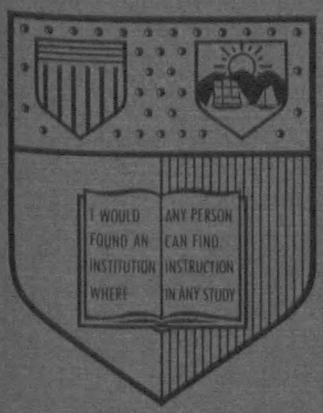
James A. Baker Institute for Animal Health

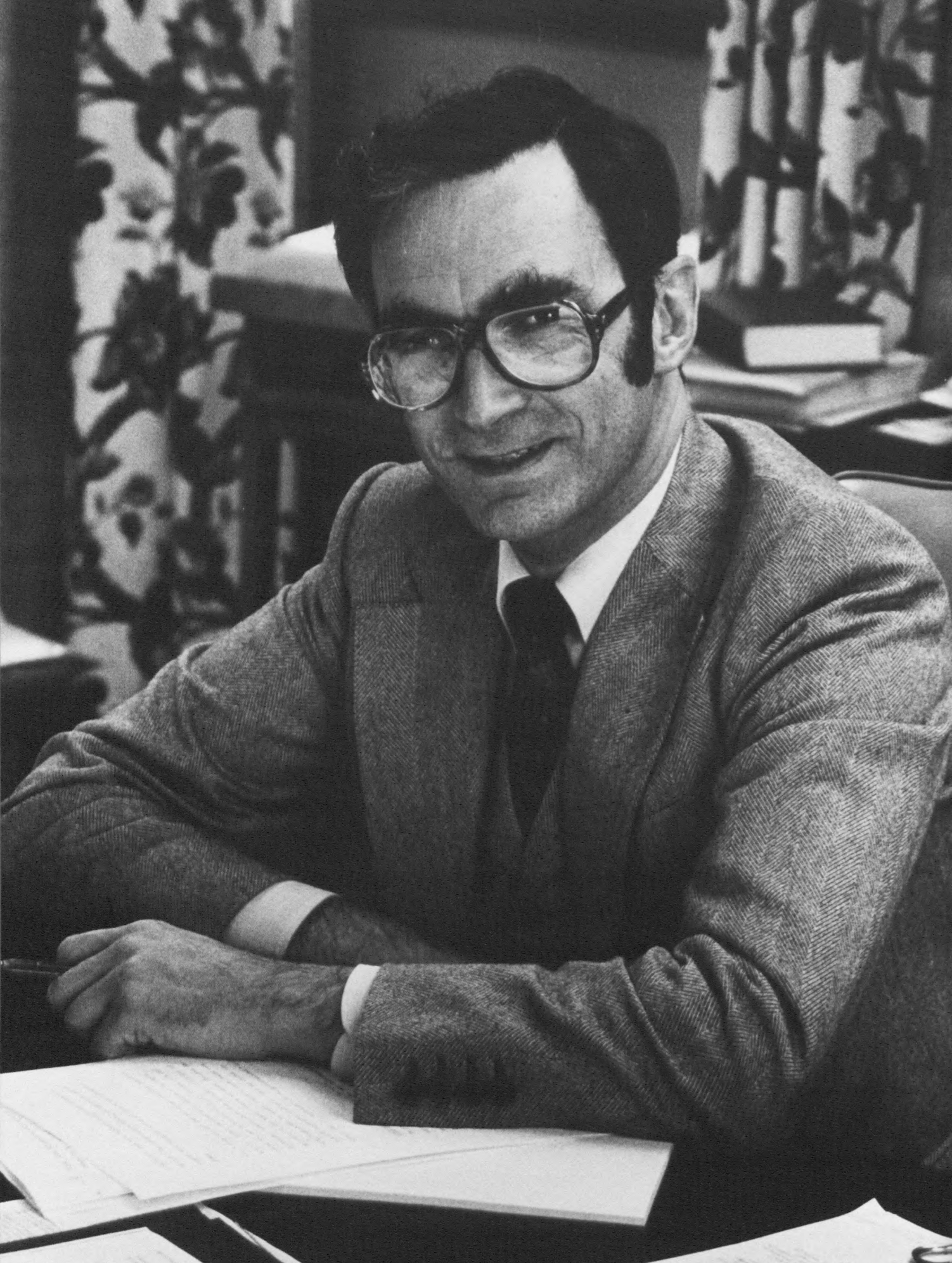
Annual Report 1978



Cornell University, Ithaca, New York

Volume 28

This report honors those
whose generosity sustains the Institute's quality and independence,
its high ideals of excellence and responsibility
in the challenge and pursuit of truth.



A Message from the Director

Nineteen seventy-eight was a banner year for the James A. Baker Institute. Important advances were made in our research; a new record was established in the number of scientific publications emanating from the Institute; improvements were made in our daily operations; and two capital projects were initiated that will enable us to broaden our research and explore new avenues in the area of disease prevention.

