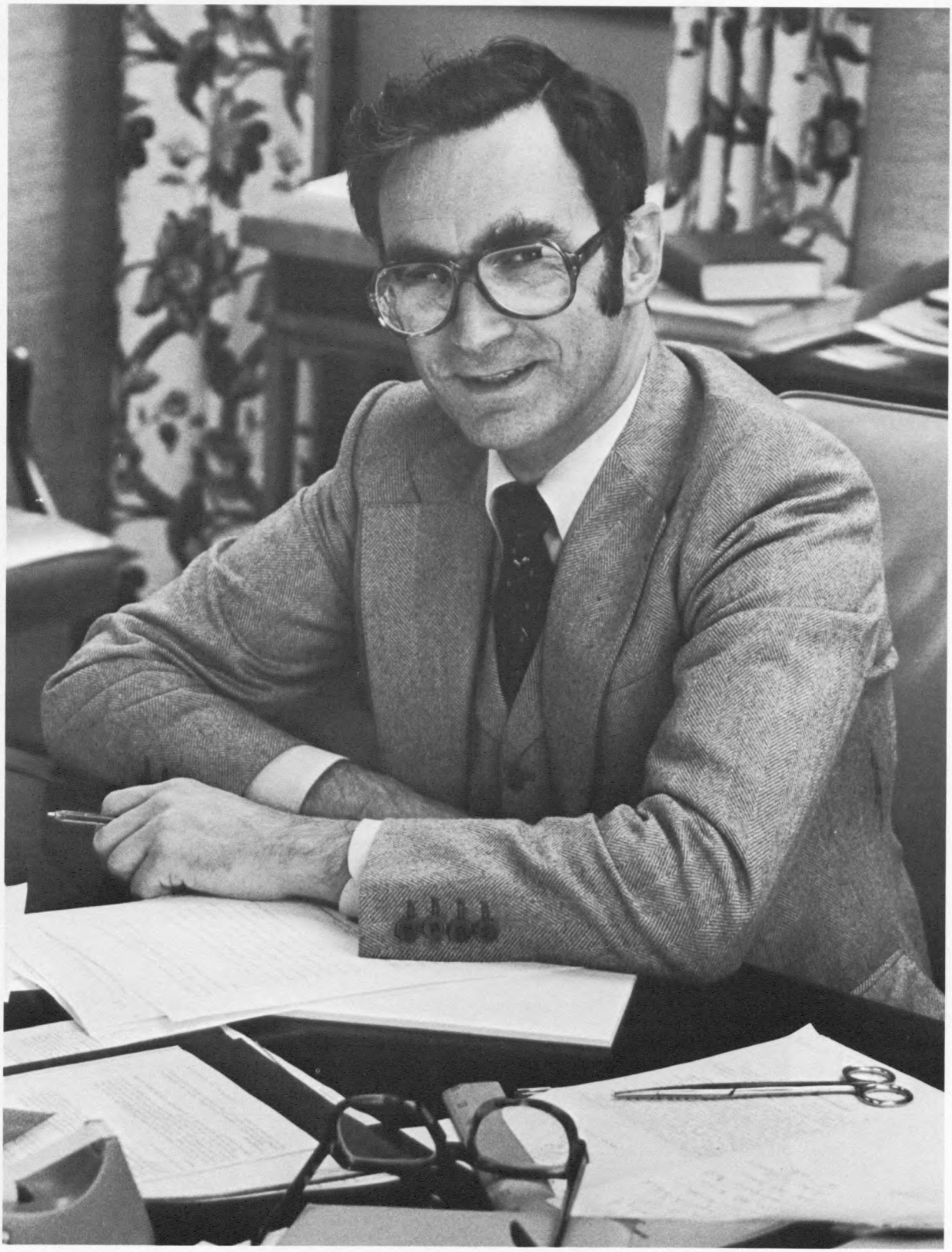
James A. Baker Institute for Animal Health
Cornell University ■ Annual Report 1983

New York State College of Veterinary Medicine Volume 33

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

We enter 1984 as a leaner and stronger institution.



A Message from the Director

Nineteen eighty-three was a year of significant accomplishments for the Baker Institute. New advances were made in both basic research and studies of diseases of dogs and horses. The full dimensions become apparent when one considers the background against which those results were attained.

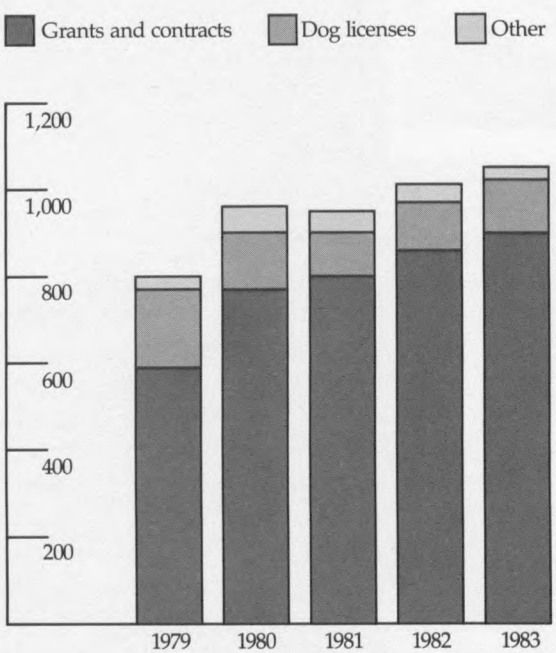
The impact of the recent recession was felt early in the year. Budget constraints obliged us to make further economies in our operations. Our electron microscopy laboratory was closed, and a number of capital improvement and maintenance projects were deferred. But our fortunes improved as the year progressed. New program support was provided by industry and the federal government, and recent investments in our facilities were rewarded in significant advances in research.

Much of our income for sponsored research is provided by grants and contracts. Our major sponsor is the National Institutes of Health. We also do research for the United States Department of Agriculture, foundations, associations, and other agencies that have an interest in animal health. Still other income for research is provided by gifts from individuals and dog clubs and by monies received by New York State through the dog license program. Of the amount collected for each license, ten cents is allocated to the Institute for health research. Although the license monies constitute only a small portion of the Institute's total operating income, they have contributed greatly to the health of the dog population. The Institute's role in developing vaccines for distemper, hepatitis, and parvovirus alone has saved countless lives and alleviated much suffering.

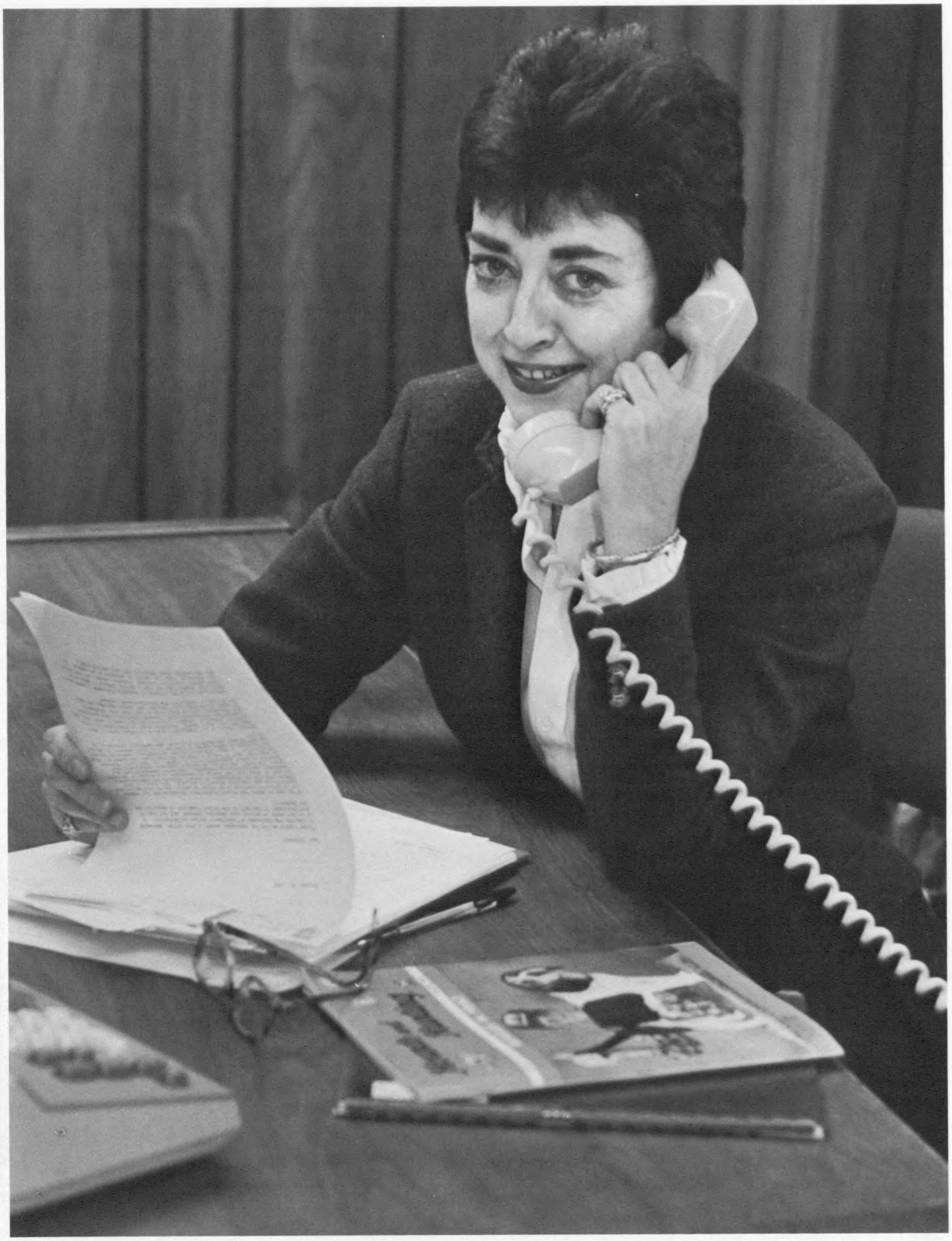
The American Kennel Club received the Arthur F. North, Jr., Canine Service Award this year. The award is made each year by the Institute to an individual or organiza-

