· ANNUAL REPORT 1996 ·

The James A. Baker

INSTITUTE FOR ANIMAL HEALTH

COLLEGE OF VETERINARY MEDICINE · CORNELL UNIVERSITY

Annual Report, 1996, Volume 46

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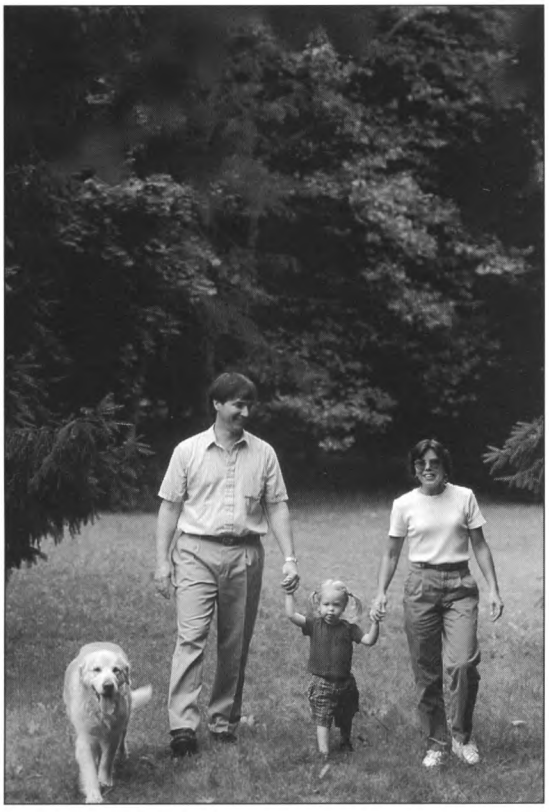
ANNUAL REPORT 1996

The James A. Baker INSTITUTE FOR ANIMAL HEALTH



A Message from the Director

THE CAPACITY of the human spirit for hard work and dedication is a continuing source of inspiration. Here at the James A. Baker Institute for Animal Health we have our own contributions to the record of progress, and it is my pleasure to highlight some of them in this message. Since our last report was published one year ago, the Institute's total staff numbers have increased by 25 percent, with virtually all of this increase in the scientific research staff. This increase in staff reflects the vigor of our research programs, which are now larger and stronger than at any time in the nearly 50 years of the Institute's history.

Our growth is all the more remarkable when considered in light of the decreasing support of medical and scientific research by the federal and state governments. To our many friends and supporters we owe a tremendous debt of gratitude.

The explosive increase in the size and scope of the Institute's scientific programs during the past three years has made necessary the expansion of our laboratory facilities. In the past year the Institute's new Molecular Biology Laboratory has become a reality. A most generous grant from the Mrs. Cheever Porter Foundation has allowed us to purchase a sophisticated new gene analyzer, which is the centerpiece of the new laboratory.

Also in 1996 the Baker Institute's Scientific Conference Series was initiated with a landmark meeting on canine hip dysplasia. Details of this meeting, which was organized by Institute

Opposite: **Doug Antczak and Jane Miller**

scientist Dr. George Lust, can be found on pages 30 and 38 of this report.

Nationally, there is a lively debate on the value of animal research to the betterment of human health. As a veterinary research institute, we are part of this debate, and our daily experience gives us a unique perspective on this topic. The mission of the Baker Institute is to improve animal health through basic and applied research. For nearly 50 years the majority of the Institute's research has been conducted "on the dog, for the dog," as former Institute veterinarian Hadley Stephenson was wont to say. This is still true today, as you will read elsewhere in this report.

The equation is not a simple one, however, and the solutions are often surprising. For example, in 1996 Dr. Max Appel and the Baker Institute were recognized by the American Association of Zoo Veterinarians for Dr. Appel's outstanding contributions to infectious disease diagnosis and control in free-living and captive wildlife species. His work included identifying the strain of canine distemper virus that devastated the lion population of the Serengeti Plain in East Africa. Could this important application have been imagined when the first vaccine for canine distemper was developed at the Institute in the early 1950s? I think not. Dr. Appel has had a career-long interest in this disease of dogs that has been successfully controlled by vaccination for over four decades. Could he or anyone else have known that his knowledge and experience would be called upon in service of the noble lion?

Other similar examples abound in the research conducted at the Baker Institute. The National Institutes of Health support seven research programs at the Institute that focus on diseases of dogs, horses, and humans. Which species benefits most from this research? It is my view that we all benefit, humans and animals alike, and that a spirit of cooperation and reason is essential for continued advances in human and animal health.

This year our annual report presents a new look; we trust that you will find it more readable and more informative. Our annual reports reflect the tremendous year-round efforts of our scientific and support staffs and their continuing commitment to excellence. Good reading!

— Douglas F. Antczak

Staff of the Baker Institute

ADMINISTRATION

Douglas F. Antczak
Director: B.A., Cornell; V.M.D., U. of Pennsylvania; Ph.D., Cambridge U.

Jane M. Miller
Administrative manager: B.S., M.P.S., Cornell

Susan Howell Hamlin
Facilities manager: B.S., Elmira College

Carlene M. Furch
Human resource assistant

Paul J. Lutwak
Systems analyst: B.A., B.S., Miami U.

Anita S. Hesser
Administrative aide and assistant systems administrator

Dorothy K. Scorelle
Secretary to the director: B.S., SUNY College at New Paltz

Laurie A. Lychalk
Office assistant

Patricia A. LaLonde
Accounts coordinator: B.S., St. John Fisher College

Sharon E. Morrow
Accounts assistant

Jeanne Griffith Truelsen
Public affairs coordinator: B.A., M.A., Miami U.

Judith L. Mordue
Public affairs assistant

EMERITUS

Ben E. Sheffy
Caspary Professor of Nutrition, Emeritus: B.S., M.S., Ph.D., U. of Wisconsin

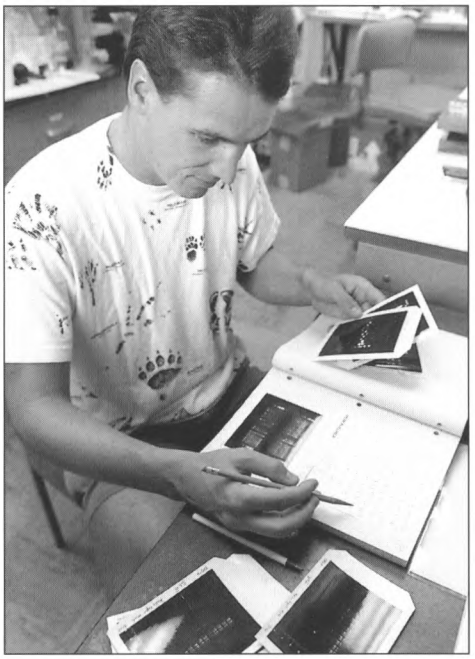
LABORATORIES

GIRALDA LABORATORY FOR CANINE INFECTIOUS DISEASES

Leland E. Carmichael
John M. Olin Professor of Virology: A.B., D.V.M., U. of California, Davis; Ph.D., Cornell; PhD*(hc)*; Diplomate, American College of Veterinary Microbiologists

David N. Peters
Graduate research assistant: D.V.M., Ohio State U.

Araceli Lucio Zavaleta
Laboratory technician: B.S., Cornell



Michael Olivier

HADLEY C. STEPHENSON LABORATORY FOR THE STUDY OF CANINE DISEASES

Max J. G. Appel
Professor of Virology: Dr.med.vet., U. of Hannover; Ph.D., Cornell

Luc Härter
Postdoctoral associate: Ph.D., C.A.U. Kiel

Alix F. Straubinger
Postdoctoral associate: Dr.med.vet., U. of Munich

Reinhard Straubinger
Graduate research assistant: Dr.med.vet., U. of Munich

Mary Beth Matychak
Research technician: U. of Evansville

ALBERT C. BOSTWICK LABORATORY OF MOLECULAR BIOLOGY

Colin R. Parrish
Associate Professor of Virology: B.Sc., Massey U.; Ph.D., Cornell

Martha J. Harding
Postdoctoral associate: D.V.M., U. of Guelph; Ph.D., U. of Minnesota

John S. L. Parker
Graduate research assistant: B.V.M.S., U. of Glasgow

Dai Wang
Graduate research assistant: B.S., Nankai U.

Wen Yuan
Graduate research assistant: M.S., Peking U.

Gail M. Sullivan
Laboratory technician: A.A.S., SUNY Agricultural and Technical College at Canton; B.A., SUNY College at New Paltz

Wendy S. Weichert
Laboratory technician: B.S., Cornell

JOHN M. OLIN LABORATORY FOR THE STUDY OF CANINE BONE AND JOINT DISEASES

George Lust
Professor of Physiological Chemistry: B.S., U. of Massachusetts; Ph.D., Cornell

Nancy Burton-Wurster
Senior research associate: B.A., M.S., PhD., New York U.

Chih-Tung Chen
Postdoctoral associate: B.S., National Taiwan U.; M.S., Ph.D., U. of Wisconsin, Madison

Lisa A. Fortier
Graduate research assistant: B.S., D.V.M., Colorado State U.

