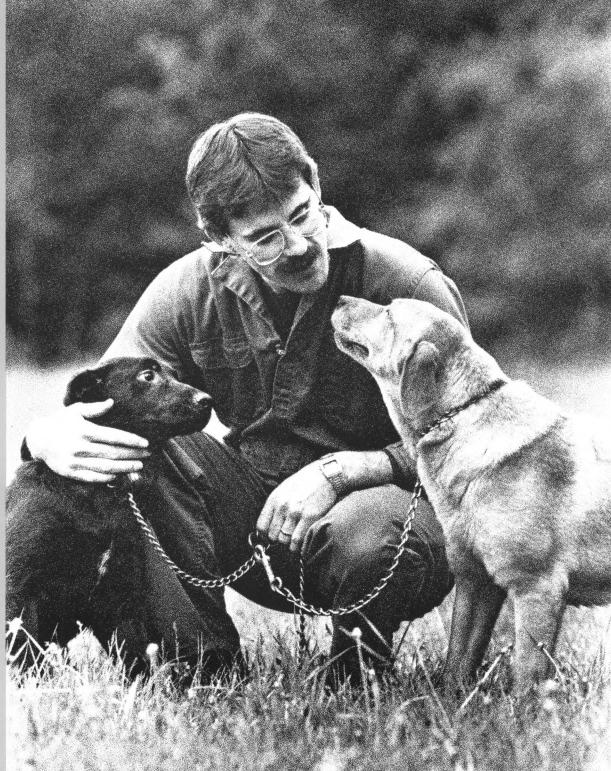
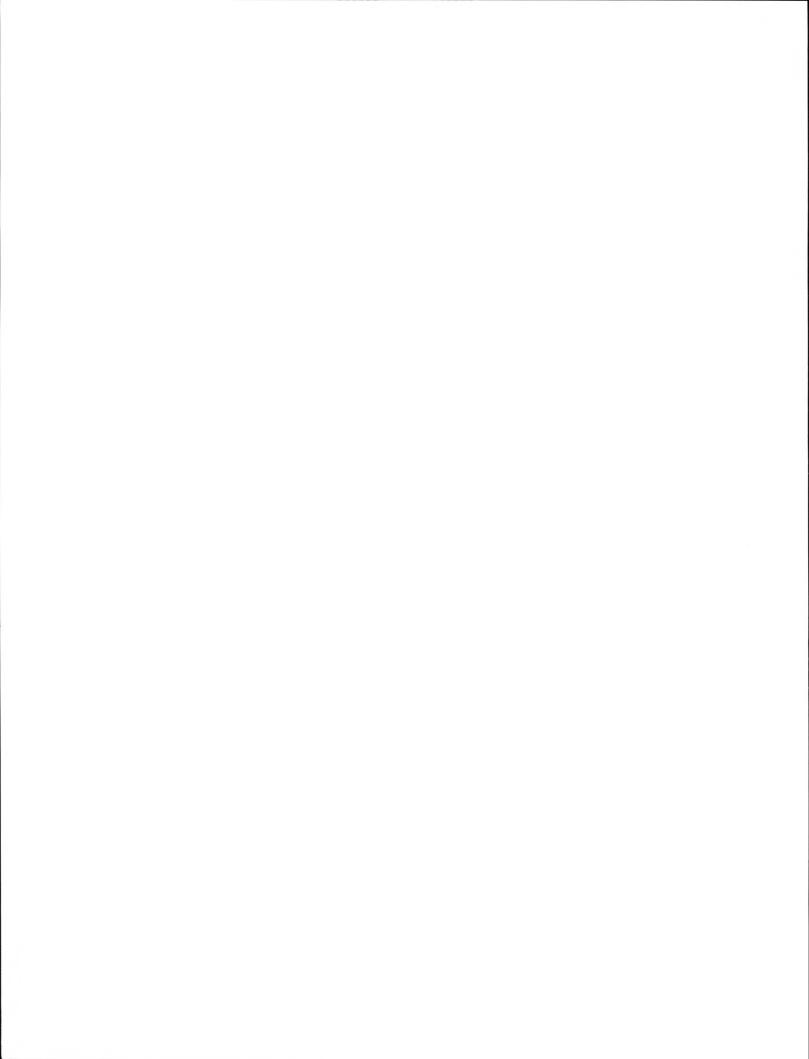
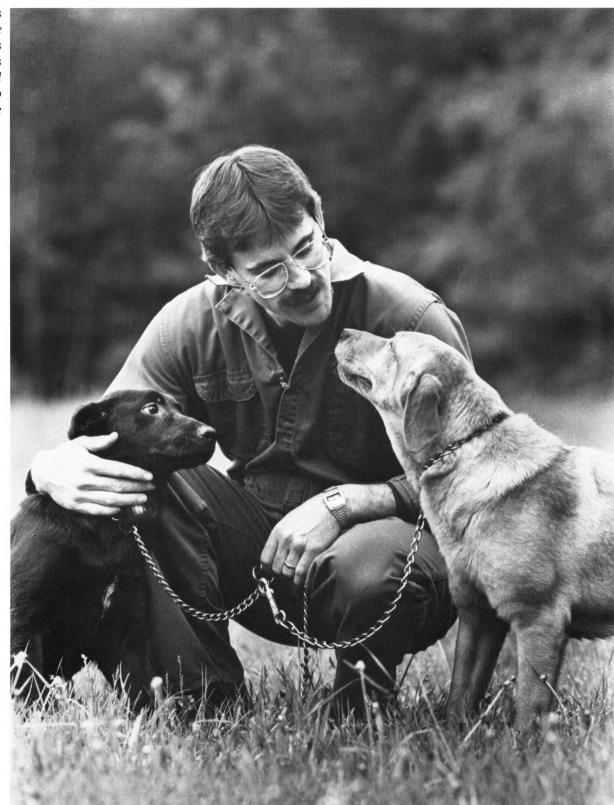
James A. Baker Institute for Animal Health Cornell University Annual Report 1985

New York State College of Veterinary Medicine Volume 35







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



The success of an institution like the Baker Institute is critically dependent on people.