Sponsored Research

(In Thousands of Dollars)



We have a well-equipped facility and a dedicated staff.

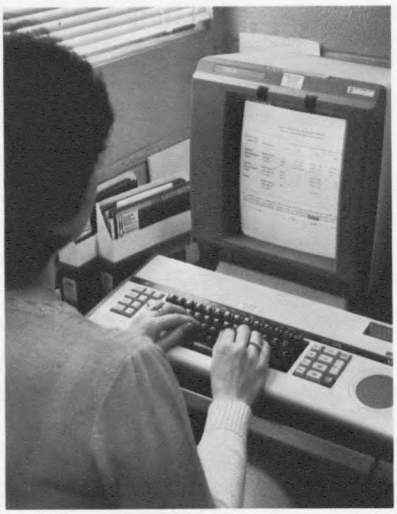


tion that has contributed in an extraordinary way to the improvement of the health and well-being of dogs. As recipient of the North Award, the AKC was cited for its contributions to purebred dogs and responsible dog ownership and its sponsorship of canine health research. We value our association with the AKC and its member clubs, and with the many individuals whose generosity has sustained the Baker Institute for more than three decades.

Our new programs in equine medicine received further impetus this year with the performance of the first successful equine embryo transplants at Cornell. The significance of that achievement is discussed elsewhere in this report. I mention it here because the embryo studies illustrate how our staff members are applying new techniques to the analysis of health problems. The opportunities are many, and the Institute intends to vigorously pursue research using embryo transplantation and other technological advances that hold promise for the future.

We enter 1984 as a leaner and stronger institution, resolute in our commitment to excellence and confident that our efforts will be rewarded in new insights into the nature of animal diseases and better methods for their treatment and prevention. Our optimism is firmly rooted in the knowledge that we have a modern, well-equipped facility, a dedicated staff of gifted men and women, a superbly capable advisory council, and a growing number of concerned benefactors. An institution like ours could wish for no finer assets.

Douglas D. McGregor
Director



Staff of the Baker Institute

Administration

Douglas D. McGregor, director: B.A., M.D., University of Western Ontario; D.Phil., Oxford University
Susan H. Hamlin, administrative manager: B.S., Elmira College
Kim T. Arcangeli, accounts assistant
Carlene M. Campbell, office assistant
Nancy D. Combs, accounts coordinator
Anita S. Hesser, secretary
Florence C. Huth, administrative aide
Ann W. Signore, administrative aide: Cornell

Laboratories

Giralda Laboratory for Canine Infectious Diseases

Leland E. Carmichael, John M. Olin Professor of Virology: A.B., D.V.M., University of California; Ph.D., Cornell
Roy V. H. Pollock, assistant professor of microbiology: B.A., Williams College; D.V.M., Ph.D., Cornell
Colin R. Parrish, graduate research assistant: B.Sc., Massey University
Jean C. Joubert, research technician
Priscilla H. O'Connell, laboratory technician: C.L.A., A.S.C.P.

Daynemouth Laboratory for Canine Nutrition

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

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

George Lust, professor of physiological chemistry: B.S., University of Massachusetts; Ph.D., Cornell
Nancy B. Wurster, research associate: B.A., M.S., Ph.D., New York University
Susan J. Harter, laboratory technician: B.S., Lock Haven State College

Hadley C. Stephenson Laboratory for the Study of Canine Diseases

Max J. G. Appel, professor of virology: Dr.med.vet., University of Hannover; Ph.D., Cornell
William J. Mitchell, Jr., graduate research assistant: D.V.M., Auburn University
Ching-chung Sheu, graduate research assistant: D.V.M., National Taiwan University; M.S., Taiwan University
Mary Beth Metzgar, research technician: University of Evansville

Donnelley Laboratory of Immunochemistry

Douglas D. McGregor, professor of immunology: B.A., M.D., D.Phil.
Judith Ann Appleton, research associate: B.S., Indiana University; M.S., Ph.D., University of Georgia
Melissa C. Woan, research associate: B.Ed., Taiwan Normal University; M.S., Ph.D., University of Illinois
Daniel H. Sajewski, graduate research assistant: B.A., Cornell
Lori S. Best, laboratory technician: B.S., Elmira College

Kristie E. Schmidt, laboratory technician: B.A., University of California, Santa Cruz
Laura M. Stenzler, laboratory technician: A.A.S., State University of New York Agricultural and Technical College, Delhi; 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
Ching-hua Wang, graduate research assistant: M.D., Peking Medical School
Lincoln S. Adams, research technician: B.S., Hobart College; AALAS accreditation
Cheryl S. DiDomenico, laboratory technician: B.S., Middle Tennessee State University
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
Anne L. Crump, graduate research assistant: B.A., Mount Holyoke College
Christopher J. Davies, graduate research assistant: B.S., D.V.M., Cornell
Jane M. Miller, laboratory technician: B.S., Cornell
Jeannette C. Poleman, laboratory technician: B.A., Cornell