The results of our research are described in the following reports by members of the Institute's senior staff. Some of our findings are immediately relevant to the health and well-being of dogs. Others have general significance in respect to disease problems not only of dogs but of food-producing animals and man himself.

The formal record of our research accomplishments is contained in our list of publications. Many of these reports have been published in leading scientific journals. They provide visible evidence of our productivity and the quality of our research and account in large measure for increased recognition of the Institute and new support in the form of grants, contracts, and gifts.

A laboratory report, *Infectious Canine Enteritis Caused by a Corona-like Virus: Current Status and Request for Information,* was published this year. A copy of this report can be obtained by writing to the Institute or by telephoning Mrs. Florence Huth (607/277-3044).

In June 1977 the Institute was certified by the American Association for Laboratory Animal Care. We are one of the few institutions in New York State to be so accredited. Five members of our animal care staff were certified this year by the American Association for Laboratory Animal Science, bringing the total number to seven. Mr. Michael Chapman and his associates are to be congratulated for these achievements, which bring great credit to the Institute.

Important improvements were made in our central washing and sterilization facility. These improvements will enable Mrs. Elizabeth Wheeler and Miss Julie Jordan to provide investigators with the scrupulously clean glassware needed for tissue culture and our research with viruses.

Two projects were initiated that will have a significant impact on our future activities. The Adele S. Colgate Tissue Culture Laboratory will be modernized and furnished with new equipment needed for the propagation and attenuation of viruses and for research using the relatively new tools of somatic cell genetics. We are deeply grateful to the Surdna Foundation for their generous gift that made this improvement possible.

The second project became a reality when the National Cancer Institute awarded the Baker Institute a grant of more than $400,000. These monies, given in recognition of our basic research, will be used to construct a facility where laboratory animals can be housed in a proper environment in an area adjacent to the existing building. New approaches to disease prevention will result from research conducted in the new facility.

Above: Neil H. McLain directs the Institute's day-to-day functions.

Above right: Sandy Borow receiving a research partner citation on behalf of the Finger Lakes Kennel Club in recognition of their outstanding support

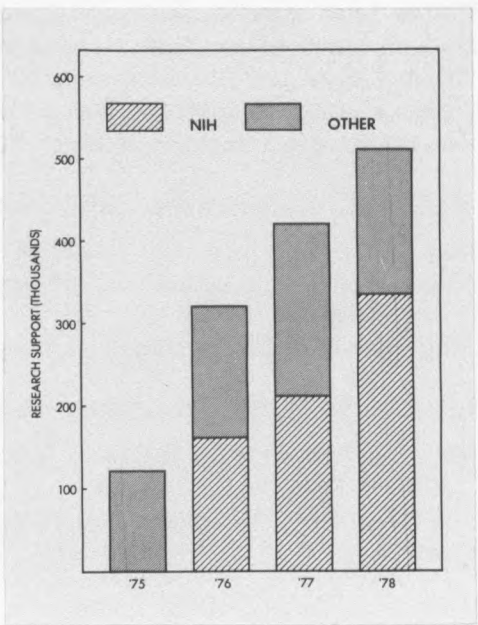
Right: Ann W. Signore's secretarial responsibilities include correspondence and the preparation of laboratory reports.

Service to the public continued not only in the publication of our research findings but also in the participation of our staff in meetings and seminars throughout the country. A visible example of our concern for the practical problems of disease was the isolation by Drs. Leland Carmichael and Max Appel of two viruses responsible for several outbreaks of canine infectious enteritis.

Work on these viruses, the nutritional requirements of dogs, hip dysplasia, and other diseases continues, but additional support is needed. The accompanying figure illustrates our increasing dependence on grants and contracts from the National Institutes of Health (NIH). Although this support has grown in recent years, the NIH is concerned mainly with human diseases; therefore support from this source will be more difficult to obtain in the future. Our ability to pursue meaningful research on canine diseases will depend in large measure on the financial support of foundations, kennel clubs, and individuals who share our interest in improving the health of dogs.

We face the future confident in the ability of our well-trained staff to meet the challenges of our changing research, academic, and economic environment. At the Institute we are particularly fortunate in being served by an advisory council that provides moral support, expert advice, and scientific guidance. With the added ingredients of material support and understanding from those interested in the welfare of dogs, we will maintain the tradition of excellence and service that has characterized the activities of the Institute during the twenty-eight years of its existence.

Douglas D. McGregor
Director



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Bernard L. Clark, research technician
James M. Ebel, animal technician
James C. Hardy, research technician: B.S., Cornell
Ronald A. Hayes, animal technician: AALAS accreditation
Gerald W. Hiller, animal technician: AALAS accreditation
David L. Watkins, animal technician: A.A.S., State University of New York, AALAS accreditation
James E. Young, animal technician: AALAS accreditation

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Gerald G. Rice, mechanic
John C. Howe, custodian

Giralda Laboratories for Canine Infectious Diseases

Much of our effort this year was directed toward identifying the canine viruses responsible for contagious gastroenteritis. Several outbreaks were reported in the United States in 1978, the first occurring in the spring. These outbreaks were characterized by occasional vomiting and diarrhea, sometimes hemorrhagic, and occasional deaths. A canine coronavirus (CCV) was isolated from stool specimens. Common features of the CCV infection were a loose, putrid orangish stool, lack of significant fever, and recovery after one to three weeks of intermittent diarrhea. Six strains of CCV were isolated in primary dog kidney cell cultures (Cornell Research Laboratory for Diseases of Dogs, Laboratory Report Series 2, no. 9, July 1978).