Douglas McGregor



A Message from the Director

The Baker Institute celebrated its thirty-fifth birthday in 1985 by establishing new records in productivity and by strengthening its firm commitment to research, teaching, and service. For the first time in its history sponsored research surpassed \$1 million; the number of published reports by the Institute staff reached an all-time high; and our scientists were invited participants in thirteen international meetings and workshops.

Institute staff members made significant progress in their studies of infectious diseases, arthritis, and reproductive disorders. Much of the work made use of new methods that employ living organisms or their parts in useful ways. Popularly known as biotechnology, those techniques have found utility in many areas of research and as practical aids for diagnosing and treating disease. Considering the power of biotechnology and the opportunities it affords for analyzing and modifying biological processes, it was inevitable that Institute scientists would use those methods to study infectious agents and the diseases they cause and to uncover information about the nature of the immune system and of the osteoarthritic process.

The success of an institution like the Baker Institute is critically dependent on the environment in which the work is conducted and, more important still, on people. The Institute is fortunate in both respects. It has a superbly equipped facility and a dedicated staff of gifted men and women. The Institute also has an outstanding advisory council and a growing number of benefactors, whose generosity and encouragement have provided the margin of excellence in the Institute's programs. Those are the ingredients of a successful research enterprise. They have served us well for more than three decades and have created a firm foundation for the future.

As the Institute's programs have grown in size and complexity, so have its needs for personnel, laboratory space, and support facilities. Recognizing those needs, the advisory council endorsed a master plan that calls for the addition of two faculty-level scientists to the permanent research staff, the extension of the Link Building to provide new laboratory and office space, and property improvements to accommodate the Institute's equine research. The College of Veterinary Medicine and the University have responded to that initiative by providing the funds to support one faculty-level scientist and operating support for two laboratories that offer services to the Cornell community. The additional funds required to implement the master plan will be sought through a three-year capital campaign.

Our satisfaction with the progress made this year and our optimism were tempered by



Ann Signore



Our staff contributed in important ways to the college and the University.

Sue Hamlin

the loss of two faithful friends, Priscilla Maxwell Endicott and Dorothy Havemeyer McConville. Both will be remembered for their generous support of our research programs and restoration of the Institute's buildings.

The Simpson Trusts were named this year as recipients of the Arthur F. North Jr. Canine Service Award. The award was made in recognition of the magnificent support provided by the trusts for our canine parvovirus and osteoarthritis research.

A reception was held in December to mark Charles Bailor's retirement after thirty-six years of service to Cornell. Bailor was a founding member of the Institute's staff. His conscientious attention to detail and friendly manner will be greatly missed.

Although the Institute is first and foremost a research institution, its staff members contribute in many ways to the educational activities of the College of Veterinary Medicine and the University. We are particularly proud that Douglas Antczak was promoted this year to associate professor with tenure. Nancy Burton-Wurster and Judith Appleton were advanced to the rank of senior research associate. The promotions were made in recognition of the leadership of the individuals concerned, their outstanding performance in research, and their contributions to the teaching and service programs of the Institute and the college.

I want to acknowledge the splendid efforts of Leland Carmichael and Robin Bell, who served as graduate faculty representatives for the Fields of Veterinary Medicine and Immunology, respectively. Other members of our staff also contributed in important ways to the college and to Cornell programs, binding us still more closely to the University of which we are a part.

Douglas D. McGregor



Carlene Campbell

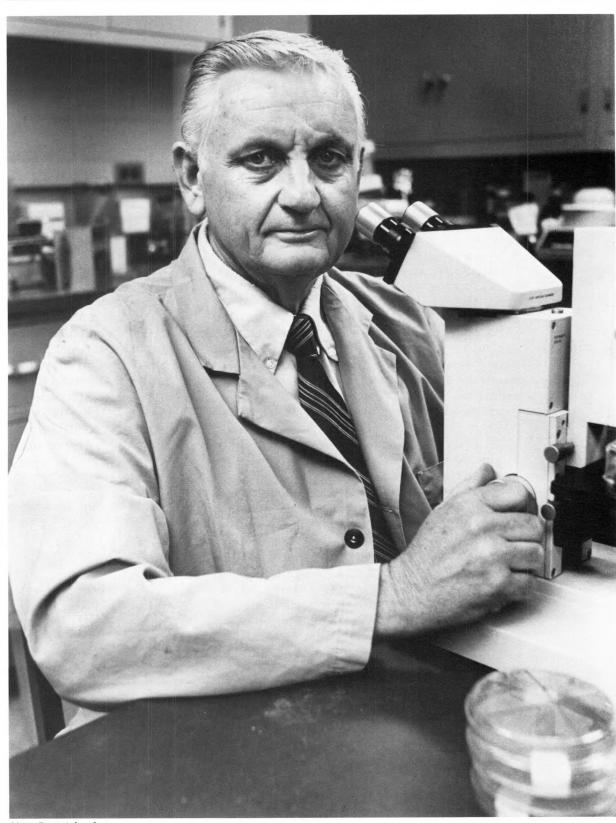


Ed Wheeler

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	Susan H. Hamlin, administrative manager: B.S., Elmira College Carlene M. Campbell, development assistant
	Nancy D. Combs, accounts coordinator
	Anita S. Hesser, secretary
	Florence C. Huth, administrative aide
	Gloria F. Mayer, secretary
	Sharon E. Morrow, accounts assistant
	Ann W. Signore, administrative aide: Cornell
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	Jean C. Joubert, research support specialist
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	Colin R. Parrish , assistant professor of virology: B.Sc., Massey University; Ph.D., Cornell Priscilla H. O'Connell , laboratory technician: C.L.A., A.S.C.P.
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	Jamie E. Levis, laboratory technician: B.S., Cornell
	Mary Beth Matychak, research technician: University of Evansville Susan E. Pearce-Kelling, laboratory technician: B.S., Cornell
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	George Lust, professor of physiological chemistry: B.S., University of Massachusetts; Ph.D., Cornell
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	Masataka Korenaga, postdoctoral associate: B.S., M.S., Kyushu University; Ph.D., Kumamoto University
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	Duzhang Zhu, graduate research assistant: M.D., Anhui Medical College
	Lincoln S. Adams, research technician: B.S., Hobart College; AALAS accreditation Jennifer A. Grigg, laboratory technician: B.R.T.C., Canberra Technical College