Colgate Tissue Culture Laboratory

Stephanie M. Oberhaus, laboratory technician: B.A., Goucher College

Glassware Department

Jeannette R. Carney, laboratory attendant
Rodney R. Riker, laboratory attendant

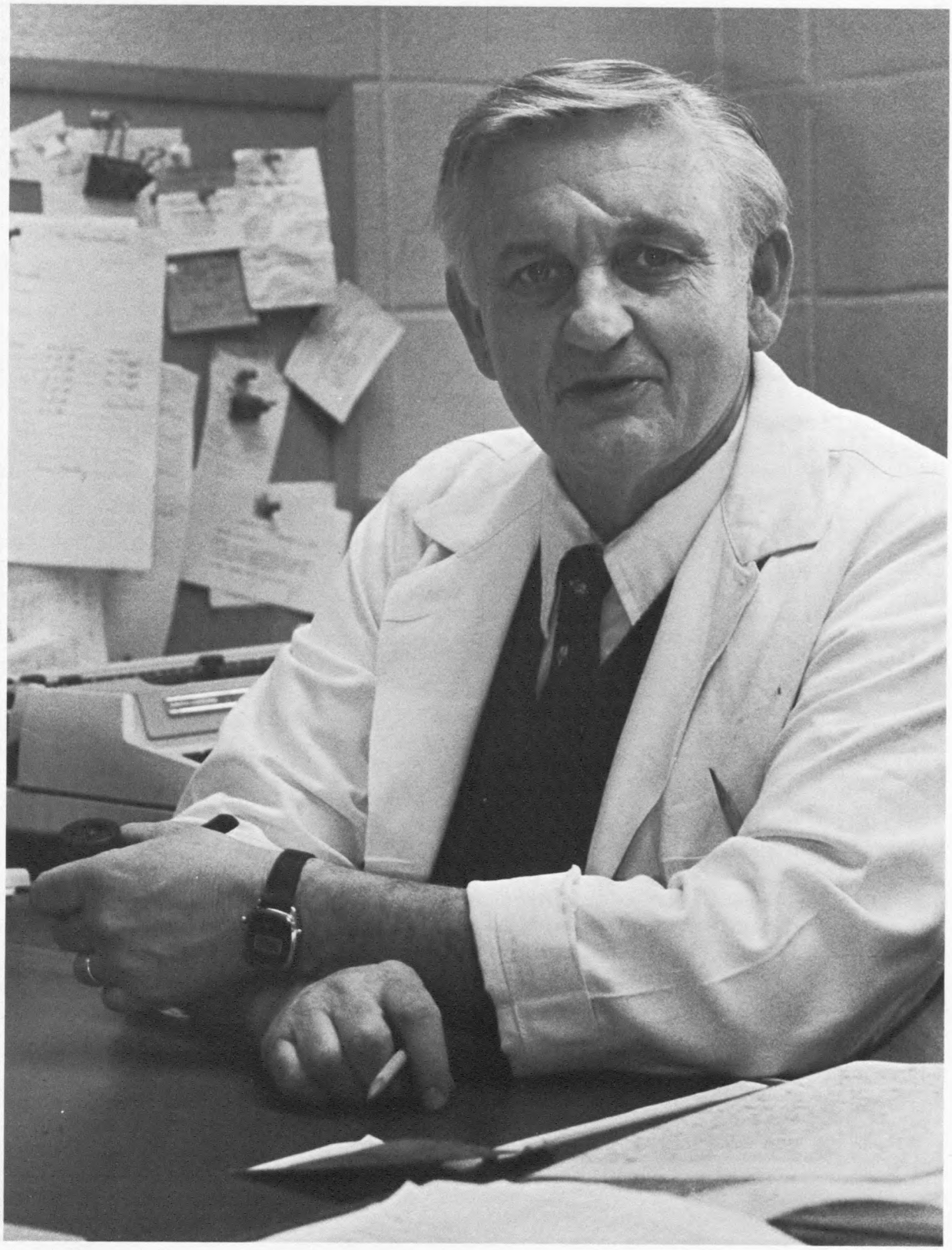
Animal Care

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, Delhi; AALAS accreditation
James C. Hardy, animal technician: B.S., Cornell; AALAS accreditation
Laura A. Michel, animal technician: A.A.S., State University of New York Agricultural and Technical College, Farmingdale; B.S., Cornell; AALAS accreditation
Anastasia E. Newell, animal technician
Gerald G. Rice II, animal technician

Maintenance

Edson Wheeler, Jr., maintenance supervisor
Arthur D. Howser, maintenance mechanic
Gerald G. Rice, vehicle mechanic

We are addressing the complex problems of viral pathogenesis and genetics.



Giralda Laboratory for Canine Infectious Diseases

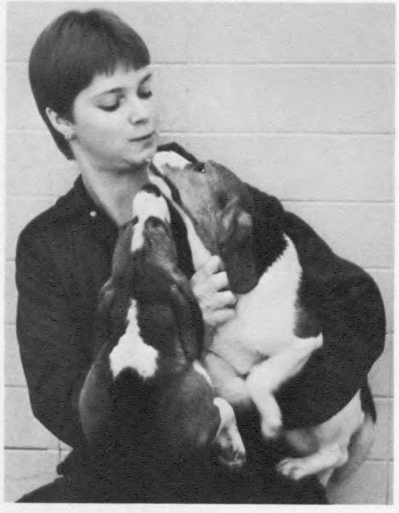
Work in our laboratories continues to focus on canine parvovirus (CPV) and canine brucellosis caused by *Brucella canis*. Although we maintain a strong commitment to questions of concern to veterinarians and dog breeders, we have extended our work to address the more complex problems of viral pathogenesis and genetics. Contemporary methods of molecular biology are being applied to CPV, as well as to the closely related parvoviruses that cause disease in cats and mink. We are also making progress in developing reliable methods to identify dogs that have been infected with *Brucella canis*.

Research during the past year led to several discoveries important to understanding the natural history of CPV infection and the reasons for immunization failures. Previous work defined the critical period in the life of a pup during which maternal antibodies are still high enough to block responses to CPV vaccines, but are too low to prevent infection by virulent virus. That period has been shown to extend as long as five months in pups born to bitches that suffered infection during the previous year. The phenomenon underscores the importance of kennel management in controlling CPV disease. Pups must be isolated from CPV until they reach the age when they can be successfully immunized.

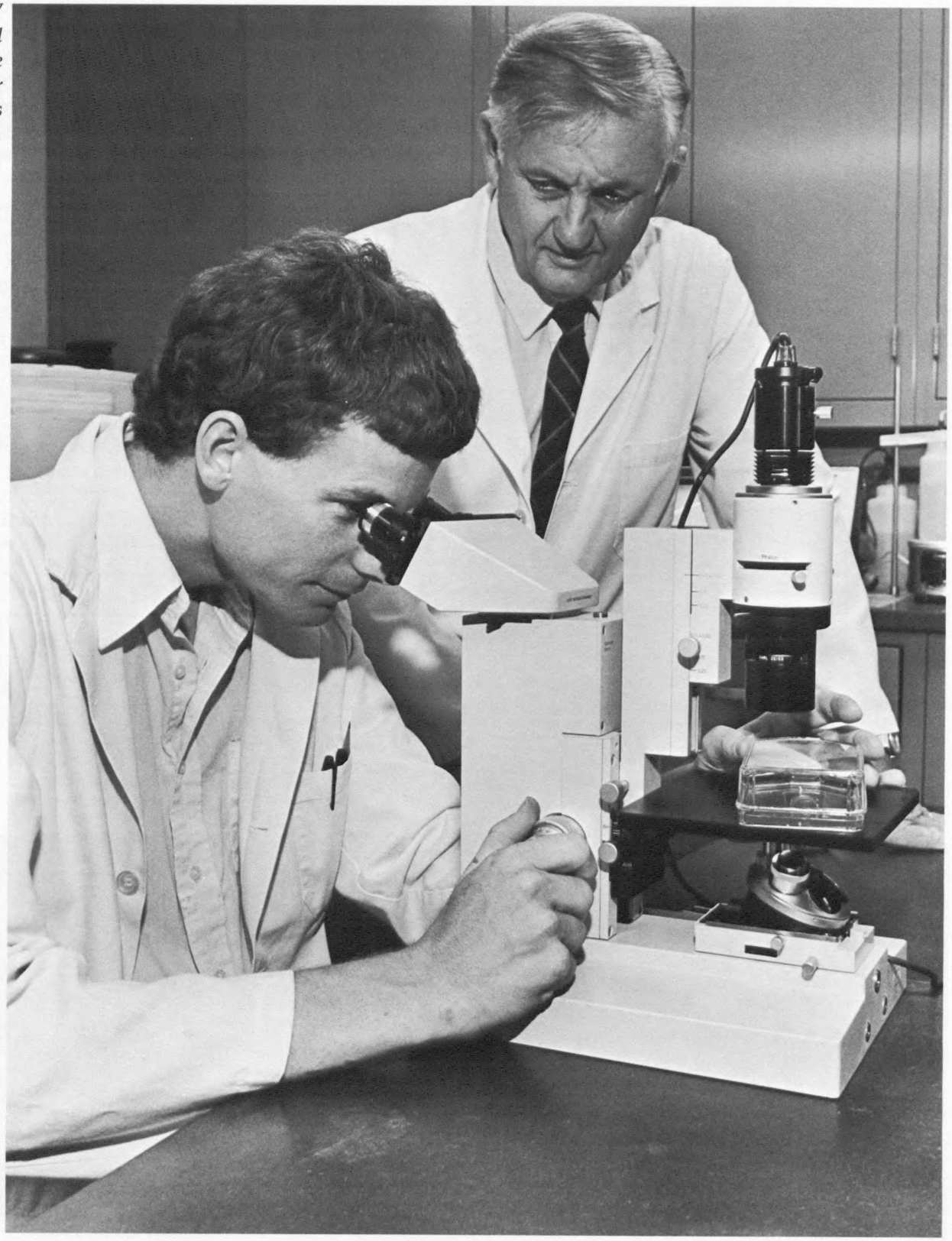
Another critical factor in the control of CPV is the availability of effective vaccines. We found that several commercial vaccines failed to immunize young dogs in which maternal hemagglutination-inhibiting antibody titers were very low ($\leq$1:10). A number of the dogs were nearly nineteen weeks old at the time of vaccination. Their poor response was unexpected, because an experimental attenuated living CPV vaccine (A-CPV) prepared in our laboratory successfully immunized more than 60 percent of pups with similar, or even higher, antibody titers. Further study revealed differences in the biological characteristics of the vaccine virus strains and different amounts of virus in the four lots tested. When a variant virus population predominated, the minimal immunizing dose of the vaccine was a thousand times greater than a vaccine containing only the original seed virus.