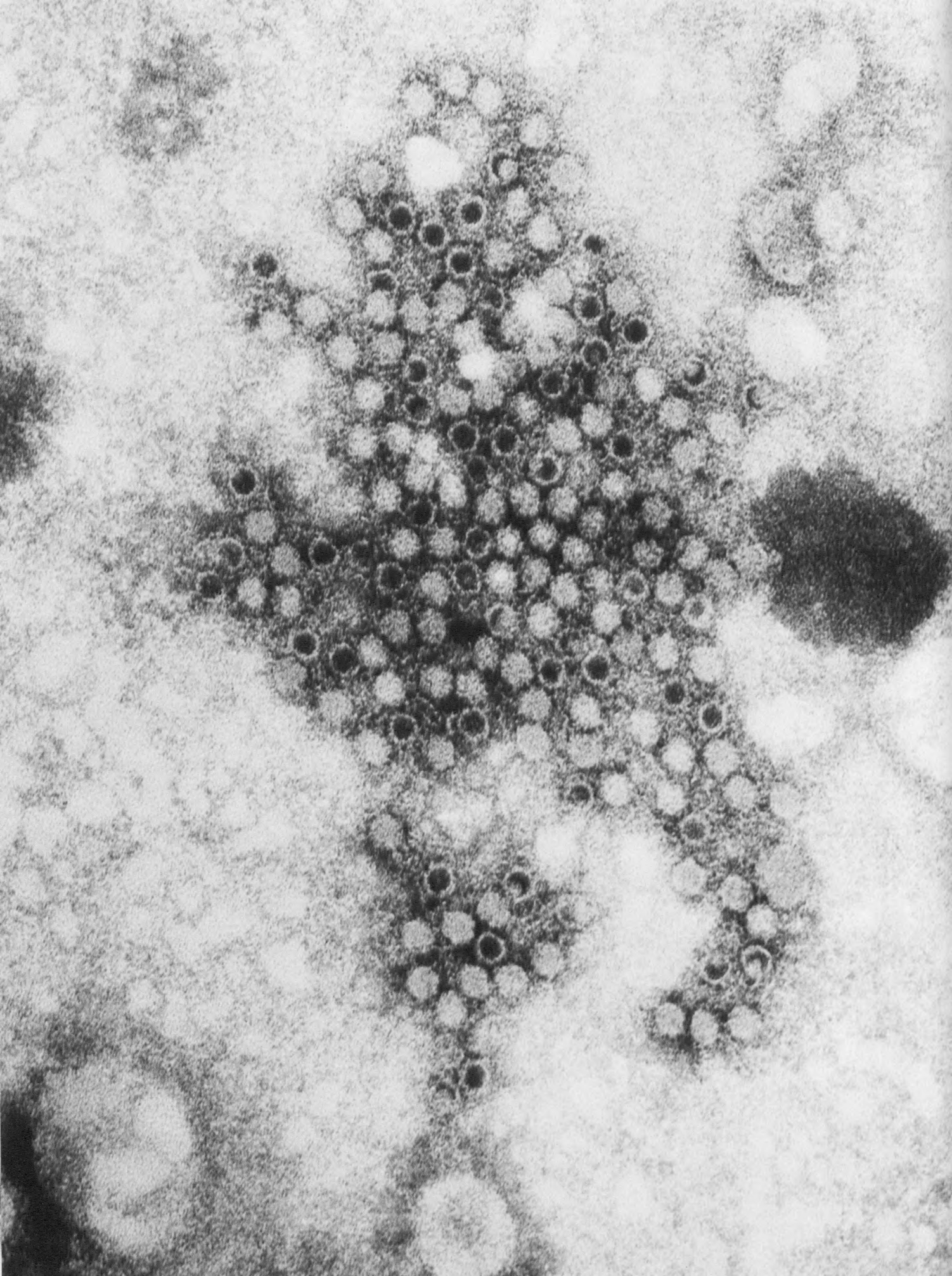
A second agent, a parvovirus, was observed by electron microscopy in stool specimens from dogs with a more severe form of contagious gastroenteritis. The parvovirus family includes a variety of viruses, among them the causal agent of feline panleukopenia. Outbreaks where the canine parvovirus (CPV) has been found in great numbers in stool samples are characterized by severe vomiting, fever (103–105°F), a marked decrease in circulating white blood cells (leukopenia), and destruction of cells lining the intestinal tract. Rapid dehydration may occur, especially in puppies, leading to death. Intestinal lesions are similar to those caused by the feline parvovirus (feline panleukemia virus). Intranuclear inclusion bodies have been observed in some cases. CPV appears to be highly contagious, especially where dogs are closely confined. A close antigenic relationship exists between the CPV and feline panleukopenia virus, suggesting reciprocal protective immunity.