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	Jeannette C. Poleman, laboratory technician: B.A., Cornell
	Flow Cytometry Laboratory
	William V. Harris, research support specialist: B.S., Ohio State University; M.S., Cornell
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Animal Care	 Roy L. Barriere, vivarium supervisor: AALAS accreditation Charles B. Bailor, animal technician 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 Kevin T. Draiss, animal technician: B.S., Cornell James C. Hardy, animal technician: B.S., State University of New York Agricultural and Technical College, Farmingdale; 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
Maintenance	Edson Wheeler, Jr., maintenance supervisor Arthur D. Howser, maintenance mechanic Gerald G. Rice, vehicle mechanic



Each of us lives in a germ-filled world, and so do our dogs.

Skip Carmichael

Giralda Laboratory for Canine Infectious Diseases

Each of us lives in a germ-filled world, and so do our dogs. Fortunately most organisms do not cause disease, and some are even beneficial. Infectious diseases that plague human beings and other animals can be expected to continue, for the evolution of disease-causing agents through adaptation to new hosts is a fundamental process of life. The circumstances that prompted the emergence of canine parvovirus (CPV-2) in the dog population are almost as mysterious now as when the disease was first recognized in the summer of 1978. Nevertheless, significant progress has been made by Colin R. Parrish in characterizing natural isolates of CPV-2 and in locating on the viral genomes the regions that seem most important in the evolution of parvoviruses.

Our research continues to address the natural history of canine infectious diseases. We are concerned with the biology of CPV-2, with the problems of vaccination, and with the occurrence of parvoviral disease in wellmanaged kennels. As reported last year, factors that influence immunization success have been identified: different amounts of virus are required to immunize with different vaccinal strains; adult dogs have more variable antibody responses than do pups to certain vaccinal strains; and vaccines often fail to immunize pups that have low levels of maternal antibodies but that are nevertheless susceptible to infection with virulent CPV-2. Most field outbreaks in kennels appear to result from the latter phenomenon. No vaccine has been shown to reliably immunize pups that have low levels of maternal antibodies.

Canine brucellosis is still a major portion of our research and service activities. We have provided assistance to veterinarians and breeders who have obtained equivocal results using routine diagnostic tests. We have provided diagnostic assistance in more than 5,700 cases. Most requests were for help in differentiating infected from "falsepositive" animals. During the past year several outbreaks of brucellosis occurred in large kennels of breeding dogs and in working animals. In one outbreak involving valuable hunting dogs we provided diagnostic assistance and, with the collaboration of University of Florida scientists, instituted a treatment regimen that proved successful in curing dogs that had been infected for less than a month. It was demonstrated that infected dogs could be successfully treated and returned to service as working animals if the treatment was instituted early. Breeding of such animals was not recommended.

During the past year we developed an improved diagnostic antigen for *Brucellosis canis*. We took advantage of differences in cell wall antigens between a variant (M-) *B. canis* strain and the wild-type (M+) organism. The variant is less cross-reactive with related bacteria and has reduced the rate of false-positive reactions. The use of the M-variant as antigen in a rapid slide agglutination test (RSAT) reduced the rate of false-positive reactions in a panel of selected sera 15 to 50 percent. Like the original RSAT, the RSAT using the variant requires 2-mercaptoethanol for accurate results. The



Silla O'Connell



The evolution of disease-causing agents is a fundamental process of life.

Jean Joubert

accuracy of the RSAT was therefore significantly enhanced without sacrifice in specificity. In preliminary studies the M– bacterium detected infection one to two weeks earlier than the RSAT employing *B. ovis* as antigen. In conjunction with a highly specific test that employs internal (cytoplasmic protein) antigens in agar gel immunodiffusion (AGID) tests, the serodiagnosis of canine brucellosis has been markedly improved. Especially during the first four to eight weeks of infection, blood cultures are essential for the definitive diagnosis, as most animals develop a bacteremia within a week of exposure.

As reported last year, Jean Joubert prepared a library of monoclonal antibodies against purified cell wall antigens of M+ *B. canis* cells, but all were cross-reactive. A different strategy was then employed, using cell wall extracts of the M- variant as antigen, and specific *B. canis* monoclonal antibodies were obtained. They are being utilized in attempts to purify specific antigens that may prove useful in diagnostic tests.

In another study Laura Jones examined protein antigens of B. canis. Such antigens are used in our laboratory because of their high specificity for members of the genus Brucella. Unfortunately they have proved useful only in AGID tests; residual cross-reacting cell wall materials (lipopolysaccharide) have plagued our efforts to exploit those antigens in more simplified procedures, such as a latex agglutination test, that could be used in a veterinarian's office. Using immunoprecipitation of radio-labeled *B. canis* proteins and polyacrylamide gel electrophoresis, we identified four to six major immunogenic proteins from the more than thirty proteins in bacterial cell extracts. We observed that early sera (less than a month after infection) contained antibodies that reacted strongly with a 31 kD and a >200 kD protein; in contrast, late sera (more than two months after infection) all had strong reactions with a 66– 69 kD protein and weak reactions to a 51 kD protein. In chronically infected animals reactions to the 31 kD and >200 kD proteins were faint or absent, suggesting that it might be possible to estimate the infection status of an animal by the pattern of antibody reactivities with the cytoplasmic antigens. In field sera, where the time of infection could not be determined, there was considerable heterogeneity in the immunoprecipitation patterns.