Study of the CPV variants in attenuated live virus vaccine stocks and those variants that develop after growth in cell cultures is a principal focus of current research. Such variants were anticipated, for earlier studies revealed the emergence of variant viral populations in an experimental A-CPV when the virus was propagated in feline or mink cells.

We are also exploring the possibility that CPV can suppress the immune system, rendering infected dogs vulnerable to other infectious agents. Preliminary experiments in disease-free dogs given virulent CPV indicated only a slight and transient (one-day) decrease in cell-mediated immunity, as measured by lymphocyte blastogenesis. Responses were normal in littermates given high doses of CPV vaccine. Further studies using different tests of immune function are in progress.



Perhaps CPV renders infected dogs vulnerable to other infectious agents.



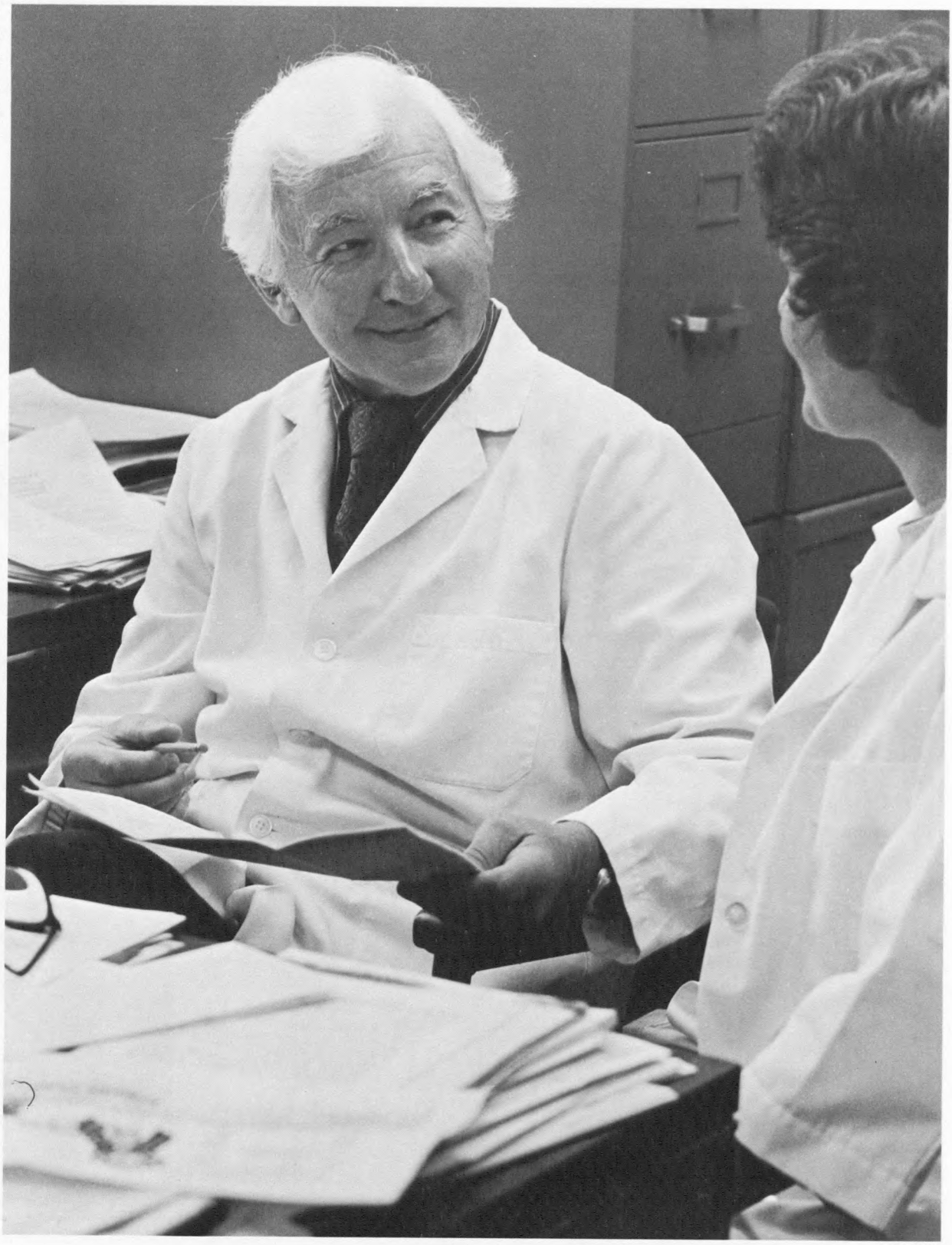
Studies of CPV and the closely related feline panleukopenia virus (FPV) and mink enteritis virus (MEV) were continued. The three viruses are being compared with respect to their host range, antigenic structure, and capacity to cause disease. Biochemical, molecular biological, and immunological methods are being used to identify and define the properties of those viruses that account for differences in their host range and pathogenicity. CPV, FPV, MEV, and a closely related virus of raccoons were distinguished by a panel of monoclonal antibodies. A significant finding was the antigenic variation observed both within and between the parvoviruses of each species. Although natural isolates of CPV were similar, MEV isolates could be grouped into three antigenic types. Analysis of the variant parvoviruses revealed that the surface of the virions contain at least two determinants, each comprising many different but overlapping epitopes.

Several recombinant and mutant strains of CPV and FPV have been prepared. They will be used to determine the roles of the viral genes and structural components of the virus in the viral replication cycle in cell cultures and in their respective animal hosts and to map DNA sequences that code for expressed antigenic and host-range differences. Similar methods are being used to analyze the extent of natural variation among isolates of the various parvoviruses.

The above studies are steps toward our long-term objective of defining the mechanisms by which these small, simple viruses vary in host range, pathogenicity, antigenic type, and virulence.

Leland E. Carmichael

Overfeeding and obesity are increasingly common problems in dogs.



Daynemouth Laboratory for Canine Nutrition

We reported last year that cellular responses were less in old dogs than in young adult dogs, and that the in vitro proliferative capacity of lymphocytes of old dogs was significantly stimulated by the addition of vitamin E and selenium to the cultivation medium. Studies now completed tested whether such responses could be stimulated by increasing old dogs' dietary vitamin E and selenium intake levels above those recommended by the National Research Council. No clear evidence for recommending such supplementation was found.

Past research established that when amino acid requirements for early growth are met, dogs require only thirty grams of protein per thousand calories of metabolizable energy (ME) consumed. Our current studies, conducted over the entire growth period from weaning to early adulthood, and using the average growth rate for the breed as the principal criteria of sufficiency, confirmed that minimum requirement. Comparisons of average synthesis and growth of red blood cells (RBC), demonstrated by packed cell volume, hemoglobin values, and total serum protein, also confirmed the protein requirement.

That requirement represents about 12.8-percent protein content in a diet supplying 4.0 calories of ME per gram of food. Since the average commercial dry dog food supplies about 3.5 calories of ME per gram, only 11.2 percent of protein is needed if the protein is of a quality equivalent to that of whole egg. If the protein is of a quality similar to that in commercial foods today, namely 80 percent of egg protein, then 14 percent would be required.

Rate of growth and level of RBC production were greater when the amino acid content of the food was increased by 30 percent, to a level equivalent to 16.6-percent protein. That corresponds to a present-day commercial dry dog food containing about 18-percent protein.