Current research on infectious canine viral gastroenteritis at the Institute, with Drs. Appel and Greisen, gives priority to developing methods for isolating, growing, and quantitating the viruses; developing serological procedures for diagnosis; defining the clinical and pathological manifestations of disease; locating the principal growth sites of each virus and determining the duration of viral shedding; characterizing the immune response; and developing appropriate immunizing agents.

Our research continued on another disease, canine brucellosis. The extent of the problem of diagnosing brucellosis in the dog was revealed in a review of diagnostic methods available.

We made significant progress toward establishing reliable serological criteria for diagnosing the disease. We tested more than four thousand canine sera: samples submitted to the laboratory and samples collected over three years from experimentally infected beagles maintained at the Institute.

The results forced us to conclude that when used alone, none of the serological methods in common use is adequate to definitively diagnose brucellosis. Such a diagnosis can be made only by isolating the causal agent, *B. canis*, from the blood. However, by employing an immunodiffusion (ID) test that uses a *B. canis* cell wall extract as antigen, one can judge field samples with 85-percent accuracy.

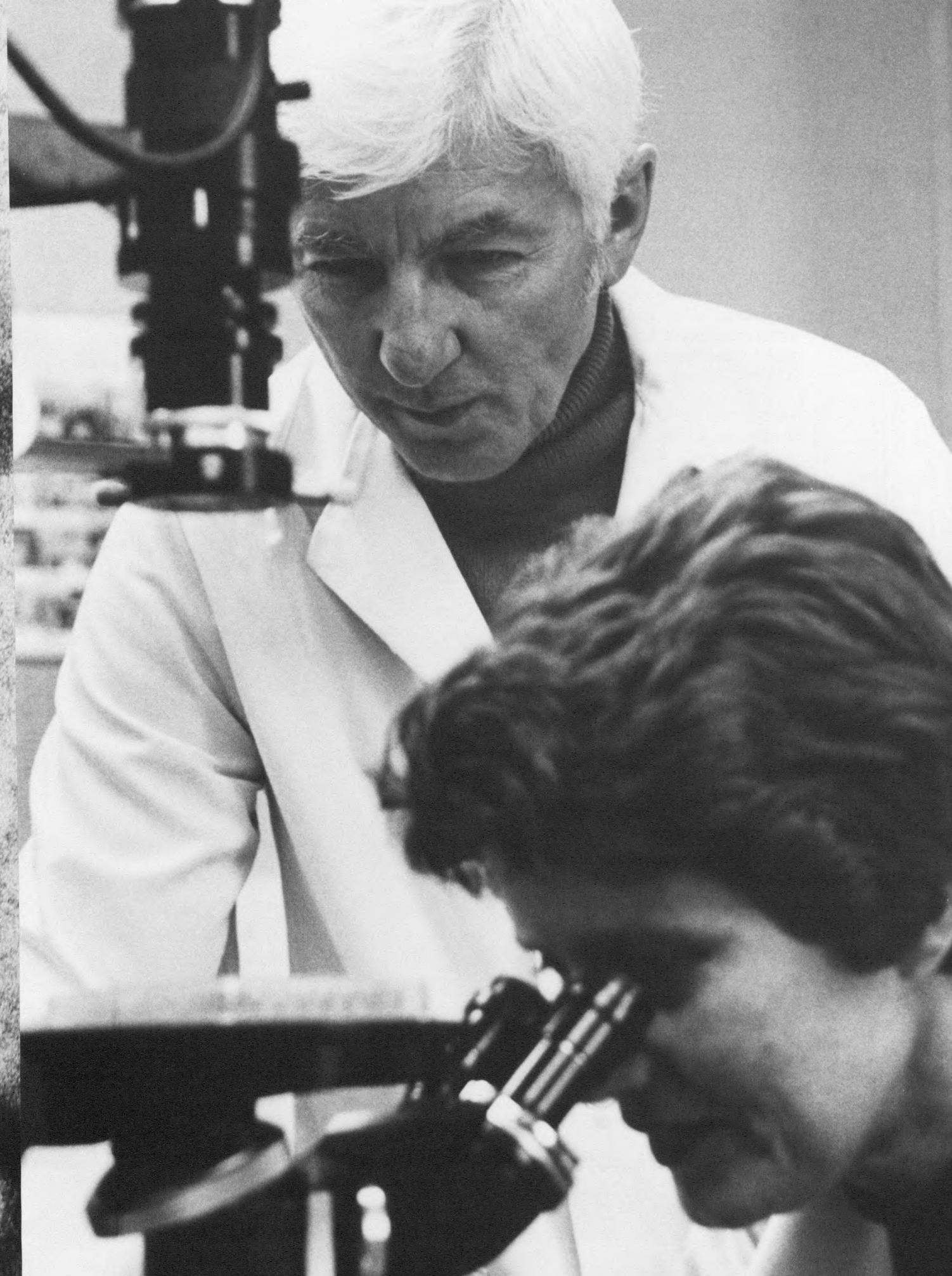


The diagnostic effort should include at least screening of sera by the rapid slide-agglutination test (SAT). A negative result with this test is reliable, but a positive result is not. More than half of SAT-positive sera contain antibodies that cross-react with antigens shared by other organisms. Sera positive by the SAT always require additional study. A two-stage screening of all SAT-positive sera is recommended. The tube agglutination test (TAT), with or without the addition of 2-mercaptoethanol, is a valuable complementary procedure. Further analysis of SAT- and TAT-positive sera by ID tests provides the best chance for accurate diagnosis. This procedure is especially important in chronically infected dogs, where attempts to isolate *B. canis* are often unsuccessful. We are now trying to isolate, separate, and purify *B. canis* cell wall antigens to improve the specificity of ID tests for canine brucellosis.

We continue to investigate the mechanisms whereby the growth of bovine herpesvirus-2 (mammillitis virus) is restricted under various environmental and physiological conditions. Research by Dr. Letchworth has demonstrated that the virus can grow at skin temperatures but not at internal body temperatures. Reasons for this fact are becoming apparent as we explore local cell-mediated immune-response mechanisms under controlled conditions. We observed diminished immune functions such as monocyte chemotaxis, lymphocyte blastogenesis, and production of interferon at temperatures normally prevailing in the skin.

Leland E. Carmichael

Left: Canine parvovirus, magnified ×165,000



Daynemouth Laboratory for Canine Nutrition

The Daynemouth Laboratory continued to study the role of vitamin E and selenium (Se) in canine nutrition. Earlier findings were substantiated: dogs deficient in vitamin E and Se either did not respond or responded poorly to vaccination with distemper and infectious canine hepatitis vaccines. In vitro studies undertaken by Dr. Langweiler revealed that this weak response was associated with the presence of a factor (or factors) in the serum of E-Se-deficient dogs that inhibits the proliferation of canine lymphocytes in cultures containing the phytomitogen PHA. Inhibition was reversed by adding vitamin E to the diet or to the serum of deficient dogs. These studies continue as we attempt to isolate, purify, and identify the suppressor factor (or factors).