Michael Olivier
Graduate research assistant: M.S., U. of Cologne

Rina Gendelman
Graduate research assistant: B.S., SUNY at Stony Brook

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Laboratory technician: B.S., State U. of Leiden; B.S., U. of Amsterdam; M.S., State U. of Leiden

Elizabeth Grisanzio
Laboratory technician: B.S., U. of Vermont

Alma J. Williams
Laboratory technician: B.A., U. of Pennsylvania; M.S., Cornell; AALAS accreditation

Sharon Morrow

LABORATORY OF CELLULAR GROWTH AND DIFFERENTIATION

James N. MacLeod
Assistant Professor of Molecular Genetics: B.S., U. of Delaware; V.M.D., Ph.D., U. of Pennsylvania

Da-Nian Gu
Research associate: B.S., M.S., Ph.D., Fu Dan U., Shanghai

Matthew C. Stewart
Graduate research assistant: B.V.Sc., U. of Sydney

Jonathan W. Tetreault
Laboratory technician: B.S., Clarkson U.

IMMUNOLOGY LABORATORY

Robin G. Bell
Professor of Immunology: B.Sc., Australian National U.; Ph.D., John Curtin School of Medical Research

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Deborah Negrao-Correa
Graduate research assistant: B.S., U. Estadual de Campinas; M.S., U. Estadual de São Paulo

Jeb B. Oblak
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Lincoln S. Adams
Research technician: B.S., Hobart College; AALAS accreditation

MUCOSAL IMMUNITY LABORATORY

Judith A. Appleton
Associate Professor of Immunology: B.S., Indiana U.; M.S., Ph.D., U. of Georgia

Barbara A. Butcher
Research associate: B.S., Gannon U.; M.S., U. of Pittsburgh; Ph.D., U. of New Mexico

Trenna L. ManWarren
Postdoctoral associate: B.S., D.V.M., Cornell

Catherine S. McVay
Postdoctoral associate: B.S., M.S., Auburn U.; Ph.D., Texas Technical U.

Lucille F. Gagliardo
Laboratory technician: B.S., Southhampton College

EQUINE GENETICS CENTER

Douglas F. Antczak
Dorothy Havemeyer McConville Professor of Equine Medicine

Christopher J. Davies
Instructor: B.S., D.V.M., Ph.D., Cornell

Maria M. Viveiros
Postdoctoral associate: B.Sc., McMaster U.; M.Sc., Ph.D., U. of Guelph

Jessica M. Baker
Graduate research assistant: B.S., Cornell

Wayne L. Gottlieb
Laboratory technician: B.A., California State U., Northridge; M.S., U. of California, Davis

Edward B. Han
Laboratory technician: B.A., Cornell

Todd J. Patton
Laboratory technician: B.S., U. of Wisconsin, Madison

LABORATORY FOR THE STUDY OF INHERITED CANINE REPRODUCTIVE DISEASES

Vicki N. Meyers-Wallen
Associate Professor of Reproduction: B.S., U. of Maryland; V.M.D., Ph.D., U. of Pennsylvania; Diplomate, American College of Theriogenologists

Christine M. Schweizer
Postdoctoral associate: B.S., D.V.M., Cornell

DONNELLEY LABORATORY OF GENE REGULATION AND EXPRESSION

Jharna Ray
Assistant Professor of Molecular Genetics: B.S., M.S., Ph.D., U. of Calcutta

Maria E. Verdugo
Research associate: M.D., Central U. of Venezuela

Fuliang Du
Graduate research assistant: B.S., Nanjing Normal U.

Wei Sun
Graduate research assistant: M.D., Tianjin Medical U.

Virginia M. Scarpino
Laboratory technician: B.S., Edinboro State College; M.A., SUNY College at Geneseo

INHERITED EYE DISEASE STUDIES UNIT

Gustavo D. Aguirre
Alfred H. Caspary Professor of Ophthalmology: V.M.D., PhD., U. of Pennsylvania; PhD*(hc)*; Diplomate, American College of Veterinary Ophthalmologists

Gregory M. Acland
Senior research associate: B.V.Sc., U. of Sydney; Diplomate, American College of Veterinary Ophthalmologists

Kunal Ray
Senior research associate: B.S., M.S., Ph.D., U. of Calcutta

Weikuan Gu
Research associate: B.S., U. of Hebei Agri, China; M.S., Ph.D., Cornell

Qi Zhang
Postdoctoral associate: M.S., Shanghai Medical U.; M.D., Harbin Medical U.; Ph.D., Cornell

Weiquan Wang
Graduate research assistant: M.D., West China U.

Caroline J. Zeiss
Graduate research assistant: B.V.Sc., U. of Pretoria

Susan E. Pearce-Kelling
Research support specialist: B.S., M.S., Cornell

Julie A. Alling
Laboratory technician: A.S., Tompkins-Cortland Community College

Victoria J. Baldwin
Laboratory technician: B.A., Colby College

ANIMAL CARE

Raymond M. Combs
Vivarium co-supervisor: AALAS accreditation

Raymond J. Corey
Vivarium co-supervisor: A.A.S., SUNY Agricultural and Technical College at Delhi; AALAS accreditation

Kevin T. Draiss
Animal technician: A.A.S., SUNY Agricultural and Technical College at Delhi; B.S., Cornell; AALAS accreditation

Stephanie Gardner
Animal technician: B.A., St. Michael's College

James C. Hardy
Research aide: B.S., Cornell; AALAS accreditation

Kevin Draiss

Clint N. Kellogg
Animal technician

Victor L. Maine
Animal technician

Heidi Maynard-Kretz
Animal technician: B.S., Cornell

Rita H. Sims
Animal technician: AALAS accreditation

MAINTENANCE

Arthur D. Howser
Farm manager

Richard E. Daniels
Maintenance mechanic

Russell F. Haus
Maintenance mechanic

Jeannette R. Sorge
Laboratory attendant

Edward H. Thompson, Jr.
Laboratory attendant

Rita Sims

RUSS HAMILTON

Perspectives

WHEN DR. JAMES A. BAKER established the Veterinary Virus Research Institute almost 50 years ago, he built a dog kennel, some isolation facilities, and two laboratories. In those days, a scientist could investigate most problems with a microscope and a room to put it in.

Times have changed. The Institute has grown continuously since 1950, in both the scale and the complexity of its research undertakings. There has been a constant increase in the size and sophistication of the staff needed to conduct and support this research. The faculty have been deservedly successful in winning peer-reviewed funding for their work. That research has also been furthered to a very great extent by the thousands of dog owners, veterinarians, and groups who have loyally supported the Institute with their generous contributions. All of these essential factors have combined to produce an unprecedented and ever-growing list of major advances in animal health, especially for the benefit of dogs.

The potential for future discoveries at the Institute is enormous. Continued growth in its research programs is a certainty. Unfortunately, it is equally certain that the buildings themselves will not be adequate in space or design to accommodate that progress. The Institute's physical plant has evolved as a patchwork of additions to the original facilities built by Dr. Baker. The buildings are aging and full to capacity. Planning is already underway to address the first necessity, to replace the original kennel building, which has become too costly and inefficient to maintain. For the same reasons, we can foresee the need to replace the main laboratory building within the next five to ten years.

The scientific staff of the Baker Institute has worked at the leading edge of technology since the Institute's inception. In the coming years, the quality of its research facilities will be critical to the Institute's ability to make the most of increasingly rapid advances in technology, and to continue to attract the top-quality faculty and students needed to sustain the momentum of discovery. It is vital that we begin to look now at the needs—and the tremendous promise—of the Institute's second half-century.

—Henry J. Travis, D.V.M.
Advisory Council Chairman

Advisory Council

Henry J. Travis, D.V.M., *Chairman*
General Practitioner, Huntington, New York

Stephen H. Blose, V.M.D., Ph.D.
President, pdi, Huntington Station, New York

Sarah R. Bogdanovitch
Lake Clear, New York

Albert C. Bostwick, Jr.
Aiken, South Carolina

Philip B. Carter, Ph.D.
Professor of Microbiology and Immunology, College of Veterinary Medicine, North Carolina State University at Raleigh

Gerald J. Chader, Ph.D., M.D. (*hc*)
Chief Scientific Officer, The Foundation Fighting Blindness, Hunt Valley, Maryland

Richard P. Henry, D.V.M.
General Practitioner, Deer Park, New York

Joseph W. Jones
Chairman, The Robert W. Woodruff Foundation, Atlanta, Georgia

Patricia Kaneb
President, Priscilla of Boston, Boston, Massachusetts

Robert R. Marshak, D.V.M., D.V.M. (*hc*)
Dean Emeritus, School of Veterinary Medicine, University of Pennsylvania

Frederick A. Murphy, D.V.M., Ph.D., M.D. (*hc*)
Professor, Department of Pathology, Microbiology, and Immunology, School of Veterinary Medicine, University of California, Davis

Roy V. H. Pollock, D.V.M., Ph.D.
Vice President, Companion Animal Division, Pfizer Animal Health, Exton, Pennsylvania