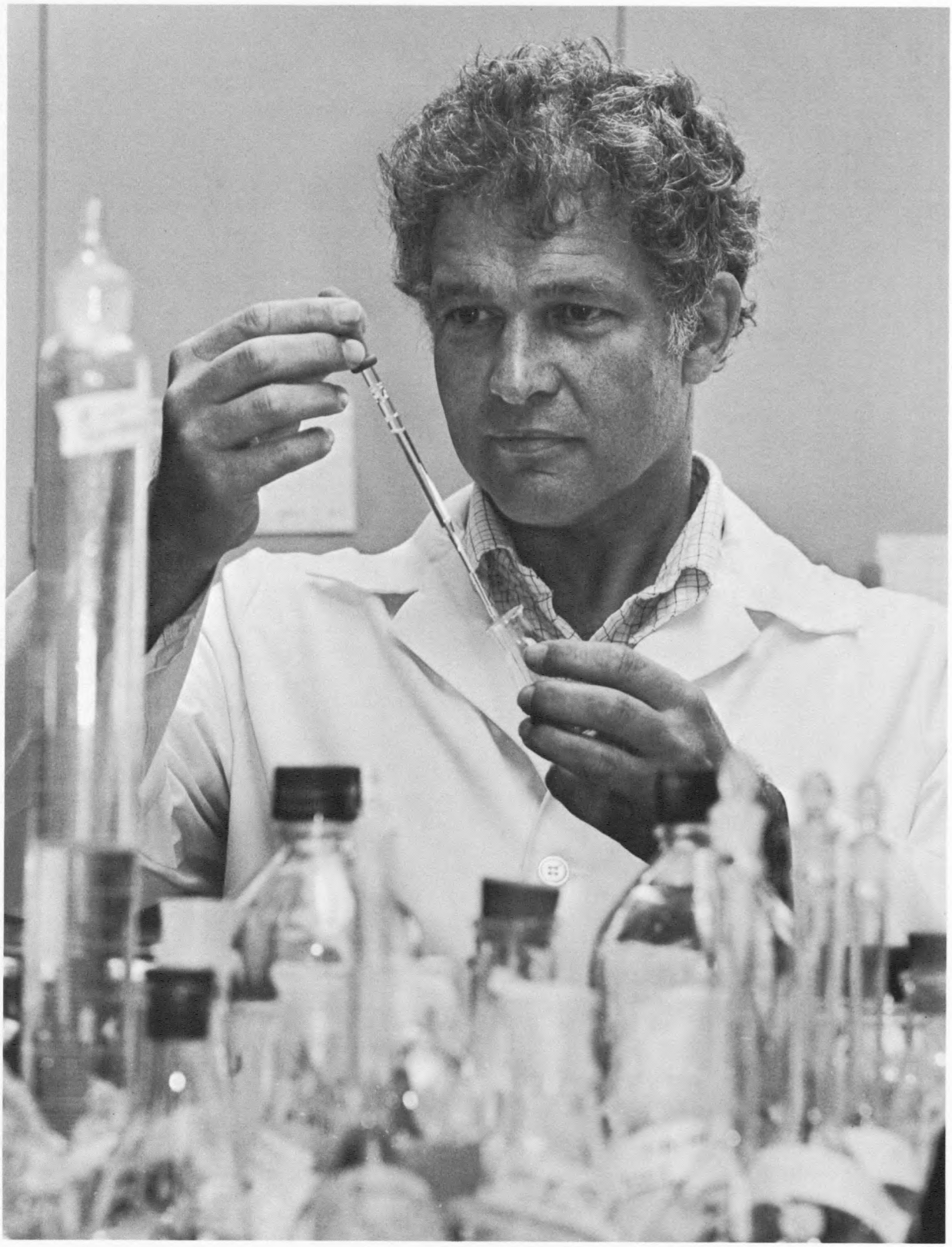
The significance of the faster growth rate and increased hemoglobin level on the health and well-being of dogs during growth and in later life remains to be shown. Earlier studies at Cornell demonstrated that maximal size for age resulting from a nutritionally induced rapid growth rate was not conducive to good skeletal development. Great Danes as well as Labrador Retrievers fed to achieve maximal growth rates developed anatomical changes similar to hypertrophic osteodystrophy and other early signs of skeletal degenerative disease. Additionally, among dogs with high parental frequency of hip dysplasia, those fed to increase the growth rate developed more severe disease at earlier ages than littermates fed to achieve only average growth rate for their breed.

Thus it seems the protein content of commercial dog foods may be significantly lowered, particularly if the protein quality is improved. Those changes, both in foods for growing dogs and foods for dogs at maintenance, should result in greater feeding efficiency without adverse effect. There are of course stress situations where greater protein intake is beneficial, such as after infection or surgery or during strenuous work.

Overfeeding and resulting obesity are increasingly common problems in dogs. We investigated the potential use of "starch blockers" to decrease digestibility of commercial dog foods and thus reduce energy intake of dogs. Addition of starch blockers to dog food at levels recommended for humans had no adverse effect on gastrointestinal function or stool form. Nor did it depress digestibility of the carbohydrates in the food. Total energy available was unchanged. Weight loss, it appears, can result only from a reduction of caloric intake and an increase in exercise. Dogs are no different from their masters.

Ben E. Sheffy

There is more fibronectin in the cartilage and synovial fluid of osteoarthritic joints.



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

A new observation of some importance was made in our studies of osteoarthritis and hip dysplasia in dogs. There is more of the protein fibronectin in the cartilage and synovial fluid of osteoarthritic joints than normal joints. The accompanying table shows the average values (and the ranges) for cartilage and synovial fluid samples obtained from normal and osteoarthritic hip joints.

Fibronectin Content of Cartilage and Synovial Fluid

	Cartilage (μg/mg)	Synovial Fluid (μg/ml)
Disease-free joints	0.07 (0–0.10)	99 (70–146)
Osteoarthritic joints	3.19 (0.60–7.01)	206 (84–460)

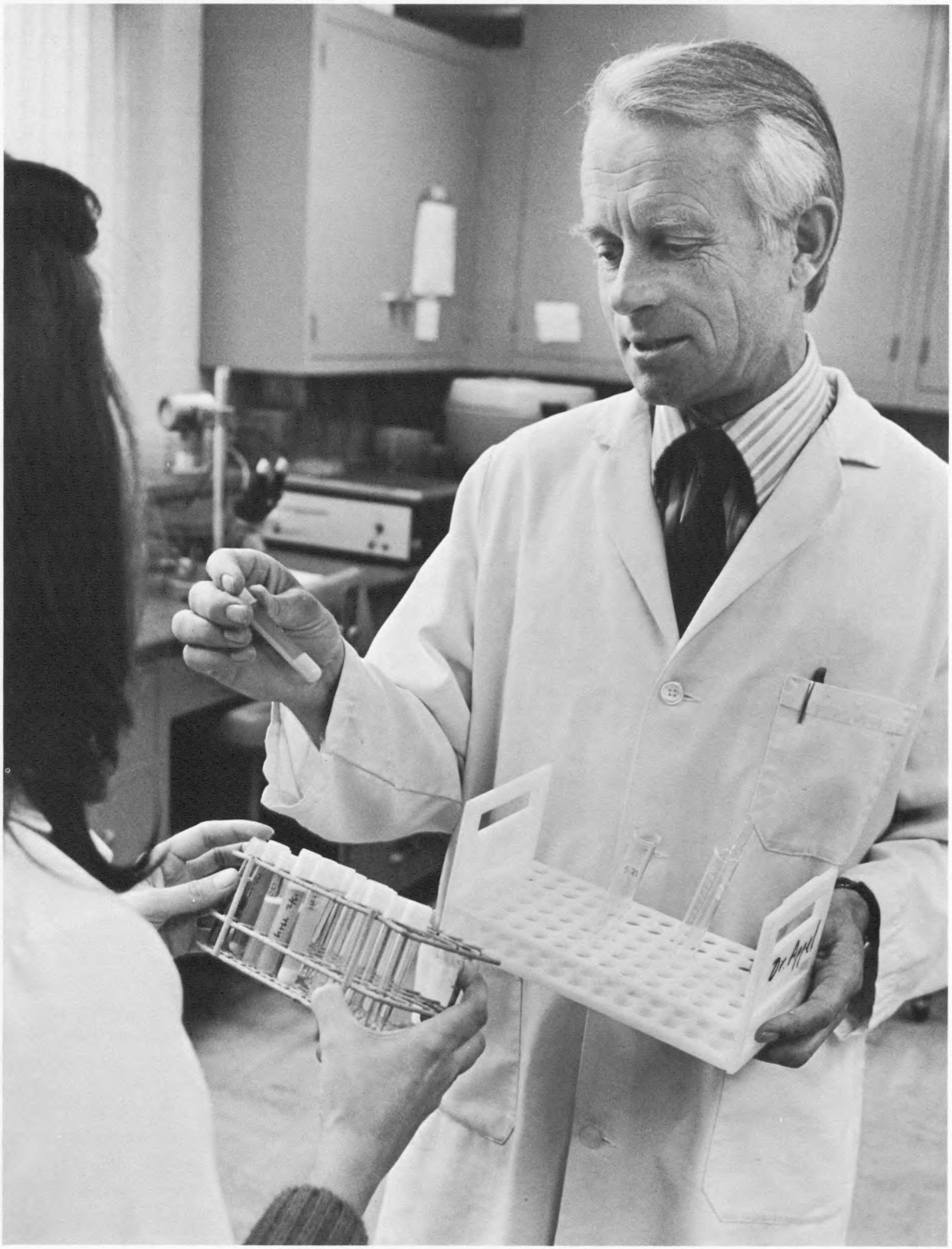
Fibronectin is a normal constituent of the blood and many tissues. It is thought to have a variety of biological functions. Although we do not yet know why there is more fibronectin in osteoarthritic joints, it is tempting to speculate that the protein is involved in some way in the degenerative process. In any case, its presence in increased amounts in cartilage and synovial fluid is an objective indicator of joint disease. On the basis of those findings, we are evaluating the possibility that fibronectin levels in joint fluid can be used to monitor the effects of drugs and procedures whose purpose is to arrest or slow the osteoarthritic process.