Two other phenomena were observed in E-Se-deficient dogs: dermatoses and retinal atrophy. Skin lesions developed in dogs fed deficient diets that were high in polyunsaturated fats. Giving vitamin E or Se could prevent or delay the development of disease but was less effective in animals with established lesions. Dr. Riis demonstrated a novel abnormality, central retinal atrophy, in E-Se-deficient dogs. Histological examination revealed the presence of lipid deposits in the retinal epithelium and degeneration of photoreceptors. The animals were night-blind.

In studies of lymphocytes from E-Se-sufficient dogs the addition of vitamin E to the diet in amounts larger than published minimum requirements significantly increased the responsiveness of lymphocytes to mitogens. This observation has encouraged us to begin an investigation of the effects of vitamin E and Se supplementation on the response of puppies to distemper and infectious hepatitis vaccination.

Our nutrition research also addressed a practical question of dog breeders: Can vitamin C prevent or cure hip dysplasia? The vitamin C studies were undertaken in collaboration with Dr. Lust and his associates in the Biochemistry Laboratory for the Study of Canine Hip Dysplasia. Dogs can synthesize vitamin C in amounts sufficient to meet their normal metabolic requirements. Labrador retrievers fed a diet lacking vitamin C maintained constant levels of the vitamin in their plasma and tissues and excreted an average of 0.87 mg of vitamin C per kg of body weight in their urine.

Addition of 1 g of vitamin C per kg of body weight per day to the diet increased the plasma level of C but did not increase its concentration in tissues. Evidently, much of the vitamin C in the diet was either metabolized, excreted in the urine, or not absorbed. Most important, dietary vitamin C had no beneficial effect in preventing the development of hip dysplasia or the osteoarthritis associated with this disease. Likewise, immune-response mechanisms were either not favorably affected or were depressed by vitamin C. Thus recommendations that canine diets be supplemented with vitamin C for the purpose of preventing hip dysplasia or improving immune responsiveness are not justified.

Ben E. Sheffy

Biochemistry Laboratory for the Study of Canine Hip Dysplasia

Canine hip dysplasia is an inherited developmental malformation of the coxofemoral joints. A characteristic feature of the disease is hip joint looseness, a term that is used synonymously with joint laxity, subluxation, or malarticulation. Joint looseness can be judged by manipulating the femoral head. Displacement of the femoral head can also be demonstrated by X-ray examination of the hip joint.

Our research this year centered on providing a comprehensive description of hip joint laxity in Labrador retrievers that show radiological evidence of hip dysplasia. We observed that loose joints contain an abnormally large amount of synovial fluid. The accompanying table indicates that the volume of synovial fluid is related to the degree of hip joint laxity as judged by manipulation of the joint and radiological evidence of displacement of the femoral head.