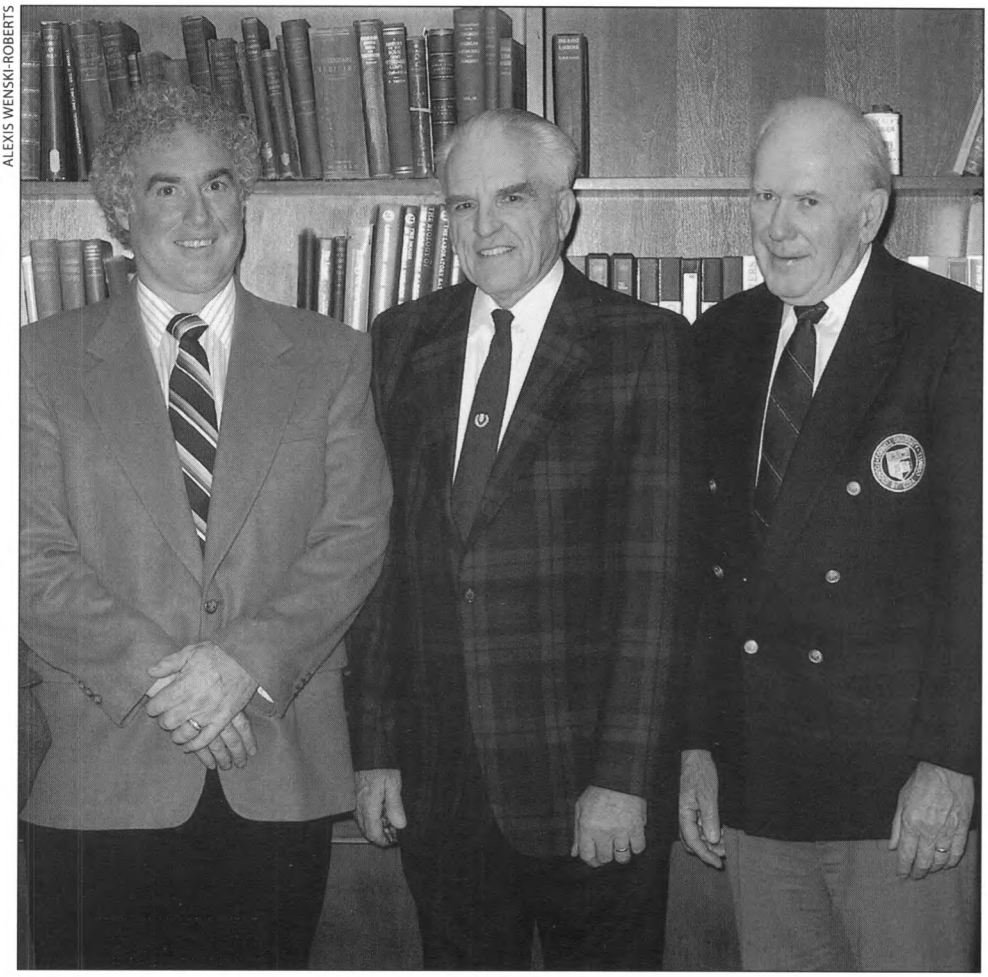
Gene M. Pranzo
President, The Dorothy Russell Havemeyer Foundation, New York, New York

Andrew G. C. Sage II
Glen Cove, New York

Robert E. Shope, M.D.
Department of Pathology, University of Texas Medical Branch, Galveston

Judith Wilpon
Locust Valley, New York

ALEXIS WENSKI-ROBERTS



From left: **Dr. Henry J. Travis, chairman of the Baker Institute's Advisory Council, with two of this year's honorees, Dr. Robert W. Kirk, the recipient of the North Award, and Dr. Robert E. Clark, who received the Founders' Award.**

FORMER ADVISORY COUNCIL MEMBERS

William C. Beck, M.D., F.A.C.S.
1982-1994

Dorothy R. Donnelley
1980-1992
Chairwoman, 1982-1988

Strachan Donnelley, Ph.D.
1990-1995

G. Watts Humphrey, Jr.
1982-1989

Richard M. Johnson
1977-1989

John A. Lafore, Jr.
1978-1984

Gary Lee
1977-1986

Irwin H. Lepow, M.D.
1978

John M. Olin
1977-1982

Niel W. Pieper, D.V.M.
1977-1993

William Rockefeller
1979-1990
Chairman, 1989-1990

Frances G. Scaife
1978-1988

Robert Winthrop
1982-1990

Robert Winthrop II
1984-1994

Recognitions

THE ARTHUR F. NORTH, JR. CANINE SERVICE AWARD

The North Award, established in 1982 to honor the memory of Dr. Arthur F. North, a 1935 graduate of Cornell's College of Veterinary Medicine, recognizes those whose contributions to canine health and well-being reflect his spirit of concern for all dogs.

This year's North Award honors a colleague, **Dr. Robert W. Kirk**, whose name is probably known to every small-animal practitioner who has reached for the bookshelf since the 1960s for help in treating a medical problem. Until his recent retirement Dr. Kirk edited the indispensable guide, *Current Veterinary Therapy*, and also co-authored and edited the *Handbook of Veterinary Procedures and Emergency Treatment*. Among academicians he is also widely known as a founder of the field of veterinary dermatology.

Dr. Kirk graduated from Cornell's College of Veterinary Medicine in 1946 but returned to Cornell as a member of the faculty in 1952. He was named director of the Teaching Hospital in 1985. He has received many prestigious awards honoring his contributions to veterinary practice, teaching, and research, including New York State Veterinarian of the Year in 1971, the gold Centennial Medallion of the New York State Veterinary Medical Society in 1990, and designation as an honorary associate of the Royal College of Veterinary Surgeons in 1993. We had the pleasure of presenting the North Award to Dr. Kirk at the dinner following the annual meeting of the Institute's Advisory Council in September.

THE FOUNDERS' AWARD

The Founders' Award is given annually to a veterinarian whose contributions to the Institute and to his or her profession exemplify our founders' commitment to the advancement of veterinary medicine.

It would be hard to name anyone who knows more about commitment than this year's honoree. While running an eminent New York veterinary hospital with his partners, Drs. Ellsworth B. Thorndike and John E. Pinckney, **Dr. Robert E. Clark** has also served Cornell in a succession of volunteer leadership roles, among them the chairmanships of the Veterinary College's Development Advisory Committee, Annual Fund, and Campaign Committee, the presidency of the College Alumni Association, and membership on the University Council and Alumni Trustee Nominating Committee. He has also served as president of the New York State Veterinary Medical Society. Dr. Clark began contributing to the Baker Institute in 1955 and remains a key supporter of our research programs.

Dr. Clark was named Veterinarian of the Year in New York State in 1969. In 1991 the Alumni Association of the College of Veterinary Medicine accorded Dr. Clark its highest honor, the Daniel Elmer Salmon Award for Distinguished Alumni Service.

THE JOHN A. LAFORE, JR. KENNEL CLUB AWARD

The John A. Lafore, Jr. Kennel Club Award honors kennel clubs "for exceptional devotion to the health and well-being of dogs of all breeds."

This year's recipient of the Lafore Award, the **Collie Club of America Foundation**, pioneered the organization of breed-sponsored support for veterinary medical research. The Foundation was formed in 1986 to foster and promote an understanding of dogs and their health needs and to support research and public education, not only for the collie breed, but for all dogs.

Since 1992, the Foundation has provided substantial funding to the Institute for the study of progressive retinal atrophy in the collie. This support has been essential to the progress described later in this report by Dr. Gustavo Aguirre. The Foundation's activism has also raised awareness among dog breeders of the need to address the health problems inherent in their breeds.

Dr. Aguirre presented the Lafore award to Foundation president Helen Denton when the foundation board met in Syracuse, New York. At that meeting Dr. Aguirre also presented an update on his group's research.

AWARD RECIPIENTS

North Award

1982 Adelaide Riggs
1983 The American Kennel Club
1984 Priscilla Maxwell Endicott
1985 The Marilyn M. Simpson Charitable Trusts
1986 Frances Rowles Van Brunt
1987 The Geraldine R. Dodge Foundation
1988 Atherton Bristol
1989 Jacqueline Lindsay
1990 Dorothy Donnelley
1991 Robert Winthrop
1992 Eleanor Gillis
1993 Albert C. Bostwick, Jr.
1994 Dolly B. Trauner
1995 Barbara J. Hartsig
1996 Robert W. Kirk, DVM '46

Founders' Award

1990 Charles Fletcher, DVM '33
1991 Du Bois Jenkins, DVM '43
1992 Niel Pieper, DVM '32
1993 Harold Kopp, DVM '42
1994 G. Clayton Dudley, DVM '64
1995 John A. Ward, DVM '36
1996 Robert E. Clark, DVM '52

Lafore Award

1993 Devon Dog Show Association
1994 Ox Ridge Kennel Club
1995 Irish Setter Club of America
1996 Collie Club of America Foundation

Memorials

STANLEY M. ALDRICH, D.V.M. '50

Stan Aldrich was a Baker Institute Research Partner who supported our work very generously from 1970 until his death this year at age 74. With his wife, Dorothy, he was a Foremost Benefactor of Cornell University.

Dr. Aldrich flew B-24 bombers during the China/Burma/India Campaign, service for which he was awarded the Distinguished Flying Cross and three Air Medals for bravery. After graduating from Cornell he established the Aldrich Animal Hospital in Babylon, New York and built a highly regarded practice.