Those preliminary studies have provided definition of the molecular weights of the cytoplasmic antigens and identified those that are most strongly immunogenic in infected dogs. Further research is in progress to define the nature of the antigens, their location in the bacterial cell, and the patterns of dog responses to them. Another goal of future research is to utilize recombinant DNA methods to clone the desired genes for defined antigens into a bacterial host that lacks cross-reacting antigens. We have developed methods to accomplish those goals, and monoclonal antibodies for certain *B. canis* surface antigens are now available.

Leland E. Carmichael



Nancy Combs



Why do viruses infect one animal species and not another?

Colin Parrish

Virology Laboratory

Why do viruses infect one animal species but not another? What happens to viruses over time? Do they stay the same or change? We are seeking answers to those questions in our studies of canine parvovirus (CPV-2). CPV-2 was first recognized in 1978 as the cause of a new disease of dogs. Soon thereafter it was recognized in many other countries throughout the world. Although a new virus, it is similar to previously known parvoviruses that cause disease in cats and mink.

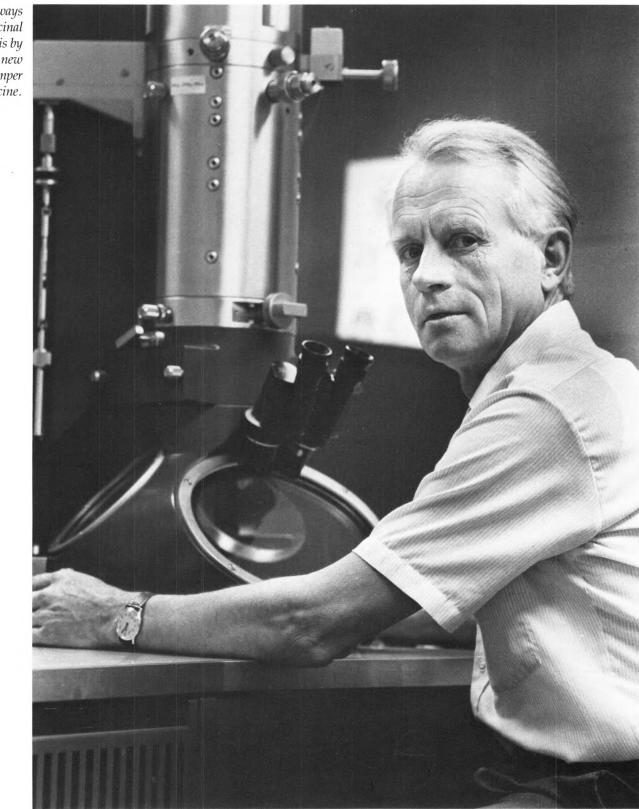
A popular hypothesis is that CPV-2 arose as a variant of either the cat or the mink parvovirus. If CPV-2 is such a variant, it must have acquired the ability to infect and cause disease in dogs, as the cat and mink viruses do not grow well in dogs or in dog cells. We have been examining relationships between those parvoviruses to pinpoint changes that might have given rise to CPV-2. One approach has been to construct variant strains of the dog and cat viruses to determine their characteristics and host range. Such variants are hybrids consisting of spliced segments of the two viruses. Individual variants constructed in that way differ from one another in the particular segment of the viral genome that codes for the coat protein of the virus. The differences are associated with alterations in both the surface properties of the virus particles and their capacity to infect dog and cat cells.

The results of our research suggest that only a few small changes are necessary to create a host-range difference between the parvoviruses of cats and dogs. As only a few changes allow the cat virus to replicate efficiently in dogs, we do not yet know the circumstances that could have brought those changes about. One way we have addressed the problem has been to determine whether the parvoviruses we are studying vary in nature—whether there are several forms of each virus or only one of each that infects cats, dogs, or mink. It appears that the viruses are relatively stable but that changes in them do occur. For example, two forms of the mink virus have been identified. Both have been infecting mink for many years, although recently one of the two forms has become much more common. Among the dog viruses the situation is more complex. We have shown that the strain of CPV-2 that first emerged in dogs and caused the initial outbreaks of disease in 1978 was replaced in 1980 by a second type of virus. While the two isolates differ only slightly, the newer type has apparently replaced the older form as the cause of CPV-2 in dogs.

Colin R. Parrish



Laura Michel and Kevin Draiss



We are seeking ways to avoid postvaccinal encephalitis by developing a new type of distemper vaccine.

Max Appel

Hadley C. Stephenson Laboratory for the Study of Canine Diseases

Although canine distemper has been controlled by vaccination for more than twenty years, vaccinated dogs sometimes develop an inflammatory reaction in the brain. That reaction, called postvaccinal encephalitis, seems to occur most frequently when live modified canine distemper vaccine and canine parvovirus vaccine are administered simultaneously. We are seeking ways to avoid encephalitis by developing a new type of distemper vaccine that is both safe and effective. In collaboration with Dr. Erling Norrby of the Karolinska Institute in Sweden, we have identified the portions of the canine distemper virus coat (the F and H proteins) that can immunize dogs against a challenge infection with virulent distemper virus. We now want to isolate the viral genetic information required for the production of the F and H proteins. That segment of the genome would then be inserted into vaccinia virus, an innocuous agent that has been used for many years to immunize humans against smallpox. The recombinant vaccine would be tested for its capacity to protect dogs against distemper.