Degree of Laxity	Volume of Synovial Fluid (ml)
Normal	0.2
Mild	0.5
Moderate	1.2
Severe	4.5

Our examination of the relationship between synovial fluid volume and hip joint laxity led to some astonishing findings. We observed that the hip joint became tighter when the synovial fluid is removed. Indeed, some dysplastic joints showed normal laxity when the fluid was aspirated through a needle inserted into the joint space. Removing the fluid apparently creates a vacuum that prevents displacement of the femoral head. In circumstances where the joint remained abnormally loose after fluid was removed, we found that the round ligament was stretched and swollen.

These findings encourage speculation about the processes underlying the development of hip dysplasia. An increase in synovial fluid in the hip joint might allow the femoral head to move more freely, setting in train processes that result in the destruction of tissue and the development of osteoarthritis. These processes occur in dogs genetically predisposed to the disease. How this genetic makeup is expressed in the structure and function of the hip joint and what factors favor the accumulation of synovial fluid in dysplastic joints are the subjects of continuing studies.

George Lust

Hadley C. Stephenson Laboratory for Study of Canine Diseases

Infectious enteritis in dogs has been ascribed to at least two viruses, a canine coronavirus (CCV) and a canine parvovirus (CPV). Several outbreaks of gastroenteritis linked to infection by CCV were reported in the United States this year, and CPV was isolated from dogs with diarrhea, fever, and leukopenia. In light of these reports, we are undertaking a thorough study of canine virus–induced enteritis. Our immediate objectives are to ascertain the prevalence of infection, the mode of spread of CCV and CPV, the persistence of these viruses in infected dogs, and the immune-response mechanisms associated with recovery from infection. We must define the dimensions of the problem in order to develop effective vaccines for disease prevention.

Distemper, of course, is far more serious than viral enteritis. Effective vaccines against distemper are widely used, yet cases continue to be reported. A major problem has been the difficulty of accurately diagnosing distemper when its only sign is encephalitis. Diagnosis often depends on the results of serological tests. We recently identified a specific antibody to canine distemper virus (CDV) in the IgM fraction of serum up to three weeks after vaccination or up to three months after infection with virulent CDV. We are now trying to develop sensitive methods to measure this antibody so that we can provide a practical and reliable test for diagnosing distemper.

We are also investigating the role of cell-mediated immunity in host resistance to distemper. Laboratory tests have shown that a particular class of lymphocytes (Tc) can kill CDV-infected cells in an immunologically specific way. We also found that other lymphocytes can kill infected target cells by a mechanism that does not depend on the sharp degree of recognition required by Tc. These "natural killer cells" are present even in dogs that have never been exposed to CDV.

A different mechanism of protection involving interferon could be important in many virus infections, including distemper. Experiments undertaken by Dr. Tsai indicate that dogs can produce interferon in response to stimulation by a synthetic polynucleotide or infection with Newcastle disease virus. We are now producing large amounts of canine interferon to use in ascertaining the part played by interferon in resistance to CDV.

There have been two reports in the scientific literature that claimed a relationship between dog ownership and multiple sclerosis. Although the supporting evidence is weak, the matter is of deep concern to dog owners. Therefore we have undertaken an epidemiological and serological study of the response of humans to the more common canine viruses.

Max J. G. Appel

Intestinal parasites are responsible for some of the most common and debilitating diseases in animals and humans. Our research is directed to preventing such diseases by developing effective vaccines. A necessary step is to identify antigens that can induce an immune response. It also is important to identify stages in the life cycle of parasites that are vulnerable to attack by antibodies or cells. The problem has been studied in rats infected with *Trichinella spiralis,* the parasite responsible for trichinosis.

We have shown that exposure to either the larval or adult stages of *T. spiralis* can protect animals against infection. The immunity induced by larvae is effective mainly against larvae, and that induced by adult worms is effective against the adult. However, both responses are expressed in the expulsion of worms from the small intestine. This finding suggests the existence of at least two antigens that can induce a protective response.

Two other responses have been identified by immunizing with defined stages in the life cycle of *T. spiralis:* one response impairs reproduction by female worms and another (rapid expulsion) prevents the establishment of larvae in their intestinal niche. These processes act synergistically to produce a high level of resistance.

Knowledge of immune processes gives us only the skeleton of a response pattern; understanding the mechanisms of these processes provides the flesh necessary for full comprehension. We are analyzing the mechanisms responsible for each of the four defense responses identified in animals immunized against *T. spiralis.*

Our studies so far have focused on rapid expulsion. This dramatic response can result within a few hours in the expulsion of up to 90 percent of the worms in a challenge inoculum. In experiments using parabiotic rats we demonstrated that rapid expulsion is an immunological response; it cannot be ascribed merely to a change in the normal function of the intestine. Parabiotic rats are animals that are surgically united and hence share a common blood circulation. Rapid expulsion can be transferred from one parabiont to another under these unusual conditions only when the intestine of the unimmunized partner is subject to a second stimulus. This stimulus is provided by *T. spiralis* or by the antigenically different parasite *Heligmosomoides polygyrus.*

This finding and more recent observations suggest that rapid expulsion involves two distinct processes: an immunological process in which either antibodies or cells are involved and a process that depends on irritation of the intestine and has no immunological basis. We are continuing to study this fascinating phenomenon with a view to identifying both the immunological component and the nature of the nonspecific stimulus required for the full expression of this defense.