Dr. Aldrich served organized veterinary medicine in a variety of capacities from the local level to the presidency of the New York State Society and, in 1980, the American Veterinary Medical Association. He received the State Society's Distinguished Life Service Award and the AVMA Award in recognition of "distinguished contributions to the advancement of veterinary medical organizations."

MOLLIE EMERSON PARKER BUTLER, M.S. '40

In July of 1996 a new bench and associated plantings were dedicated in Cornell's Plantations, the University botanical garden that borders the Institute's McConville Barn. The bench is unique in the Plantations in that it faces away from the interior and towards the paddocks and pastures of the Baker Institute's McConville Barn. The view from the bench is thus not only of trees, shrubs, and flowering plants, but of the horses and ponies of the Equine Genetics Center herd.

CHARLES HARRINGTON / CU

Grandchildren of Mrs. Karl D. (Mollie) Butler gather for a photograph during the ceremony dedicating the Mollie Butler Memorial Bench in the Cornell Plantations.

Such a view would have suited Mollie Butler, in whose memory the bench has been dedicated. Mollie Butler was one of the premier breeders and exhibitors of Welsh ponies in America for a 25-year period until her death in 1992. Her ponies won many national championships, and the GlanNant bloodlines are still among the most sought-after in the U.S.

After her own children outgrew their ponies, Mollie began to offer lessons to local children, who carried on the Butler tradition in the show ring. In addition to her practical equine skills, Mollie was a natural scientist with a very inquisitive mind. She kept the Cornell equine vets on their toes with her perceptive and difficult questions. Her generosity to her *alma mater* included a Welsh pony stallion or two for the University's horse herds, and descendants of those stallions are among the Baker Institute's ponies. Mollie's long association with Cornell, and with the Baker Institute in particular, made the choice of site for this memorial an easy one.

HELEN ALBERTA GREISEN, Ph.D. '73

Helen Greisen, who worked as a research associate at the Institute from 1972 until her retirement in 1983, died in September, several weeks before her seventieth birthday. Dr. Greisen was the Institute's electron microscopist and a vital member of the team working with Drs. Max Appel and Leland Carmichael in the critical period after the initial outbreaks of canine parvovirus were reported.

Dr. Greisen was also an active choral singer, gardener, and mother of four. In addition to her children, she is survived by her husband, physics professor Kenneth Greisen.

LOUISE A. McBEE, PH.D. '52

Louise McBee was one of the Baker Institute's earliest staff members. She had come to Cornell in 1943 and worked in the Department of Pathology and Bacteriology until 1950, when Dr. Baker hired her as a technician for the new Veterinary Virus Research Institute. She left Cornell after receiving her Ph.D. in 1952 and later spent three years as a fellow at the School of Tropical Medicine and Hygiene in London, England. She and her late husband, John McBee, then made their home in Tipp City, Ohio.

ROBERT B. McCLELLAND, D.V.M. '34

Bob McClelland was a Founder of the Baker Institute and a friend from its earliest days. When the Cornell Research Laboratory for Diseases of Dogs was dedicated on January 5, 1951, Dr. McClelland was there to speak at a virus symposium held to mark the importance of the venture just begun. His title was, "The Need for Research on Virus Diseases of Pet Animals." When the Veterinary Virus Research Institute was re-dedicated in memory of Dr. Baker in 1975, Dr. McClelland was there again to speak.

Bob McClelland and his brother, Frank E. McClelland, Jr., were partners in the McClelland Veterinary Hospital, a landmark Buffalo, New York practice that had been acquired by their father, Dr. Frank E. McClelland, Sr., in 1915. All three men served terms as president of the New York State Veterinary Medical Society, with Bob's term beginning in 1957.

In 1995, Dr. McClelland wrote in a letter to Leland Carmichael:

RUSS HAMILTON

Dr. Helen Greisen at the electron microscope in 1979

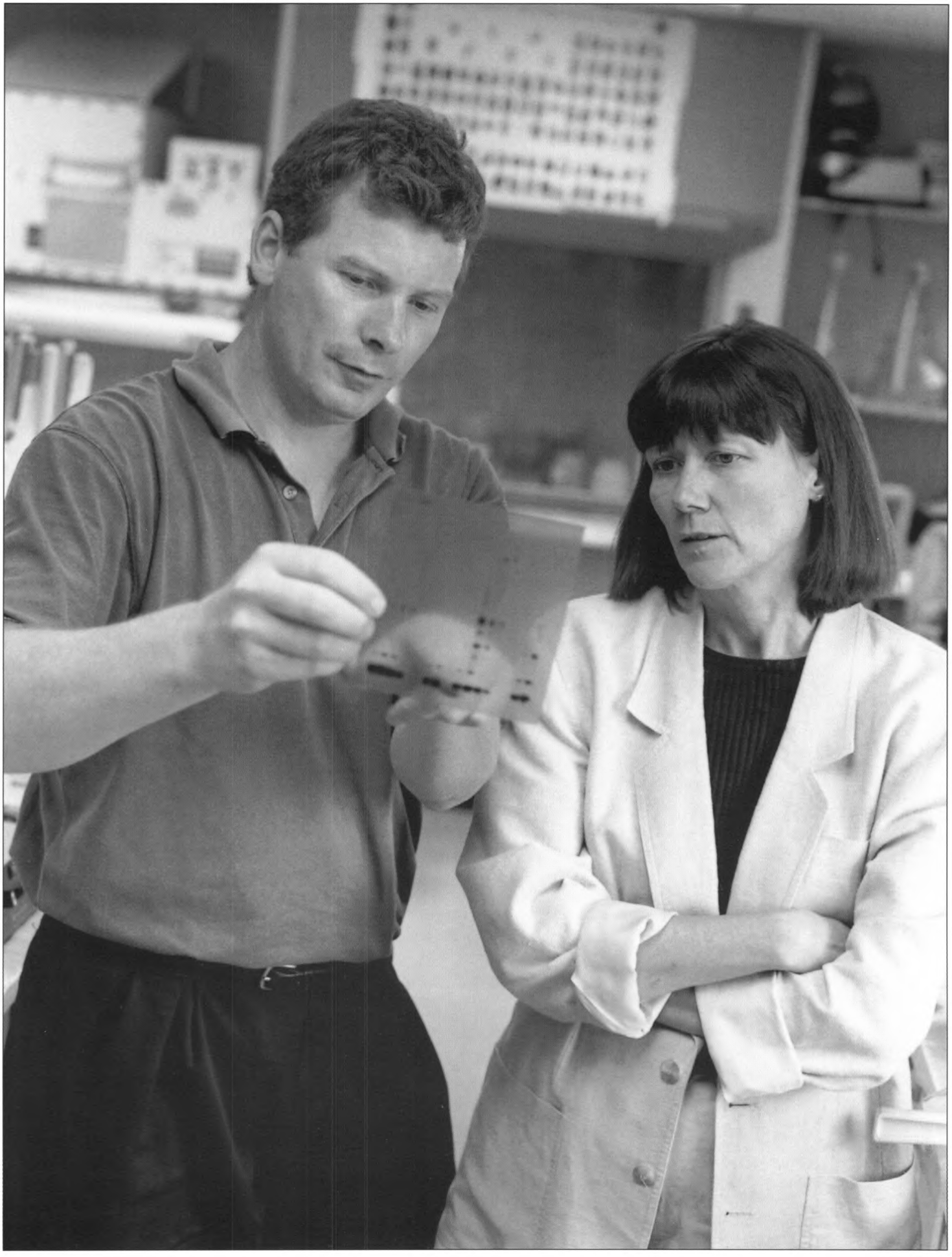
> Sixty years ago, honestly, 60 years ago, canine distemper was the foremost killer of dogs in our practice. Only those puppies that became naturally immune lived over one year. We agreed that, if it were not for distemper, we would have to depend on our equine practice to make a living. . . . Now, Leland, I understand that canine distemper is rare in this area. Thanks to the Baker Lab.

And thanks to good veterinarians like Bob McClelland.

JOHN A. WARD, D.V.M. '36

John Ward was a Research Partner and longtime benefactor of the Baker Institute. The Institute acknowledged Dr. Ward's exceptional support in naming him the recipient of the 1995 Founders' Award. Ill health prevented Dr. Ward from traveling to Ithaca for the presentation, but his delightful wife, Maryclare, made the trip from Melbourne, Florida with her daughter Lynn to accept the award on his behalf. Mrs. Ward brought along a videotaped address from Dr. Ward that is now a treasured part of the Institute's archives.