Zoo animals such as red pandas, kinkajous, and black-footed ferrets can develop encephalitis when given vaccines containing live modified distemper virus. To avoid that complication, we have prepared special vaccines containing inactivated distemper virus for the vaccination of animals at the National Zoo in Washington, D.C., the Buffalo Zoological Gardens, the Baltimore Zoo, the Game and Fish Department of Cheyenne, Wyoming, and the University of California, Berkeley.

On a related front, we are studying how distemper virus infects brain cells. Persistent infection is associated with the loss of myelin, the sheathing material of nerve fibers. As demyelination is the hallmark of diseases like multiple sclerosis, it may be that those diseases are caused by viruses. Using the electron microscope, Dr. Brian Summers of the Department of Veterinary Pathology is studying demyelination in the brain tissue of dogs infected with distemper. He and Sue Pierce-Kelling are also using brain cell cultures to study the infection itself. Their investigations have shown that distemper virus strains that differ in their pathogenicity for dogs also differ in their capacity to infect and persist in brain cells.

A laboratory test that would detect demyelination would be a valuable diagnostic aid in both human and veterinary medicine. In collaboration with Dr. John Whittaker of the University of Alabama, we are exploring the possibility of developing such a test, based on our capacity to detect myelin fragments in the cerebral spinal fluid of distemperinfected dogs.

Still another aspect of our research on persistent viral infections involves the development of methods for detecting virus or viral constituents when they are present in vanishingly small amounts in infected cells. Jeff Mitchell has been addressing the problem in distemper-infected dogs. The results obtained so far are encouraging and will be extended this year in efforts to detect viral nucleic acid in brain cells after the virus can no longer be cultured from the brain.

Max J. G. Appel



Osteoarthritis is the principal cause of the pain and immobility associated with hip dysplasia.

George Lust

John M. Olin Laboratory for the Study of Canine Bone and Joint Diseases

Osteoarthritis is a common debilitating disease of many species. It often occurs in dogs with hip dysplasia and is the principal cause of the pain and immobility associated with that disease. Our research is concerned with the underlying process. We are studying how changes in protein elements of cartilage relate to the progression of osteoarthritis, whether those changes can be modified to arrest the disease, and whether a conspicuous increase in a particular protein, fibronectin, can be detected in synovial fluid or blood, thereby providing the basis of a diagnostic test.

Cartilage is rich in proteoglycans. Those large molecules give that tissue its normal resiliency. It has long been known that proteoglycans are diminished in the cartilage of osteoarthritic joints. Initially we thought that the observed increase in fibronectin in diseased cartilage was related to the loss of proteoglycans. But our most recent results indicate that that is not the case. Fibronectin increases early in the disease process, before proteoglycans are lost. We are now focusing on the questions of whether fibronectin is produced locally by cartilage cells and whether local accumulation of that protein is related to the progression of disease.

The accumulation of fibronectin in cartilage seems to be a general feature of the osteoarthritic process. It occurs not only in dogs but also in rabbits and human beings. In the latter two species the increase occurs in the absence of hip dysplasia. That finding implies that although hip dysplasia may promote the observed changes in the protein composition of cartilage, the changes are an independent expression of the osteoarthritic process.

We used biochemical and immunological methods to measure the accumulation of fibronectin in the cartilage of osteoarthritic joints. The procedure involves staining frozen sections of cartilage with antibodies to fibronectin and examining the stained tissue under the microscope. Those studies showed that fibronectin does not accumulate in isolated foci but is distributed uniformly throughout the cartilage. In collaboration with CIBA-GEIGY Corporation, we evaluated the ability of several new antiarthritic drugs to modify the level of fibronectin in the articular cartilage of rabbits and dogs with osteoarthritis. Such studies may be useful in selecting drugs for the treatment of that disease.

In a related area we confirmed that the fibronectin of synovial fluid differs in biochemical composition from the fibronectin of blood. Our intention is to prepare a monoclonal antibody that is specific for the synovial fluid protein. If we are successful, we will ascertain whether that antibody can detect trace amounts of synovial fluid fibronectin in blood. A test based on that finding might be a useful adjuvant for the diagnosis of osteoarthritis and a means for identifying dogs that will develop hip dysplasia.

George Lust Nancy Burton-Wurster



We want to understand how parasites suppress the host's immune system.



Robin Bell

Immunology Laboratory

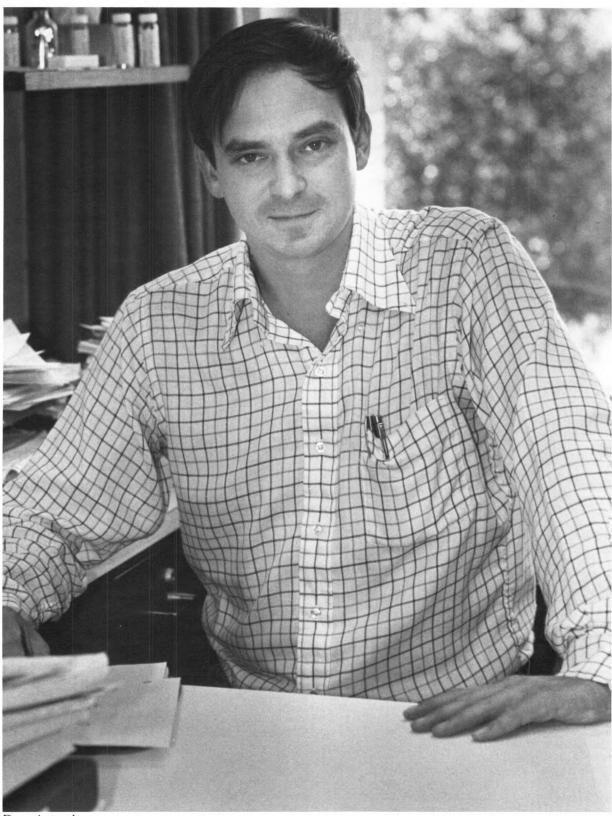
Our research is concerned with immunity to parasites, organisms that cause disease in all animal species, including human beings. Parasites are an astonishingly diverse and complex group of organisms that live in every site in the body. We have been studying nematodes (roundworms), a group of parasites that reside for the most part in the intestine. Such worms represent a formidable challenge to the host, not only because of their size but also because many have developed mechanisms for evading the host's immune defenses. For example, some parasitic worms impede the development of antibodies or protective cells that might otherwise promote rejection of the intruder. Others alter their antigenic characteristics as they grow and mature, thereby escaping recognition by antibodies that were formed against earlier stages in their development. For those, and possibly other, reasons many worms survive in their animal hosts for months or years. In doing so, they interfere with normal body functions and injure the tissues that harbor them.