Robin G. Bell

Above: Thomas W. Jungi measuring the influence of infection on macrophage function

Above right: The microbiology wing of the Institute, which houses modern laboratories for virus research

Right: Melissa C. Woan preparing tissue culture medium

Our research on mechanisms of acquired resistance to infection continued with the goal of ascertaining how immune animals recognize infectious agents and eliminate them from the body. These processes have been studied in rats infected with *Listeria monocytogenes,* a bacterium known for its capacity to survive and grow in macrophages. Many organisms are phagocytosed and killed in macrophages, but some can survive and grow in this potentially hostile environment. *L. monocytogenes* is a well-studied example; others include the tubercle bacillus and the bacteria that cause brucellosis and typhoid fever. But the macrophages of immune animals can kill these organisms or limit their growth, thereby arresting the progression of disease. This defense reaction depends for its full expression on the activity of T lymphoctyes, which are formed as part of the animal's immune response to infection.

Our research and that of others have shown that *L. monocytogenes* can stimulate the production of activated T cells in the lymph nodes or spleen of infected animals. These specifically sensitized lymphocytes migrate to centers of infection, where they are stimulated by microbial antigens to release a variety of molecules that have powerful effects on macrophages. Some attract macrophages and move lymphocytes to the reaction site; others enhance the capacity of macrophages to kill ingested organisms.

A major goal of our research is to understand how these important processes operate, for only with this knowledge can we expect to improve the capacity of animals to defend themselves against a variety of infectious agents. The problem is being studied in rats, where inbred strains can be used to ascertain the role of genetic factors in the interaction of T cells and macrophages.

Thomas W. Jungi

Recovery from many viral infections depends on the host's capacity to destroy infected cells. Macrophages and several classes of lymphocytes can recognize and kill cells that harbor virus and express viral antigens on their surface. But the relative importance of each of these types of protective cells has not yet been determined, nor have the conditions been defined that favor the production of protective cells. The problem has been studied in hamsters and rabbits infected with vaccinia virus, the vaccine agent used to protect humans against smallpox. Methods were developed for measuring the protective capacity of various types of cytotoxic effector cells.

Our data indicate that infection with a low dose of vaccinia virus stimulates a cell-mediated immune response in which lymphocytes of the T cell class operate as specific mediators of cytotoxicity. In contrast, infection with a large dose of virus stimulates the formation of both cytotoxic T cells and "natural killer cells." The latter are present in the tissues of normal, nonimmunized subjects as well as in the tissues of immune animals. Further studies are under way to ascertain the part played by various types of cytotoxic cells in resisting infection.

Melissa C. Woan

Osteoarthritis, often associated with canine hip dysplasia, is a major cause of discomfort and loss of function in affected animals. As part of our investigation of this disease we are using the electron microscope to study the ultrastructural morphology of the synovial membrane from the joints of dysplastic dogs. Earlier studies with the light microscope showed that a layer of cells on the surface of the membrane facing the joint cavity thickens and forms fingerlike projections (villi) that protrude into the joint space.

Using the electron microscope, we identified two distinct cell types in this region of the membrane. The most conspicuous was a fibroblast that has an extensive network of internal membranes, the endoplasmic reticulum. These cells may be the source of hyaluronic acid, found in high concentration in synovial fluid. The suggestion is based on the knowledge that cells with a well-developed endoplasmic reticulum often secrete large amounts of protein, and the suspicion that hyaluronic acid is secreted in a similar way. The second cell type had the structural characteristics of a macrophage and was found in smaller numbers in the membrane. The purpose of our study is to determine the relative numbers of these two cell types in the normal synovial membrane and in the membranes of diseased joints. By documenting these relationships and studying other changes in the membrane, we expect to gain insight into pathogenic mechanisms underlying the development of osteoarthritis.

The electron microscopy laboratory also collaborated with other laboratories in the Institute in studies of canine distemper encephalitis and viral-induced gastroenteritis of dogs. Indeed, our finding two distinct viruses in fecal specimens was an early indication that more than one virus was responsible for the outbreaks of gastroenteritis described elsewhere in this report. One of these viruses had the structural characteristics of a coronavirus; the other was a parvovirus. These studies continue as part of the Institute's effort to define these diseases and develop practical methods for their prevention.

Helen A. Greisen

Advisory Council

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 Mr. and Mrs. Gaylord Donnelley, Mrs. Priscilla Maxwell Endicott, Mr. John M. Olin, Mr. William F. Stifel, and Mr. Robert W. Woodruff.

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Publications for the first ten years are listed in the Institute report for 1960. Those for each year thereafter appear in the annual report for that year. Since 1960, articles have been numbered consecutively. Some of the following publications have been listed in a previous year's report as *in press*. They are repeated this year, with their original numbers, to record full bibliographic details. Articles completed during the past year are those numbered 431 to 459.