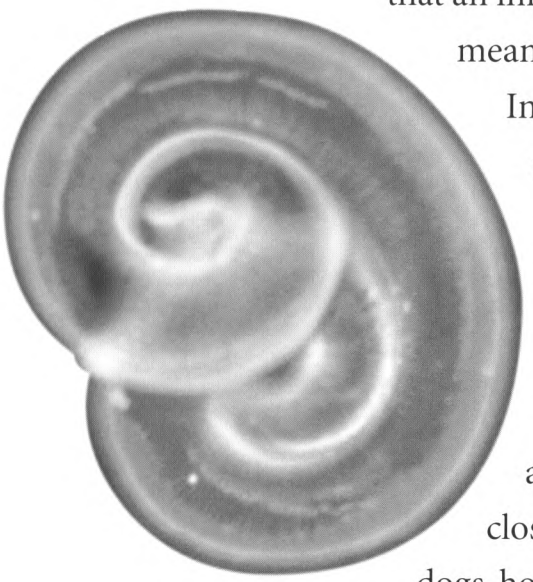
Until illness forced Dr. Ward to retire at the age of 71, he practiced in Staten Island, New York. He began his career at the newly built Staten Island Zoo in 1936. He worked at the zoo for two years, building a private, mixed practice among his neighbors in the evening hours (at that time livestock herds—and backyard pigs—were still a common sight on the island!). Dr. Ward opened his own practice in 1938, the same year that daughter Robin was born. As the family grew to include Lynn and Bethe, the Wards continued to make their home above the practice, waiting 17 years to buy a house. Their sacrifices were ultimately rewarded, and they, in turn, have rewarded Cornell most generously.



Infectious Diseases and Immunology

FOR NEARLY FIFTY YEARS the foundation of research at the Baker Institute has been our studies of infectious diseases of animals. This work encompasses two distinct parts. First is the identification and characterization of infectious agents that cause diseases of animals. These include viruses, bacteria, fungi, and parasites. Many of the important infectious agents of dogs were discovered by Institute scientists over the past half century.

Second is the study of the effect of those infectious agents in animals, and the development of vaccines that can protect animals from disease. It is often essential to understand the mechanisms that an infectious agent uses to invade a host in order to devise means to circumvent that process. Hence, research at the Institute continues to characterize the pathogenic mechanisms of viruses, bacteria, and parasites, and the components of the immune systems of horses and dogs.



The Golden Age of infectious disease research, that is, the period marked by the rapid discovery of new viruses and bacteria, and the development of vaccines and antibiotics that combat them, may be coming to a close. Importantly, however, the infections that still plague dogs, horses, and other animals, including humans, are those that have not yielded easily to vaccine development. Furthermore, new strains with antibiotic resistance and sometimes even new infectious agents continue to emerge. We must remain vigilant against the invisible world of microbes that surrounds us and threatens our own health and that of our companion animals.

Opposite: **Collin Parrish and Judy Appleton**

Above: **Photo micrograph of *Trichinella spiralis*, an important parasite of all mammals, that has been studied at the Baker Institute for two decades.**

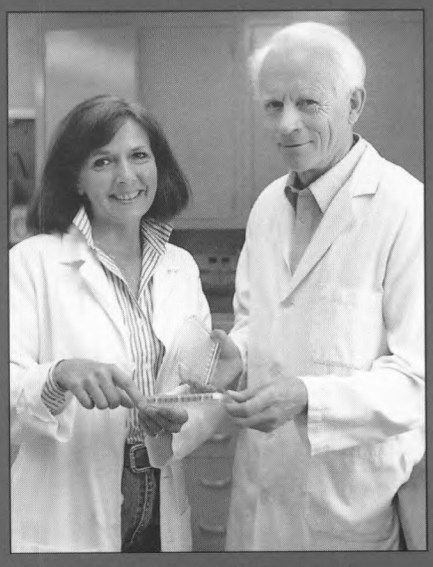
Hadley C. Stephenson Laboratory for the Study of Canine Diseases

Lyme disease manifests itself in a variety of tissues of the body, beginning with the skin and progressing to the joints, cardiovascular system, and central and peripheral nervous systems in humans and other animals. In dogs, the most prominent clinical sign is a recurrent lameness caused by acute arthritis in one or more joints. We have been interested for several years in understanding the process by which Lyme arthritis develops in dogs following the bite of an infected tick. It has generally been believed that the spirochetes that cause Lyme disease travel via the bloodstream to target organs like the joints or the brain. Our work this year has cast doubt on that assumption, at least where dogs are concerned, and likely for human infections as well. Even three to five months after exposure to infected ticks we found that dogs have a higher concentration of spirochetes in the tissues near the site of exposure than on the opposite side of the body. This finding suggests that the disease-causing organisms are migrating through tissues rather than by the bloodstream, a route that would be expected to produce a much more even distribution of organisms after a period of several months.

Reinhard Straubinger, a veterinarian and graduate student in our laboratory, together with Alix Straubinger, a postdoctoral associate and also a veterinarian, produced further evidence this year that interleukin-8 (IL-8) plays an important role in generating and sustaining a response that causes the development of acute arthritis in dogs infected with Lyme disease. IL-8 is one of a class of non-antibody proteins called cytokines that are secreted by a wide variety of cells upon contact with certain antigens. In the case of infection with the Lyme disease organism, *Borrelia burgdorferi,* spirochetes migrating into the joints stimulate IL-8 production. The IL-8 in turn attracts specialized white blood cells, called neutrophils, that induce the acute arthritis. Luc Härter, a post-doctoral associate in our laboratory, has purified cloned canine IL-8, and we have produced polyclonal and monoclonal antibodies to this cytokine for additional testing.

There is also considerable concern about other tick-transmitted diseases in dogs and humans. The deer tick, which is responsible for the transmission of *B. burgdorferi,* can also transmit additional agents, such as *Ehrlichia equii* or a close relative that causes human granulocytic ehrlichiosis. Dogs also become infected with this agent but few details are known about its effect on them. An intriguing question is whether infection in dogs with *E. equii* can mimic Lyme disease. It is also unknown whether dual infections with *B. burgdorferi* and *E. equii* pose any special risks. We may address these questions in the future.

Our findings suggest that disease-causing organisms are migrating through tissues rather than through the bloodstream.



Mary Beth Matychak and Max Appel

We have completed a study of dogs that were exposed to *B. burgdorferi* infected ticks and treated with antibiotics two months later. This study was undertaken because of conflicting reports about the efficacy of antibiotic treatment of Lyme disease in humans. We treated the infected dogs for 30 days with recommended doses of doxycycline or amoxicillin, the most commonly used antibiotics for Lyme disease in both humans and dogs. Initially after the treatment it appeared that the infection had been cleared. Attempts to isolate *B. burgdorferi* from skin biopsies were negative and antibody levels dropped. After an additional six months of isolation, however, the dogs' antibody levels increased again and the agent could be detected by PCR and culture in some dogs. These findings therefore suggest that recurrent disease and lameness in dogs could result from persistent infection, even after antibiotic treatment. We now have initiated another experiment to test the capacity of two other antibiotics, ceftrioxane and azithromycin, to eliminate *B. burgdorferi* infection in dogs. Intravenous application of ceftrioxane is the treatment most commonly used in humans with chronic Lyme borreliosis. Because Lyme disease in dogs is very similar to Lyme disease in humans, we expect the information we gather from treating dogs to be pertinent for humans as well.

—Max J. G. Appel

Albert C. Bostwick Laboratory of Molecular Biology

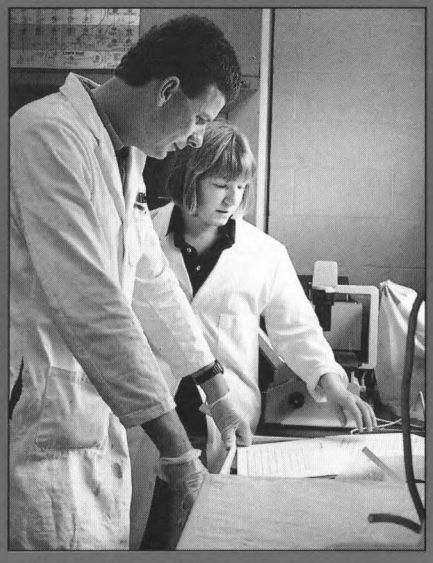
In the past year we have been extending our studies of the host ranges of canine parvovirus and feline panleukopenia virus, two widespread parvoviruses that can cause severe and frequently fatal diseases when they infect susceptible animals. Canine parvovirus had never been observed prior to the late 1970s, when it emerged quite suddenly as the cause of a new disease in dogs. We have established that canine parvovirus arose as a variant of the feline virus or of some other very closely related virus infecting another species—perhaps raccoons or foxes.

Shaped like a 20-sided sphere, a parvovirus particle consists only of an outer protein coat and its contents, a short, single strand of DNA that directs the synthesis of four proteins. In earlier work we showed that feline panleukopenia virus can be made to resemble canine parvovirus very closely by the substitution of only two or three small sections of canine parvovirus DNA in the DNA sequence of the feline virus. This mutated virus is then capable of infecting canine cells in culture and can also cause disease in dogs. Our current studies are aimed at determining how that small number of changes can completely change the host range of the virus.

In order to be taken up into a cell and infect it, the virus particle, or virion, must bind to the cell surface, most likely to a specific cell receptor. Once "docked", the virion is enveloped within a vesicle, a sort of pocket that forms on the cell's surface. The surface membrane surrounds the virion entirely, pulling it inside the cell, and then floats free of the rest of the cell membrane. Thus encapsulated, the virus particle must next escape from that vesicle in order to deposit itself or its genetic contents—its DNA genome—into the cytoplasm of the cell. From the cytoplasm the intact virus particle or its DNA enters the cell's nucleus, where it replicates.