We are currently investigating the origin of the fibronectin in the synovial fluid of diseased joints. Is it formed in the cartilage or derived from the blood? The protein is produced in the cartilage of osteoarthritic joints five times faster than in disease-free cartilage. That finding suggests that at least some of the fibronectin in synovial fluid originates in the cartilage.

We also want to determine the biochemical composition of the fibronectins in cartilage and in blood. A difference between those proteins could provide the basis for a diagnostic test of osteoarthritis in dogs.

George Lust

The result of our studies should be a better CDV vaccine.



Hadley C. Stephenson Laboratory for Study of Canine Diseases

Vaccines for the prevention of viral diseases are often the product of many years of careful study and testing. A major objective is to identify constituents of viruses capable of inducing an immune response that protects against infection. This year we undertook such a study of canine distemper virus (CDV). We immunized dogs with an antibody to a single CDV antigen and demonstrated that those animals were protected against distemper. We are continuing our analysis by determining how much antibody is required to confer protection, when during the course of infection an injection of the antibody can modify disease, and whether immunity to CDV can be generated by antibodies to other antigenic constituents of the virus. The result of our studies should be a better CDV vaccine.

Vaccination of some carnivores—for example, red pandas, black-footed ferrets, and kinkajous—with modified live CDV vaccine can result in fatal infections. We have provided the National Zoo in Washington with inactivated CDV vaccine for those species.

We found that cell-mediated immunity is a reliable indicator of the course of a CDV infection. Dogs that develop acute encephalitis usually have little or no cell-mediated immunity, whereas those that recover from a CDV infection have the highest levels. Reduction, or delay in the onset, of cell-mediated immunity is often associated with persistent infection by CDV.

We continued our research on how CDV causes persistent infection and brain disease in dogs. In cooperation with Dr. Brian Summers, from the Department of Pathology, we demonstrated that chronically infected dogs harbor CDV in uveal tissue of the eye. Inflammation of the uvea, or uveitis, is often a feature of multiple sclerosis, a pathologically similar disease in human beings. Our finding has encouraged others to search for a viral cause of multiple sclerosis in the uveal tissue of patients.

Dr. Summers and Dr. Jeffrey Mitchell demonstrated that certain cells of the brain, called astrocytes, can be infected with a strain of CDV known to cause persistent infection. The virus persisted in astrocyte cultures for several weeks without causing cell death. That finding may explain how CDV can continue to exist in the brain after it has been eliminated from most tissues.

Interferon has attracted much attention because of its potential use in treating viral infections and cancer. Efforts to identify a nontoxic inducer of interferon have been frustrated because repeated administration of interferon inducers results in progressively feeble responses. We studied that phenomenon in dogs and found that macrophages and their secretions inhibit interferon induction.

We continued our studies of infectious diarrhea in dogs. Canine parvovirus and canine coronavirus are the most frequent causes of that disease. However, we demonstrated that canine rotavirus can cause a mild infection and diarrhea in newborn susceptible laboratory dogs. Further studies will be required to ascertain the significance of rotavirus infection as a cause of diarrhea among dogs reared in conventional kennels or as house pets.

Max J. G. Appel

Milk confers on suckling animals a powerful protection against infection.

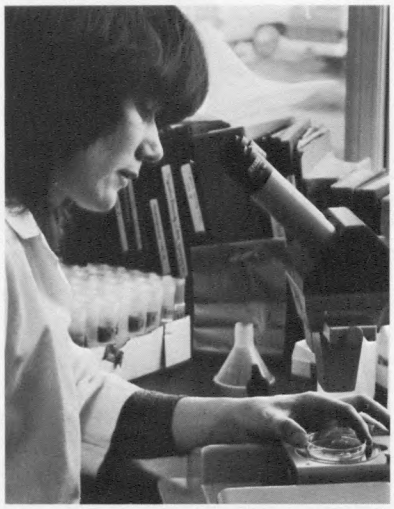
Donnelley Laboratory of Immunochemistry

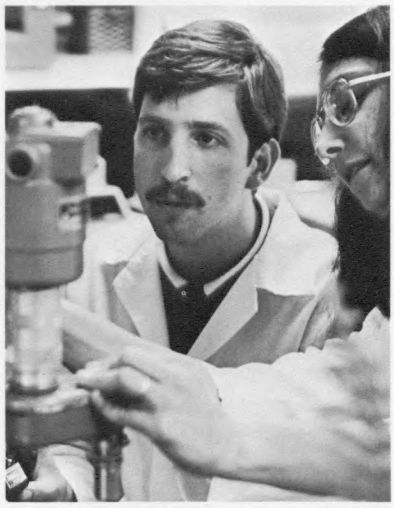
Recovery from infection with *Listeria monocytogenes* (LM) depends on the host's cellular immune response to antigens of that bacterium. Our studies point to thymus-dependent lymphocytes, or T cells, as the mediators of host resistance to LM. During the past year we demonstrated that the protective activity is a property of a subset of T cells. That subset was identified by monoclonal antibodies. We were able to maintain LM-protective cells in culture for a time by repeatedly stimulating the cells with LM antigens. However, the cultured cells eventually lost their ability to protect normal animals against an LM infection. We are now investigating whether the LM-responsive cells from such long-term cultures can regulate the immune response to infection.

We have also been studying the immunity transferred to suckling animals in their mother's milk. We found that young rats born to and suckled by rats infected with the parasite *Trichinella spiralis* are protected from infection with that parasite. We know that the protection is conferred by a component of the milk, because rats born to infected rats but suckled by uninfected rats are susceptible to the infection. The protection conferred by milk is capable of eliminating 95 percent of a challenge dose within just a few hours.

It appears that antibodies from the blood of the mother rats can provide the same protection when fed to suckling rats. That finding confirms that antibodies in milk protect suckling rats from infection with *Trichinella spiralis,* underscoring the importance of mothers' milk to the health of young animals and human beings.

Melissa C. Woan
Judith A. Appleton





Our experiments show a complex interaction between different parasites.

Oswald R. Jones Laboratory of Immunology

Our laboratory remains committed to a basic biological and immunological analysis of the relationship between hosts and parasites, particularly intestinal helminths.

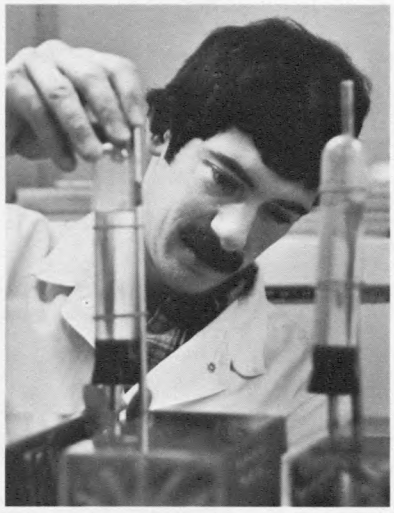
During the past year we introduced a new parasite, *Trypanosoma musculi,* into our studies. That parasite is related to the trypanosomes that cause sleeping sickness and Chagas' disease in human beings and to the trypanosomes that prevent effective utilization by cattle of large segments of prime grazing land in Africa. The economic and human costs are enormous. We are examining the basic host-parasite interaction, thereby increasing our understanding of trypanosomal infections of animals and human beings. We are also examining the effects on the host and parasite of two concurrent infections—with the intestinal nematode *Trichinella spiralis* and with the trypanosome.