A major objective of our research is to understand how parasites suppress the host's immune system. To make progress in that endeavor, we must first know how antibodies or cells of the immune system impede the growth and development of parasites or promote their expulsion. The infection caused by *Trichinella spiralis*, the causal agent of trichinosis, has been instructive in that connection. Over the last several years we examined how immunity to T. spiralis develops in mice and rats. Those studies showed that immune responses develop against antigens that are expressed by the parasite at different stages in its life cycle. Immunity results in failure of the parasites' larvae to establish themselves in the intestine, in development of a weakened adult worm that has a diminished reproductive potential, and in premature expulsion of the worm from its intestinal niche. A related aspect of our research has been to identify host genes that dictate the success or failure

of those antiparasite responses. In recent experiments using mice we showed that two major genes control the rate at which *T. spiralis* worms are expelled.

During the last year we discovered that lymph draining the intestine of rats infected with T. spiralis carries a population of lymphocytes that can protect normal rats against a challenge infection with that parasite. The cells that mediate the observed protection are added to the lymph three to four days after infection. Many are actively dividing. They enter the blood by way of the thoracic duct but rapidly leave the circulatory system and accumulate in the intestine, at the very site where worm rejection occurs. Using antibodies that detect surface antigens on some but not all lymphocytes, we intend to characterize the cells concerned. We also want to pinpoint the location of the cells in the intestine and to determine how they realize their protective function. Our analysis is expected to shed light on an important but poorly understood aspect of intestinal immunity and to pave the way for an investigation of how the parasite impedes the host's immune defenses.

Robin G. Bell



Our most important work involved the transfer of embryos between donkeys and horses.

Doug Antczak

Equine Genetics Center

Research in the Equine Genetics Center is currently directed toward three areas: fundamental investigation of immunogenetic systems of the horse, study of immunological aspects of pregnancy in mares, and study of equine influenza virus involving the use of monoclonal antibodies. The latter research, conducted by Judy Appleton, is described elsewhere in this report.

Studies of the equine major histocompatibility complex, called ELA, for equine lymphocyte antigen, have been under way at the Institute since 1979. The work has resulted in the discovery and characterization of over twenty new genetic markers of the horse. The markers have utility as improved methods of horse blood typing. They are also being used in studies of equine pregnancy.

The most important work undertaken at the whole-animal level this year involved the transfer of embryos between donkeys and horses. Members of the horse family (horses, donkeys, and zebras) have the rare ability to hybridize between species, producing viable, but nearly always sterile, offspring, including the well-known mule and less-known hybrids such as the zebronkey. It is also possible after successful embryo transfer for donkey mothers to carry horse foals to term, and horses, zebra foals.

The donkey-in-horse pregnancy established by embryo transfer is the only type of embryo transfer between member species of the horse family that usually ends in abortion. The pregnancy loss often occurs between days 80 and 95 of gestation. The abortion is thought to result from the failure of formation of endometrial cups, small ulcerlike structures of placental origin that are found in the uterus of mares between 40 and 120 days of pregnancy.

It is expected that donkey-in-horse pregnancies will help scientists determine the functions of the endometrial cups, especially their role in regulating maternal antifetal immune responses. One approach to the problem has been an attempt to develop

treatments that will permit mares to carry transferred donkey conceptuses to term. The work is being done in close collaboration with Dr. W. R. Allen, director of the Equine Fertility Unit of the British Thoroughbred Breeders' Association, in Cambridge, England. The most promising avenues explored thus far involve immunotherapy based on either transfer of serum from mares carrying normal horse fetuses to mares carrying donkeys or immunization of mares with white blood cells from the genetic parents of the donkey embryo that was transferred to each recipient. The mechanism by which those treatments protect donkey-in-horse pregnancies is not known, and the treatments do not always work. Nevertheless the results are encouraging.

In the laboratory cultures of endometrial cup cells have been established that permit study of hormone production by cup cells and investigation of interactions between maternal lymphocytes and placental tissues. In addition a panel of monoclonal antibodies with specificity for endometrial cup cells has been produced in the Institute's Cell Fusion Laboratory. Those antibodies are being used to identify functionally important molecules of the endometrial cups.

Two important meetings were organized by the Equine Genetics Center in cooperation with the Havemeyer Foundation. The First International Symposium on Equine Embryo Transfer was held in Ithaca in late 1984. Over fifty veterinary scientists attended the meeting, which resulted in the publication of an extensive volume of proceedings as a supplement to the Equine Veterinary Journal in March 1985. The Fourth Workshop on Lymphocyte Alloantigens of the Horse, held in Lexington, Kentucky, in October 1985, drew participants from four countries and resulted in the identification and recognition of six new antigens of the equine major histocompatibility complex.

Douglas F. Antczak



Influenza viruses are known for their tendency to drift.

Judy Appleton

Donnelley Laboratory of Immunochemistry

We continued our research aimed at improving methods of vaccination against equine influenza. We have been studying antigenic drift, the gradual change that occurs in the surface proteins of certain viruses. Influenza viruses are known for their tendency to drift, but the process has not been studied closely in the equine virus. We are particularly interested in one protein of that virus, the hemagglutinin, because antigenic drift in the hemagglutinin of human influenza viruses accounts in large measure for the capacity of those viruses to evade the host's immune defenses and of the disease to spread through a population.