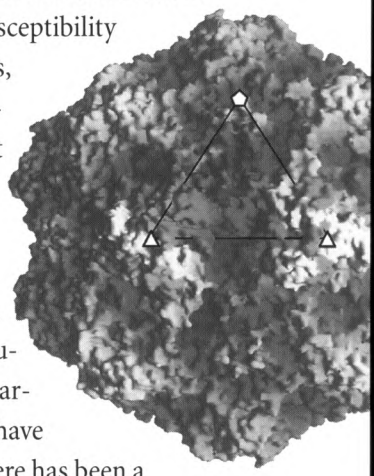
To understand how host range is controlled, we are now defining the process of cell infection, determining what changes occur in the virus that allow it to penetrate the cellular membrane, and also examining how the DNA is released from the virion. We are also comparing the infectious processes of canine parvovirus, which can infect dog cells, with the feline counterpart, which cannot. As part of this study, we seek to identify the molecules on the surface of the cell that bind the different viruses, to determine the types of vesicles the viruses are taken up into, and then to follow the structural changes in the virus that allow the virus to enter the cell and release its DNA.

We are now defining the process of cell infection: What changes occur in the virus that allow it to penetrate the cellular membrane?



Colin Parrish and Wendy Weichert

In addition, we are also defining the genes in the cells of the canine or feline host that control the process of cell infection. To accomplish this we will transfer genes from cat cells to dog cells in tissue culture, thereby determining whether certain of the genes can also transfer feline virus susceptibility to the dog cells, which are normally resistant to that virus.



In other studies examining the DNA sequence evolution of these parvoviruses, we have shown that there has been a continuing evolution and adaptation of canine parvovirus since it emerged as a canine pathogen in 1978. Our previous work identified two antigenic variants of canine parvovirus, designated types 2a and 2b, that spread around the world beginning in 1979 and 1984, respectively. In the past year we have identified a third genetic variant of canine parvovirus that is commonly found in Taiwan, Japan, and Europe. We have not yet detected this newest virus strain in the U.S., but expect that it will turn up here within the next few years. We do not know the biological significance of the sequence changes observed, but the widespread nature of the variant virus suggests that, like each of its predecessors in turn, it has a selective advantage over the previously existing strains of canine parvovirus.

—Colin R. Parrish

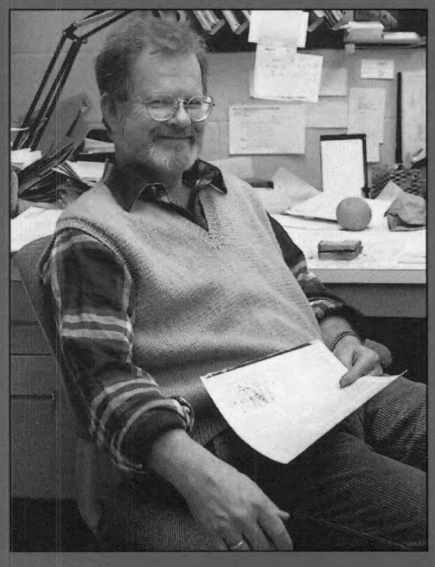
Immunology Laboratory

In this year's report I would like to return to a subject we discussed in the 1993 annual report and update you on our progress since then. This work has focused on the biological role of an immunoglobulin, known as IgE, that has enigmatic and perplexing biological properties.

In humans, dogs, and cats, IgE is recognized as the principal cause of allergies. IgE can react with allergens such as pollen, dander, mite or insect proteins, or drugs. Usually, allergen-reactive IgE adheres to specialized receptors on the surface of mast cells. When an appropriate pollen or dander allergen binds to the specific IgE the mast cells are triggered to release cytoplasmic granules containing histamine and other substances. This degranulation leads to the symptoms of asthma, eczema, rhinitis, or even anaphylaxis. Despite years of investigation and highly detailed knowledge of the physiology, biochemistry, and pathology of mast cells, we don't know what these cells, or the allergen-reactive IgE that triggers their degranulation, do that is useful. It is an established tenet of biology that there must be a reason why evolution would favor those individuals capable of exhibiting a sophisticated physiological response like mast cell degranulation. Generally speaking, biologists have also argued that a better understanding of how biological systems function will provide insights that will help us control their activity more effectively. For IgE and mast cells, however, our insights have faltered at the most important step of knowing how and why these reactive ingredients are present.

For several decades immunoparasitologists have recognized that some parasites—particularly nematodes and trematodes ("worms") have the ability to induce high IgE responses and substantial elevations of mast cell numbers. This response pattern has been recently classified as "stereotypical," as it almost always occurs during infection with these organisms. Despite the suggestion that the response pattern must be beneficial to the infected animal that mounts it, direct evidence has been hard to find. A few years ago a student in this laboratory, Ali Ahmad, did produce direct evidence for a role for IgE by using highly purified IgE from immune rats to transfer to non-immune rats the capacity to reject larval *Trichinella spiralis*. This began a series of experiments in which we demonstrated that, during the course of a *T. spiralis* infection, a specific mechanism develops in the intestine that transports substantial amounts of IgE to the intestinal lumen.

We are developing* in vitro *systems in which both the transport process and the potential protective effects of IgE can be examined.



Robin Bell

Over the last several years this work has been followed up by another student, Deborah Negrao-Correa, who has quantitated natural IgE transport in the intestine and in the systemic circulation during infection with *T. spiralis*. These were quite difficult experiments to conduct, involving careful and tedious quantitation of small amounts of rapidly degraded IgE in the intestinal lumen. Dr. Negrao-Correa showed that 100-500 times more of the IgE produced during a *T. spiralis* infection enters the intestine than the bloodstream. Once the IgE enters the intestinal lumen, where we measured it, it is rapidly broken down. This leads us to speculate that the real function of IgE is defined somewhere in the cellular interface between the lamina propria of the intestinal wall, where IgE is produced by B cells, and the epithelial lining of the intestine, from which the IgE is released into the lumen.

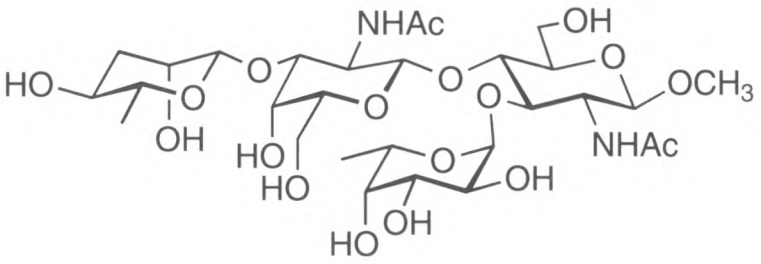
Current work is focused on developing *in vitro* systems in which both the transport process and the potential protective effects of IgE can be examined in detail.

—Robin G. Bell

Mucosal Immunity Laboratory

The ideal arrangement between parasite and host is one that allows for the survival of both, although the parasite sometimes thrives at some cost to its host. Nematodes, parasitic worms that colonize the intestine, are causes of morbidity in many species and of economic loss in food animals around the world. Our lab seeks to understand the ways by which one nematode, *Trichinella spiralis,* survives in the intestine of its mammalian host. Despite its relatively large size of 30 micrometers by one millimeter, the larval stage of *T. spiralis* resides inside a series of intestinal epithelial cells that are only 10 by 30 micrometers. The reason for selection of this rather snug habitat is not clear; however, when the immune system interferes with the parasite in this habitat the worm is expelled from the intestine. We are studying the process of intestinal invasion by the parasite and the host's immune defense of the intestine. This defense is mediated by antibodies that we have shown to bind to unusual sugars synthesized by *T. spiralis.*

Postdoctoral associate Catherine McVay has been studying the chemical structure of the sugars that are bound by protective antibodies. Since the native sugars are difficult to isolate in quantities sufficient for study, she tested the antibodies for binding to synthetic sugars that mimicked those found in *T. spiralis.* Synthetic sugars were prepared in the laboratory of our collaborator, Dr. David Bundle at the University of Alberta. Dr. McVay found that the antibodies bound to the sugar tyvelose, but only when it was prepared in the beta anomeric conformation, telling us that this is the structure that is synthesized by the parasite. This chemical detail is unique to *T. spiralis.*



Molecular structure of the glycan found in *Trichinella spiralis*

During this year, Lucy Gagliardo and I developed an assay that allows us to study epithelial cell invasion by *T. spiralis* in cell cultures. We found that if we inoculated epithelial cell lines with infectious larvae, the larvae would invade the cells and migrate through them in a manner similar to that described for worms in the intestine. This was highly significant because it was the first time that anyone had observed *T. spiralis* invade host cells. Furthermore, Ms. Gagliardo showed that the parasite deposits glycans in the cells that it invades, a result that suggests that the molecules may play a role in invasion of or migration through the cells. Postdoctoral associate Trenna ManWarren developed methods for quantifying the damage caused to the cells by the worm. She showed that epithelial cells from a variety of animals are susceptible to invasion, a result that corresponds with the host range of the parasite in nature. This technique has proven to be a powerful tool for the study of the nematode's behavior during invasion and is currently being applied to the study of antibody-mediated expulsion of the parasite from epithelial cells.