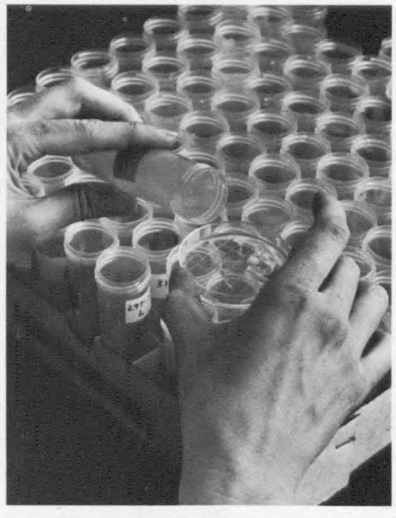
Since many individuals in natural populations harbor parasites of several different species, it is important to know how those parasites influence the host and each other. Some parasites impair the host's ability to respond immunologically to other infectious agents. Immunosuppression can occur even when the primary infection is not apparent.

Our experiments show a complex interaction between different parasites. The outcome of infection is influenced by the species of parasites, the timing of the infection, and the genotype of the host. The results have important implications for the control of infections in natural environments. We are also learning about the evolution of resistance in animal and human populations in circumstances where multiple infections are common.

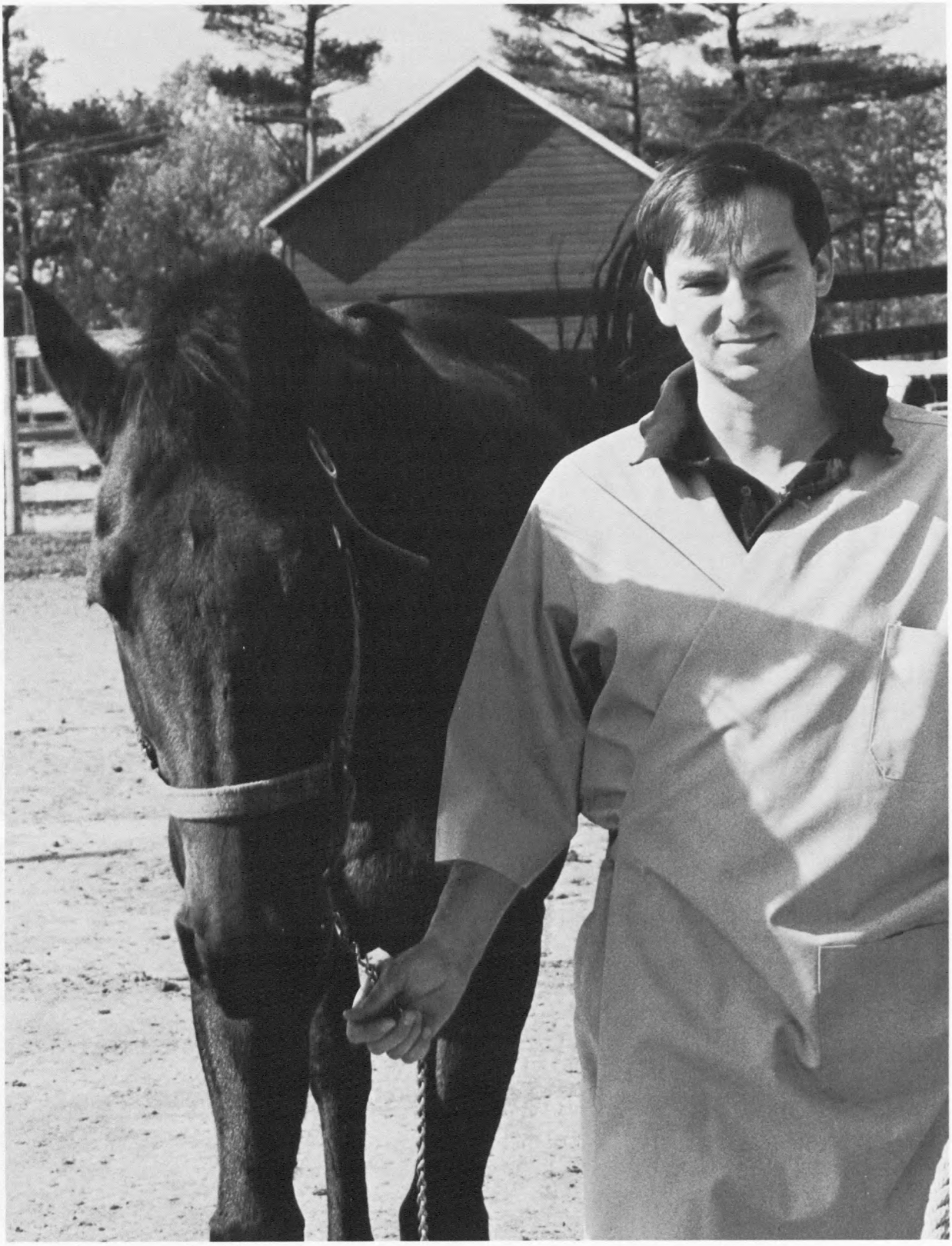
Our continuing studies of the genetics of resistance to *Trichinella spiralis* have disclosed that the major determinants of intestinal rejection are genes that are not linked to the major histocompatibility complex. We have identified three patterns of worm expulsion in mice: strong, intermediate, and weak. Each type is genetically heterogeneous. Furthermore, the pattern can vary depending on the infectious dose of worms. Those results disclosed an unsuspected diversity in the genome of the mouse, implying that there are several distinct processes or targets of worm expulsion. Experiments now under way are directed at determining the number of genes involved. We also want to map the chromosomal location of those genes through classical linkage studies.

Robin G. Bell





We are studying the relationship between equine histocompatibility antigens and pregnancy.



Immunogenetics Laboratory

We have established a comprehensive program for long-term genetic investigations of the horse, of both theoretical and practical interest. The research is designed to provide fundamental knowledge of the genetic control of immune responses in the horse and to produce an immunogenetic testing service for the equine industry in New York State. We have concentrated on studies of the equine lymphocyte antigens (ELAs), which are probably the major histocompatibility complex (MHC) molecules of the horse. The MHC controls a number of immune responses in both animals and human beings. Of particular importance during the past year were our studies of the relationship between equine histocompatibility antigens and pregnancy.

In 1981 our laboratory organized the First International Workshop on Lymphocyte Alloantigens of the Horse. A second workshop on the subject was held in Ithaca last year. Both were funded by the Dorothy Russell Havemeyer Foundation. The workshops defined ten ELAs. Antisera prepared in our laboratory can define all those ELAs and five others, as well as the ITH-12 antigen, which is not linked to the ELA region.

Six new monoclonal antibodies to equine lymphocyte antigens were produced by cell hybridization. Those antibodies recognize at least three different molecules. One of the molecules represents a new genetic marker for horse lymphocytes. The new marker will add to the discriminating capacity of the conventional typing sera.

For the third year in a row we performed ELA typing on yearlings from a large standardbred farm in New York State. The aim of the service is to confirm the pedigrees of the yearlings before they are sent to the yearling sales. We expanded our horse-typing service by adding testing capabilities for five serum enzyme polymorphisms. Those systems are all used by other laboratories offering a blood-typing service for horses. The expansion of our services was accomplished through collaboration with Dr. Bernard May of Cornell's Laboratory for Ecological and Evolutionary Genetics.

We also responded to requests from veterinary practitioners in need of blood typing in cases of suspected hemolytic disease of the newborn foal. We tested sera from pregnant mares for the presence of antibody to red-cell antigens of the stallions to which they were mated. Such antibodies can bind to the foal's red blood cells, releasing their content of hemoglobin and causing serious, or even fatal, illness. About 1 percent of foals are at risk.

During the past five years we have pioneered studies of the strong immune responses in mares against their developing fetuses. That response has many characteristics in common with reactions to organ transplantations in human beings that result in the rejection of the organ. The consequences of the maternal immune response to the equine fetus are not known. However, with a foaling rate of about 60 percent, the horse is probably the least fertile of the major species of farm animals. Furthermore, it has been estimated that

The lab responded to requests from veterinarians in need of blood typing.

