker Institute celebrates its thirtieth anniversary. It is a time to look back as well as forward to review accomplishments and set new goals.

Much has been achieved during the first three decades of the Institute's existence. Space does not allow me to catalog these accomplishments. Suffice it to say that the Baker Institute has been at the forefront of research on infectious diseases and that it has had a hand in developing nearly all vaccines now used by veterinarians to protect dogs against disease. The Institute has also helped develop vaccines for other species and has contributed in other ways to the alleviation of suffering through research on hip dysplasia, parasitic infections, and diseases of the eye.

The Institute has also provided opportunities for the advanced training of scientists in comparative medicine. Many of the Institute's trainees now occupy positions of responsibility in academic institutions, government, and industry throughout the world.

These accomplishments are a credit to the individuals whose vision and financial support made possible in 1950 the founding of the Cornell Research Laboratory for Diseases of Dogs as a section of the Institute. In the ensuing years the Institute has grown into a modern, well-equipped enterprise that has attracted international recognition.

Throughout this period it has been nurtured and sustained by the contributions of many benefactors. Further progress will require a still greater effort in research and a significant new commitment of money to preserve the Institute's independence and vitality.

The Institute is positioning itself to attack health problems that still exact an unacceptable toll in suffering and economic loss. A new immunochemistry laboratory is being constructed for the isolation and purification of microbial antigens and other biologically important molecules. Next year a cell-hybridization unit will be created as a section of the Colgate Tissue Culture Laboratory. This unit, the first of its kind in veterinary medicine, will be used to generate antibodies of exquisite specificity. Such antibodies formed by artificially created cells will provide the Institute's scientists with powerful new tools with which to study disease-producing agents, as well as practical methods for diagnosing and preventing disease.

It has been my good fortune not only to have been involved in the creation of the Baker Institute, but also to have participated for three decades in planning and implementing the Institute's programs. I invite you to join in this effort and experience the same satisfaction. Together we can strive to preserve the spirit of innovation, productivity, and commitment that are the cornerstones of the Institute's success. The beneficiaries will be dogs and all who would improve the health and well-being not only of animals but of man himself.

John M. Olin

Acknowledgments

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.

In appreciation for their exceptional interest in the Institute, we should like to express our gratitude to Mrs. Warren Bicknell, Jr., Mr. Warren Bicknell III, Miss Wendy H. Bicknell, Mr. O. D. Carlton II, Mr. and Mrs. Gaylord Donnelley, Mrs. Priscilla Maxwell Endicott, Mr. and Mrs. Raymond F. Evans, Mrs. Pamela Firman, Mrs. Gordon H. Gillis, Mrs. Jeanne Lehman, Mrs. Trisha A. Murphy, Mr. John M. Olin, Mrs. Adelaide C. Riggs, Mr. William Rockefeller, Dr. Clarence C. Sapp, Jr., Mrs. Richard M. Scaife, Mrs. William E. Snee, Mr. William F. Stifel, Mr. Judson L. Streicher, Mr. and Mrs. Harwood Warriner, Mr. John Hay Whitney, Mr. Robert Winthrop, and Mr. Robert W. Woodruff.

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Afghan Hound Club of Greater Houston, Inc.
Afghan Hound Club of Hawaii
Afghan Hound Club of Northeast Florida
Airedale Obedience Club of Southern California
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American Belgian Tervuren Club
American Bouvier des Flandres Club, Inc.
American Boxer Club, Inc.*
American Brittany Club, Inc.*
American Bullmastiff Association, Inc.
American Pomeranian Club, Inc.*
American Sealyham Terrier Club, Inc.
Ann Arbor Dog Training Club, Inc.
Anne Arundel Kennel Club, Inc.*
Antioch Kennel Association‡
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Australian Terrier Club of America
Badger Golden Retriever Club, Inc.
Basset Hound Club of British Columbia
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Baton Rouge Kennel Club*
Bellfontaine All-Breed Kennel Club, Inc.
Bennington County Kennel Club, Inc.*
Berks County Kennel Club
Bloomington Obedience Training Club
Boomer Boxer Club, Inc.
Boxer Club of Oklahoma, Inc.
Boxer Club of Western New York
Brevard Kennel Club*
Bulldog Club of America
Bulldog Club of Connecticut
Bulldog Club of Northern California
California Rare Breed Dog Association*
Carolina Dog Training Club, Inc.*
Catonsville Kennel Club, Inc.†
Cavalier Doberman Pinscher Club*
Central Florida Kennel Club, Inc.*
Central Illinois Shetland Sheepdog Club
Central Jersey Beagle Club
Central New Jersey Chapter of the Owner Handler Association of America*
Central Ohio Kennel Club, Inc.
Central States Collie Club*
Champaign Illinois Kennel Club
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Chapparral Bulldog Club, Inc.
Charleston Kennel Club, Inc.‡
Chemung County Federation of Sportsmen's Club*
Chesapeake Kennel Club
Cheshire Kennel Club, Inc.
Chicagoland Belgian Sheepdog Club
Chicagoland Shetland Sheepdog Club
Cleveland Animal Protective League*
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Coastal Crescent Kennel Club
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Empire Kerry Blue Terrier Club
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German Shepherd Dog Club of Greater Washington, D.C., Inc.
German Shepherd Dog Club of Louisiana, Inc.
German Shepherd Dog Club of San Diego County, Inc.
German Shorthaired Pointer Club of Idaho

*Gave $100–$499 since January 1, 1980.
†Gave $500–$999 since January 1, 1980.
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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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