The* T. spiralis *parasite deposits glycans in the cells it invades, a result that suggests the molecules may play a role in invasion of or migration through the cells.

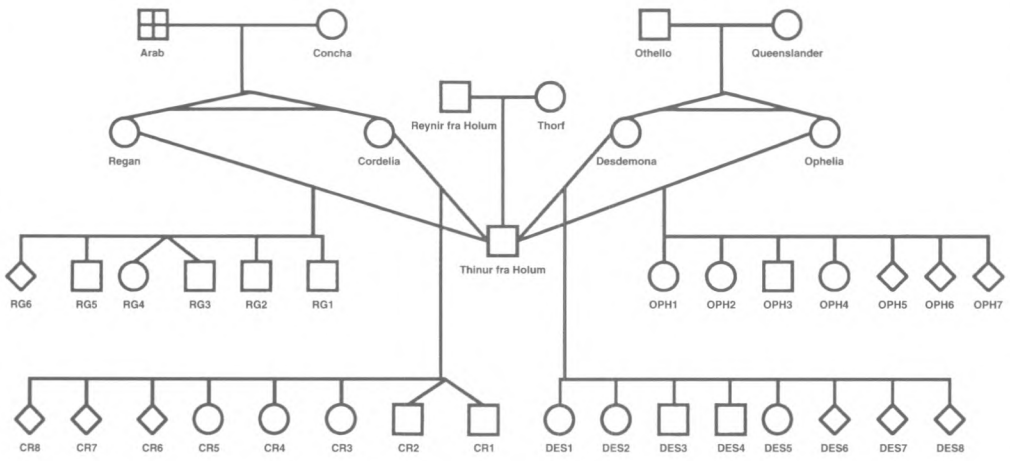
Phil Peters and Trenna ManWarren

—Judith A. Appleton

Equine Genetics Center

The past year brought a new project to the Equine Genetics Center: participation in a worldwide initiative to map the genome of the horse. This cooperative venture kicked off with an international workshop held in Lexington, Kentucky in October of 1995. The workshop brought together representatives from over 30 laboratories in 15 countries, including the U.S., Japan, Australia, New Zealand, England, Sweden, Germany, France, Switzerland, Poland, and Holland. Participants in the meeting agreed to share information and reagents and developed a five-year plan for collaborative research efforts. The goal is to construct a first-level map of the horse genome and to bring knowledge of horse genetics up to the level of knowledge of better-studied species, such as cattle and pigs. The advances in gene mapping technology made in the Human Genome Project make the goals of the equine geneticists attainable.

The annual international Horse Gene Mapping Workshops and other aspects of the Horse Genome Project are being supported in part by the Dorothy Russell Havemeyer Foundation. The Foundation is playing an important role as a catalyst to promote cooperation among the participating laboratories.



Unique pedigree of full siblings produced for linkage mapping in the Horse Genome Project by Prof. W. R. Allen of Cambridge University

Our goal is to construct a first-level map of the horse genome and to bring knowledge of horse genetics up to that of better-studied species.

Jessica Baker

Here at Cornell the Zweig Memorial Fund for Equine Research has funded the efforts of the Equine Genetics Center to identify and characterize new genetic markers, called microsatellites, in the horse genome. By the end of 1996 over 125 new horse microsatellites had been identified and partially characterized. This project involves collaboration with Dr. Chip Aquadro's laboratory in Cornell's Division of Biological Sciences. It has already attracted an enthusiastic group of undergraduate and veterinary students who are participating in this research.

During the past decade, genetic studies at the Baker Institute have focused on diseases involving the immune system, in particular a skin tumor of horses known as equine sarcoid. Sarcoid is caused by the infection of horses with bovine papilloma virus. In collaboration with veterinary scientists at the School of Veterinary Medicine of the University of Berne in Switzerland, we have found that susceptibility to sarcoid is strongly influenced by genes of the major histocompatibility complex (MHC). Although the mechanism for this susceptibility is not yet known, it is likely to be a common and important one. In two other species, humans and rabbits, the development of tumors after infection by papilloma viruses is also influenced by MHC genes. Horse breeding can already be aided by the application of new genetic knowledge, and it is very likely that this progress will accelerate as the Horse Genome Project continues.

REPRODUCTIVE IMMUNOLOGY OF THE MARE

In continuing studies of reproductive immunology, Equine Genetics Center scientists are working to decipher the complex interactions between mother and fetus that occur during pregnancy. In particular, our studies focus on the early stages of maternal recognition of the developing fetus in the mare. This crucial time sets the stage for the growth and development of the fetus throughout gestation.

During the past two decades we have learned much about how the immune system of the pregnant mare identifies the developing fetus as foreign tissue. In mares, specialized cells of the placenta invade the uterus and stimulate a strong maternal antibody response. Such responses are much stronger in the horse than in any other species that has been studied. These responses may be the cause of some cases of early pregnancy loss, but the evidence for this is not yet definite.

ALEXIS WENSKI-ROBERTS

Stud manager Jim Hardy and Bear, senior Thoroughbred stallion, at the McConville Barn

The strong immune responses mounted against the fetus appear superficially like the destructive immune reactions that destroy most tissue and organ grafts. Part of the remaining mystery of this aspect of reproduction is how the fetus escapes from the potentially lethal maternal immune attack. Although these studies have focused on the particular characteristics of the equine placenta, many of the mechanisms that operate are likely to be conserved among species as diverse as humans, rodents, and dogs.

Over the past two summers two veterinary students have conducted important projects as part of the Institute's reproductive immunology program. Both students were supported by the Havemeyer Foundation Summer Fellows Program. In 1995 Mark Stidworthy of Cambridge University extended our studies of horse pregnancy to interspecies hinny pregnancy. A hinny, the outcome of a cross between a stallion and a jenny donkey, is one of the rarest of the equine hybrids. In 1996 Tamara Gull from Tufts University studied maternal anti-fetal immune responses in the opposite cross, that is, in mule pregnancy established by breeding a jack donkey with a mare. The two students determined the time course of the maternal antibody responses mounted against hybrid fetuses, and they also investigated the specificity of those responses. They made the unexpected discovery that the antibodies were specific for major histocompatibility complex antigens of the mating stallions or jack donkeys, and not directed against antigens common to all horses or donkeys. These results have led us to undertake new genetic studies of the major histocompatibility complex that probe the evolutionary history between members of the horse family.

The horses, donkeys, and mules and hinnies of the Equine Genetics Center have been invaluable in these investigations. Bear, the senior stallion, has been in the herd since his birth in 1980, and several other horses have equally long tenures. The Baker Institute's equine collection is unique in the world, and it represents a real treasure and resource for scientific studies of man's noblest companion.

—Douglas F. Antczak

A Retrospective

THIS YEAR I submit my concluding report, *mon chant du cygne*, as I plan to formally retire during the coming fall to an unstructured life as befits the venerable—Ovaltine and lawn bowling. I have enjoyed an exceptionally exuberant and satisfying career at the Baker Institute and shall always be indebted to my colleagues, past and present, who have provided the splendid environment in which I've been granted the privilege to pursue, with few constraints, research on diseases that affect dogs.

It's been especially rewarding to have been associated with exceptional graduate students and to have experienced the excitement attending the isolation of novel pathogens and the development of methods for the prevention and treatment of several diseases of dogs, with a couple of diversionary whacks at maladies of cattle and sheep.

Opposite: **Dr. Carmichael in 1975**

Right: **The newly designated John M. Olin Professor of Virology, 1964**

Giralda Laboratory for Canine Infectious Diseases

At retirement, I will have been a member of the faculty in the Baker Institute's Cornell Research Laboratory for Diseases of Dogs for 41 years. During that time, I have witnessed the scientific and physical growth of the Institute. Initially, there were three full-time faculty, James ("Drew") Baker, Jim Gillespie, and Ben Sheffy; five graduate students; and a visiting investigator, Dr. Alan Betts, on leave from Cambridge University. Alan later became Dean of the Royal Veterinary College of the University of London. The Institute staff of about 20 included Dr. Baker's wife, Dudley, who edited virtually all of the papers and reports that issued from the Institute until the 1970s. The faculty has now quadrupled, as has the number of graduate students, post-doctoral research associates, and support staff, who, throughout the history of the Institute, have been extraordinary in their commitment to investigators' needs.

The original facilities were sparse, but sufficient for the work at the time. One of the developments that attracted me to Dr. Baker's Veterinary Virus Research Institute in the fall of 1956 was the recent adoption of reliable and practical tissue culture methods. The Institute was among the first to incorporate those methods into the study of animal viruses. The facilities included isolation units modeled after those at the Rockefeller Institute in Princeton, New Jersey. Such facilities, unavailable elsewhere, placed the Institute at the forefront of infectious disease research.

My interest in infectious diseases was first aroused by Dr. Karl Meyer, whose lectures on zoonotic diseases and their modes of transmission inspired several of his students to pursue that discipline. After I had been at Cornell for a few months, I was fortunate to attend one of Dr. Baker's evening seminars and learn of the existence of the laboratory on Snyder Hill where the focus was infectious disease research. After petitioning the director to accept me as a graduate student, I was accommodated with some indifference and told to "get to work and prove your worth." I spent the first year transferring *Leptospira pomona* in guinea pigs, teaching Dr. Baker's course in virology to veterinary students, and assisting in the preparation of tissue cultures for Institute researchers.