394 **Jungi, T. W.:** 1978. Evaluation of various filter membranes for Boyden-type leukocyte migration studies. *J. Immunol. Methods,* 21:373–82.

395 **Jungi, T. W.,** and **McGregor, D. D.:**1978. Impaired chemotactic responsiveness of macrophages from gnotobiotic rats. *Infect. Immunity,* 19:553–61.

396 **Jungi, T. W.,** and **McGregor, D. D.:** 1978. Activated lymphocytes trigger lymphoblast extravasation. *Cell. Immunol.,* 38:76–83.

402 **Lust, G.,** and **Miller, D. R.:** 1978. Metabolic changes in cartilage from young dogs with degenerative joint disease. In *The aetiopathogenesis of osteoarthrosis.* Edited by G. Nuki. Pitman Medical Press, London.

403 **Lust, G., Farrell, P. W.,** and **Sheffy, B. E.:** 1978. Development of degenerative hip joint disease in young dogs. In *Models for osteoarthrosis.* Edited by G. Nuki. Pitman Medical Press, London.

413 **Lefford, M. J.,** and **McGregor, D. D.:** 1978. The lymphocyte mediators of delayed hypersensitivity: The early phase cells. *Immunology,* 34:581–90.

428 **Sheffy, B. E.:** 1978. Nutrition and nutritional disorders. In *Symposium on canine pediatrics.* The Veterinary Clinics of North America, Vol. 8. W. B. Saunders, Philadelphia.

431 **Appel, M.:** 1978. Reversion to virulence of attenuated canine distemper virus *in vivo* and *in vitro. J. Gen. Virol.,* 41:385–393.

432 **Appel, M.,** and **Bemis, D. A.:** 1978. The canine contagious respiratory disease complex (kennel cough). *Cornell Vet.,* 68 (suppl. 7): 70–75.

433 **Banta, C. A., Clemens, E. T., Krinsky, M. M.,** and **Sheffy, B. E.** Sites of organic acid production and patterns of digesta movement in the gastrointestinal tract of dogs. *J. Nutr.* In press.

434 **Bell, R. G.** Undernutrition, infection and immunity. The role of parasites. *Papua New Guinea Med. J.* In press.

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Ways of Giving

In establishing the Institute, of which the Cornell Research Laboratory for Diseases of Dogs is an important part, the Cornell University Board of Trustees authorized the Treasurer's Office of Cornell University to act as custodian of all funds given in support of the Institute. As a donor, you are thus assured your gift will achieve the maximum benefit.

There are many ways you can give to advance the work of the Institute. Some of these opportunities offer substantial income tax and estate tax benefits.

Checks. All checks should be made payable to Cornell University and mailed to:

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for the uses and purposes of the Cornell Research Laboratory for Diseases of Dogs.

Appreciated stocks. Selling appreciated stocks is almost certain to increase your taxes. You gain maximum tax benefits by giving the stocks to Cornell outright and deducting their *full* current market value as a charitable contribution, thus avoiding capital gains tax. The transaction can be completed with maximum speed and at lowest cost by following these steps:

1. Decide what securities you want to give and take the certificate to your bank or broker.
2. Inform your bank or broker that you want to make a gift of these shares or securities to Cornell University for the Institute.
3. Instruct your bank or broker to telephone the Office of University Investments, at 607/277-0022.
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Bequests. Charitable bequests provide substantial estate tax benefits. They can be made in many forms: gifts of land or buildings, securities, personal property, or cash. The University counsel of Cornell University suggests the following provision in making a bequest for dog research: "I hereby give, devise, and bequeath [*description of property*] to Cornell University, an educational corporation located at Ithaca, New York, for the uses and purposes of the Cornell Research Laboratory for Diseases of Dogs."

Deferred giving — income-producing trusts. An income-producing trust enables you to make a meaningful gift to the Institute, gain spendable income for life, and derive important tax benefits. A beneficiary may be named to receive this income, too. The Institute can offer three plans: the Pooled Life Income Fund for gifts of $5,000 or more, the Annuity Trust, and the Unitrust for gifts of $50,000 or more. Currently each plan supports an income of about 7 percent a year.

Financial planning involving deferred gifts is a highly complex subject requiring expert advice from your attorney and other specialists. If you are interested in this way of supporting the James A. Baker Institute for Animal Health, please notify the director, who will make arrangements for you to receive more specific information.

Financial Situation

To assure donors that their funds will sustain and advance research on dogs now and in the future, the Cornell University Board of Trustees made a provision for disposal of excess income as follows: "The Institute's income is in excess of its operating expense, and the balance of the funds is added to the Institute's Endowment."

July 1, 1977, to June 30, 1978

Funds Available

Gifts and earnings budgeted	$208,855
State of New York general support	122,587
State of New York dog license fees	144,000
Federal grants	254,781
	$730,223

Expenditures

Salaries	$607,240
Operational costs	211,566
	$818,806
Reserves used to balance budget	$ 88,583

Research that involves species other than dogs is supported by other sources.

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