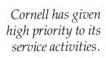
The equine influenza vaccine now in use was prepared from a virus isolated in 1963. The question therefore arises whether the equine virus has drifted sufficiently since then to reduce the effectiveness of the vaccine. To address that question, we produced a panel of ten mouse monoclonal antibodies specific for the hemagglutinin of the equine virus. Using those antibodies, we found that the hemagglutinin on recent virus isolates has changed considerably from the hemagglutinin on the vaccine virus. The change seems to have occurred at only one of three demonstrable sites on the hemagglutinin molecule; the other two sites have been preserved. The equine virus hemagglutinin is apparently changing much more slowly than its counterpart on the human viruses. However, we found that variants of equine viruses exist in numbers similar to those of human viruses. Thus the equine virus is similar in its ability to change, but it probably has fewer opportunities to replicate in the relatively small horse population. The findings suggest that more recently isolated viruses should be incorporated into vaccines, even though less frequent vaccine changes are required for equine viruses than for human viruses.

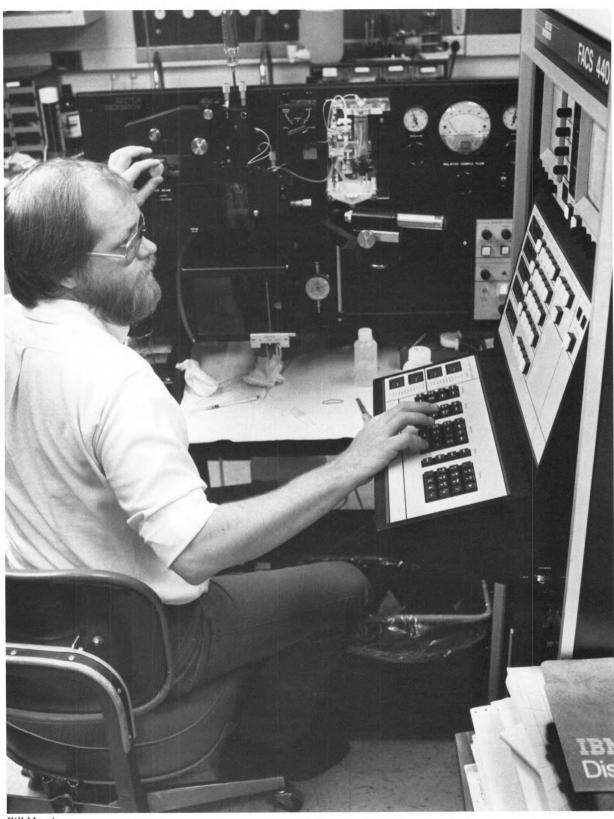
We are also analyzing the immunity conferred on suckling animals by their mothers' milk. In our work we have employed *Trichinella spiralis*, a parasite that initiates its life cycle in the gastrointestinal tract of a susceptible animal. That characteristic of the parasite makes it an excellent model for the study of other intestinal parasites, intestinal cancer, and neonatal diarrhea.

When female rats are infected with T. spiralis, their suckling offspring are protected from challenge with the parasite. We showed previously that antibody in the dam's milk mediates that protection and that the antibody is induced by larvae encysted in her muscles. The immunity is specific in that it is directed at infectious larvae when they first enter the pup's intestine. The immunity is similar to the phenomenon of "rapid expulsion" in adult rats. Rapid expulsion can cause the rejection of as many as 99 percent of a dose of challenge worms within twenty-four hours. To better understand how antibody mediates expulsion, we developed sensitive binding assays using monoclonal anti-immunoglobulin reagents to detect parasite-specific antibodies of each of several subclasses. We showed that the mechanism of action of the antibody is not likely to involve mast cells and that the antibody mediator is not of a class traditionally associated with immunity at mucosal surfaces. Protection is associated with a subclass of IgG, an immunoglobulin found in the blood.

We have begun to characterize the parasite components that are the targets of the antibody. We have collected proteins larger than thirty thousand daltons that the target muscle larvae release into protein-free medium during culture in vitro. That preparation contains only a few of the parasite's proteins. We found that a single dose of as little as twenty-five micrograms of this protein can stimulate immunity as strong as that induced during a natural infection. We are continuing our studies of those proteins, their function and how antibodies interact with them to inhibit larvae from establishing themselves in the intestine.

Judith A. Appleton





Bill Harris

Research Service Laboratories

The Institute operates two research service laboratories, one for the production of antibodies and another that uses computercontrolled laser methods to analyze and separate cells. The Cell Fusion Laboratory was established in 1977. Its principal function is to produce molecularly homogeneous antibodies. Such monoclonal antibodies are used in the Institute's own research programs and by investigators in other departments and divisions of the University.

Although the Flow Cytometry Laboratory has operated for only two years, it too provides a wide range of services to Institute staff and other Cornell investigators. The mainstay of the facility is a fluorescenceactivated cell sorter, which can analyze and separate individual cells at high rates (about five thousand cells per second). The cells pass through a laser beam in a fluid stream, one at a time, allowing several properties of the cells to be measured, such as their lightscattering characteristics, size, and fluorescence. The information is analyzed by a computer and can be displayed in useful ways. Cells that can be distinguished in those ways can also be isolated for further study.

Cornell has given high priority to those service activities and has provided operating support for them. Similar facilities will be established on the main campus in 1988. They will be integrated with our own to provide a wide range of services and training opportunities for faculty members and students. That arrangement will benefit the Institute in several ways. It will foster collaboration between our staff and the faculty of other departments, and it will provide new opportunities to utilize contemporary methods of biotechnology in our animal health research.