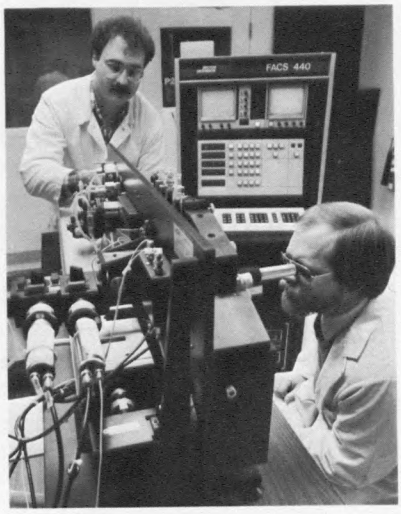
about 10 percent of equine pregnancies abort or resorb about the time antibodies to fetal antigens first appear in the mare's serum.

Our observations and expertise in equine histocompatibility testing encouraged us to pursue these studies in collaboration with Dr. W. R. Allen, an internationally recognized authority on equine reproduction. That collaboration is now in its third year. It has encouraged several exchanges of personnel between the Baker Institute and Dr. Allen's unit in Cambridge, England, and considerable progress has been made. The results may soon be applied to problems of concern to horse breeders.

In the course of our investigations we developed a new model for studying the differences between the maternal immune responses to fetal histocompatibility antigens made by horses and donkeys. Those differences could be important to our understanding of normal horse pregnancy and the many causes of infertility in mares. The idea is to use female mules as recipients for horse or donkey embryos. The development of the embryos can thus be compared in a neutral maternal environment. Our first transplants using mules as recipients were performed in the summer of 1983. At the time of this writing we have five mules pregnant by embryo transfer. Four are carrying horse embryos, and one is carrying a donkey.

Those experiments represent the first equine embryo transfers performed at Cornell, and possibly the first embryo transfers using mules as recipients ever performed. They have already demonstrated that although the mule is a sterile hybrid, its reproductive tract is capable of supporting a pregnancy, at least through the first third of gestation. We do not know if the horse or donkey embryos will be carried to term. One mule carrying a horse embryo has already made a strong immune response to her fetus. The outcome of the embryo transfers will form the basis of experiments in our laboratory during the coming year.

Douglas F. Antczak



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.

567 **Carmichael, L. E., J. C. Joubert,** and **R. V. H. Pollock.** 1983. A modified live canine parvovirus vaccine. II. Immune response. *Cornell Vet.* 73:13–29.

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The Institute is deeply committed to biotechnology research.

The Institute in Perspective

As one reads this report, one is impressed with the remarkable advances in health research made possible by the new methods being used in studies of animal diseases at the Institute. At our annual meeting in Ithaca this year we heard how our staff scientists are unlocking the genetic information in viruses to better understand how those agents cause disease, and how the nucleic acid that carries the information can be modified and rearranged in useful ways.

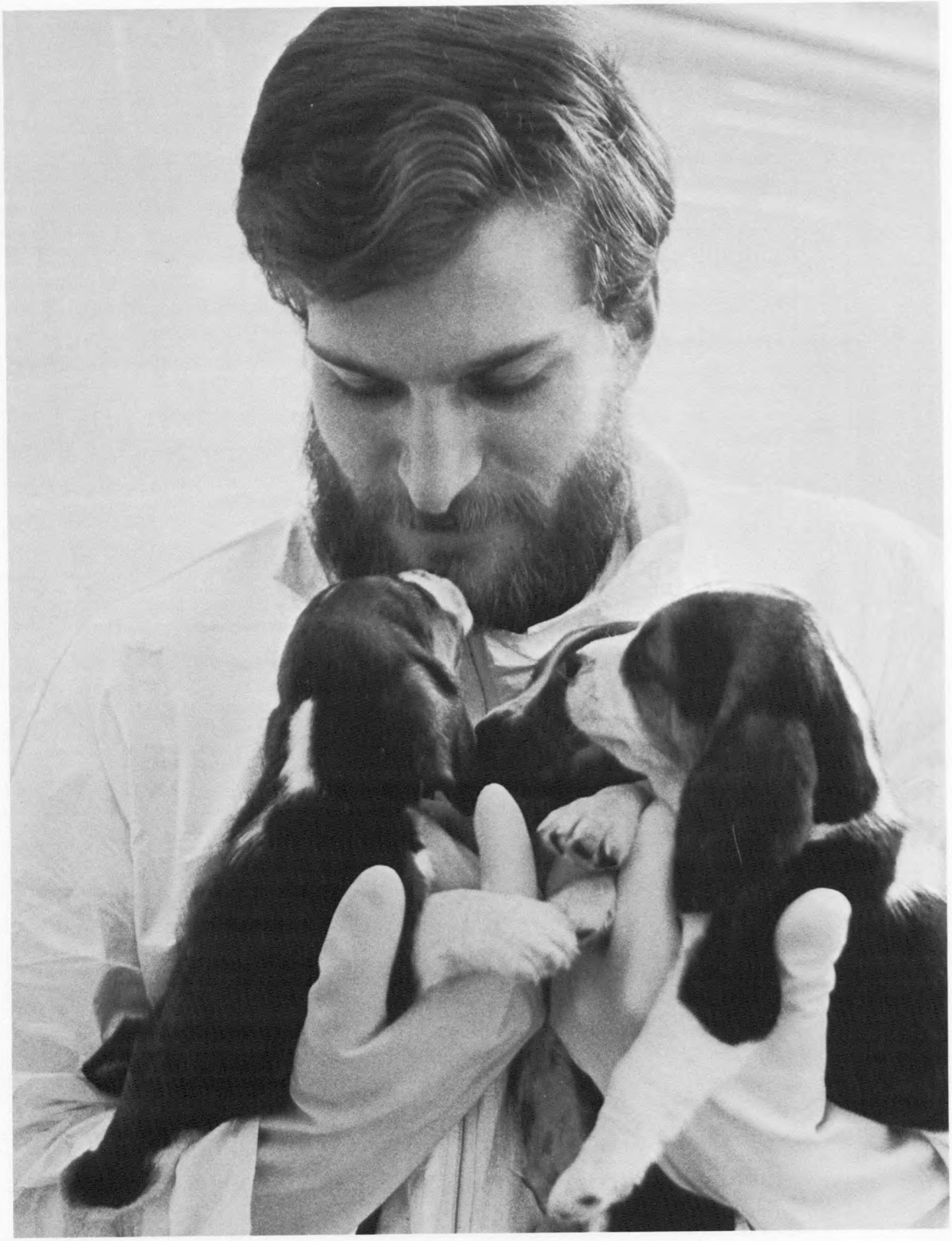
We also heard how antibodies can be tailor-made for the diagnosis and treatment of illness, and how such antibodies can be used in conjunction with new advances in laser and computer technology to reveal valuable information about how our immune system is mobilized to defend us against a myriad of agents and toxins that provide a threat to our health.

Finally, and perhaps most remarkably, we learned how studies of the developing embryo are shedding new light on pregnancy. The immediate benefit of that research will be to improve the reproductive efficiency of horses, and even more remarkable advances are close behind. They include the improvement of domestic stock, the propagation of endangered species, and the early detection and even correction of certain genetic abnormalities.

Gene splicing, flow cytometry, cell fusion, embryo transplantation. That is the language of biotechnology. I am gratified to report that the Institute is deeply committed to this research, and as a result of its efforts one can expect a continuing flow of new information and products that will provide a better life for our animals and for us.

Dorothy R. Donnelley
Chairwoman

Gifts enable us to carry out our mission.



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. and Mrs. Robert S. Boas, Mr. Albert C. Bostwick, Jr., Mr. Alan P. Cusick, Mr. and Mrs. Gaylord Donnelley, Mrs. N. Clarkson Earl, Jr., Mrs. Priscilla Maxwell Endicott, Mrs. Pamela Firman, Mrs. Gordon H. Gillis, Mr. R. L. Ireland III, Mr. and Mrs. David J. McFadden, Mr. George A. Motter, Mrs. Adelaide C. Riggs, Mr. William Rockefeller, Mrs. Richard M. Scaife, Mrs. Anita E. Seits, Mr. Jack B. Ward, Mr. and Mrs. Harwood Warriner, Mr. Gilbert H. Wehmann, Mr. Robert Winthrop, and Mr. Robert W. Woodruff.

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