William V. Harris



Clare Poleman



Chris Davies and Anne Crump

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The Institute in Perspective

Thirty-five years ago the Baker Institute was established as a permanent research facility for the study of animal diseases. Its main component was the Cornell Research Laboratory for Diseases of Dogs. Research undertaken in that facility was initially concerned with the study of distemper, hepatitis, and other infectious diseases. That work has continued in studies of canine parvovirus, distemper, and brucellosis. But the investigations have broadened to include research on canine hip dysplasia and osteoarthritis. Much has been learned about those diseases, including methods for their diagnosis, treatment, and control.

That new knowledge in itself has justified the initial investment in the Institute, for the investment has been amply rewarded in the greatly improved health that dogs now enjoy. However, research undertaken at the Institute has further significance that touches on our own health. Knowledge gained about distemper or canine parvovirus has increased our understanding of other viruses-their relationships to one another, how they evolve in nature, and, most important, how they can be controlled. Likewise, studies of canine hip dysplasia have encouraged investigations into the nature of osteoarthritis, a disease that affects virtually every species and is the cause of much suffering and economic loss.

Canine health continues to be the major concern of the Institute. In the last few years, however, the Equine Genetics Center was established as a second division of the Institute. Like the Research Laboratory for Diseases of Dogs, it coordinates research on health problems of horses. Reproductive failure and equine influenza are the major concerns at this time. Although the programs are new, they have already attracted considerable support and interest because of the information they are providing about practical problems of disease in horses.

From a modest beginning, the Institute has grown into a research enterprise that oper-

ates at the forefront of veterinary medicine. Its success is largely due to the support it has received from dog clubs, foundations, veterinarians, and a growing number of concerned individuals. That interest will sustain the Institute in the years ahead and will ensure its capacity to respond rapidly to health problems that concern us all.

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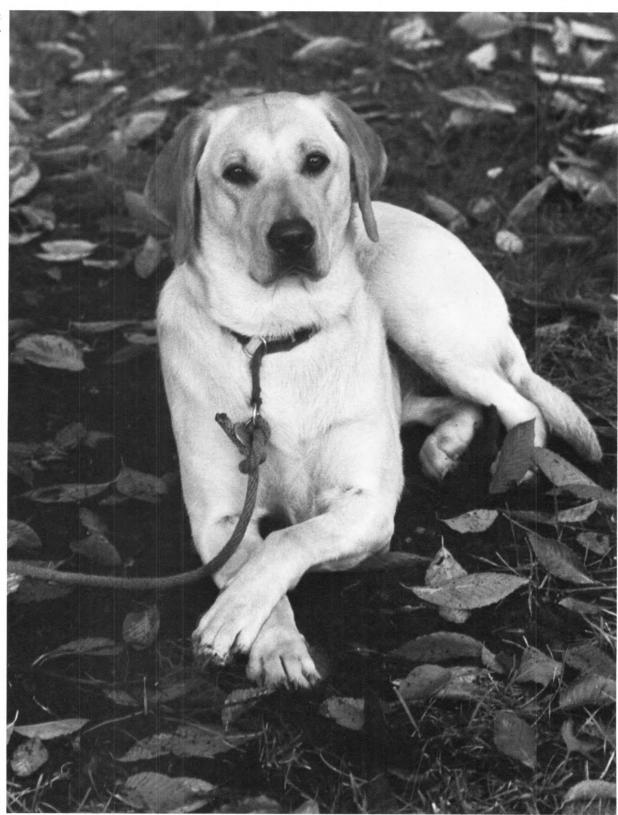
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Your interest in the James A. Baker Institute for Animal Health, expressed by your gift, enables us to carry out our day-to-day mission. With your support we can respond swiftly to opportunities as they arise and improve the quality of animal health.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

In appreciation for their exceptional interest in the Institute, we should like to express our gratitude to the American Kennel Club, Mrs. Warren Bicknell, Jr., Mr. Warren Bicknell III, Miss Wendy H. Bicknell, Mr. and Mrs. Louis G. Bissell, Jr., Mr. and Mrs. Robert S. Boas, Mr. and Mrs. Albert C. Bostwick, Jr., Mr. Atherton Bristol, Mr. and Mrs. Gaylord Donnelley, Mrs. Pamela Firman, Mrs. Ellen Frenkel, Mrs. Gordon H. Gillis, Mr. and Mrs. B. Douglas Gordon, Mrs. Dona E. Hausman, Mr. and Mrs. Clifford P. Hickok, Mrs. Gilbert W. Humphrey, Miss Kate Ireland, Mr. and Mrs. R. L. Ireland III, Mr. John P. Kendall, Mr. and Mrs. H. Peter Kriendler, Mr. and Mrs. David J. McFadden, Mr. John Morgan, Dr. Seeley W. Mudd II, Mrs. Adelaide C. Riggs, Mr. J. B. Robinson, Dr. Ben E. Sheffy, Mrs. Clare E. Thaw, Mr. Jack B. Ward, Mr. and Mrs. Harwood Warriner, Mr. Gilbert H. Wehmann, Mr. Robert Winthrop, Mr. Robert Winthrop II, Mr. and Mrs. C. Martin Wood III, and Ms. Shirley E. Wright.

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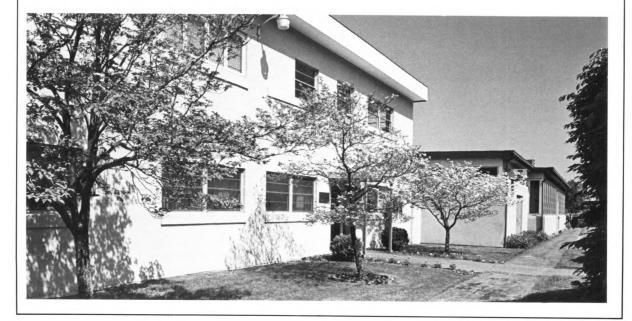
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