One of the developments that attracted me to Dr. Baker's Veterinary Virus Research Institute in the fall of 1956 was the recent adoption of reliable and practical tissue culture methods.

At that time, it was often difficult to find time to conduct research because of the constant flow of visitors who came from throughout the U.S. and abroad to observe our methods and to take advantage of the infectious agents that were isolated here; in several instances, the strains had been attenuated and rendered suitable for vaccines. At that time they were distributed freely, without patent or cost.

I learned a new way of life in academe from Douglas McGregor, who assumed the directorship in 1975 after the death of Dr. Baker—the necessity of obtaining external research support. Doug McGregor maintained that "good science" was done only with external support, preferably by peer review—the way virtually all research is now conducted. Douglas launched the Institute into the modern era of molecular biology and immunology and amplified the Institute's financial basis. His example taught the merit of selective parsimony.

Doug Antczak capsulized the essence of the Institute's role in veterinary research in last year's annual report: "...the Institute holds a special place in veterinary medicine by virtue of its distin-

PHOTO SCIENCE / CU

Skip Carmichael with Drew Baker and Hadley Stephenson in the early 1960s

PHOTO SCIENCE / CU

Dr. Carmichael, *center*, with Dr. Stephen Bistner and Dr. Lenora Sammons in 1972. Dr. Bistner was examining a beagle for the presence of "blue eye," a reaction that sometimes followed vaccination with canine adenovirus-1.

guished history of practical accomplishments," and, I might add, the successes of its graduates. I take great satisfaction in the accomplishments of all the graduate students, but particularly those of two who later became members of the Institute faculty—Professors Max Appel and Colin Parrish. Both Max and Colin have attained prominence in both the veterinary and scientific communities and have brought distinction to the Institute through their research. I wish to acknowledge the reflected eminence I have enjoyed from their achievements.

Canine parvovirus (CPV-2) was initially recognized by Max Appel at the Baker Institute in the summer of 1978. During the frenzied three years that followed, Roy Pollock, then a graduate student and now Vice-President of the Companion Animal Division at Pfizer Animal Health, fielded over 10,000 telephone calls from veterinarians and dog owners. Studies in our laboratory quickly led to a basic understanding of the pathogenesis of CPV-2 and the development of rapid and specific diagnostic tests. The development of safe and effective vaccines within three years of the initial discovery was particularly gratifying.

Colin Parrish's contributions to parvovirology have been immense. The knowledge that has emanated from his laboratory has provided an understanding of the true nature of CPV-2, its natural history, and continuing evolution. Because of his efforts, canine parvovirus and its close relatives are better understood than

RUSS HAMILTON

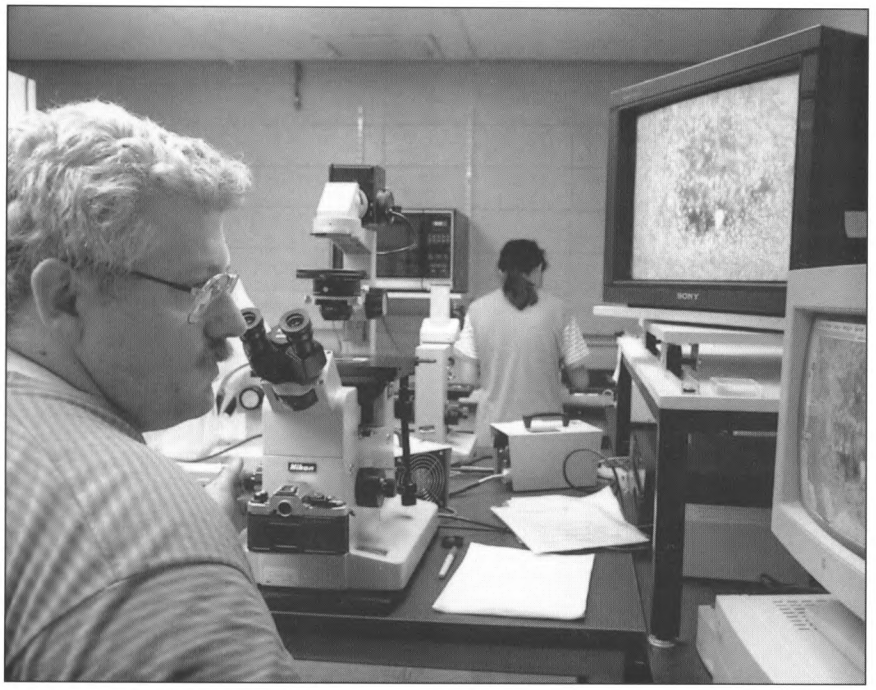
Top: **Dr. Carmichael during a 1991 visit to Africa as a consultant for the United Nation's Food and Agriculture Organization.** ***From left:*** **Dr. Joseph Musiime, Dr. Fikre Yosef, Dr. Daouda Sylla, and Dr. Carmichael**

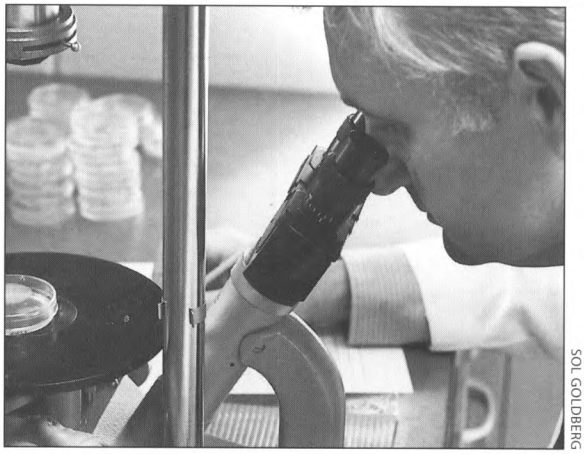
Bottom: **Dr. Carmichael with Jean Joubert, a longtime member of his laboratory staff, in 1978**

virtually any other canine virus. Colin's commitment to delving into complex areas of molecular biology and genetics substantiated the critical importance of embracing advances in technology. Sometimes the right individuals appear at the proper time—serendipity.

There are several other undertakings in which I have taken special pleasure:

- Together with Max Appel, the development and improvement of canine adenovirus vaccines. The substitution of canine adenovirus-2 vaccine eliminated the problem of "blue eye" and other adverse reactions engendered by canine adenovirus-1.
- The discovery of canine herpesvirus and explanation of its pathogenic role in neonatal puppy deaths and reproductive disease. Knowledge of the profound influence of temperature on the pathogenesis of canine herpesvirus was exploited by Geoff Letchworth, then a graduate student and now on the faculty of the University of Wisconsin, to explain the pathogenesis of bovine herpesvirus-2. In those studies, Geoff constructed thermoregulated bras for heifers in order to perform his experiments.
- The discovery of *Brucella canis* and its identification as the cause of abortions and reproductive disease in dogs. We assisted countless kennels and dog owners in controlling the disease on their premises. We have been frustrated, however, in our efforts to awaken dog breeders to the seriousness of the disease and the need to control it.
- The isolation and characterization of a mycoplasma organism, now called *Mycoplasma bovis*, from several herds of New York cattle with severe, untreatable mastitis. The cause was originally believed to be a virus. Mycoplasma mastitis caused by *M. bovis* remains a serious problem in several states.
- The investigation while on sabbatical leave in Brisbane, Australia, of a chronic, interstitial pneumonia of sheep in Queensland. This work was done with Drs. Toby St. George and Neil Sullivan of the Commonwealth Scientific Research Organisation (CSIRO). The causal agent, which we named *Mycoplasma ovipneumonia*, has now been identified in sheep with chronic pneumonia in several countries.
- The establishment, in 1965, of the Laboratoire du Vaccin in the Republic of Mali. That laboratory was expanded into a modern research complex a few





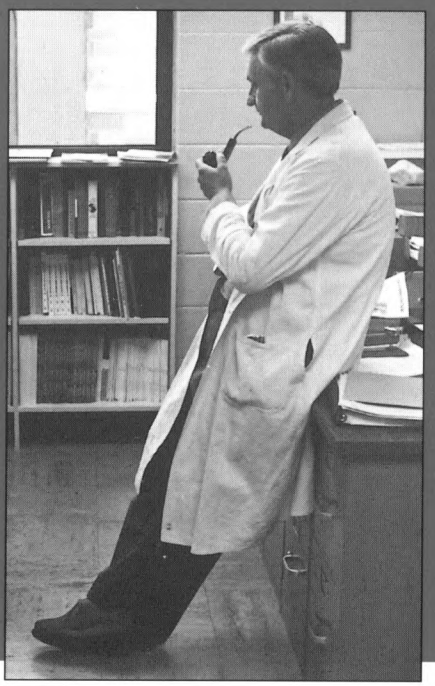
SOL GOLDBERG

Left: **Dave Peters, the last in a long line of Carmichael's graduate students, studies a microscopic sample using advanced video-imaging technology.**

miles down the Niger River from Bamako and renamed the Central Veterinary Laboratory in August 1977. The "Cornell Team" included Dr. Baker, Charles York, Ben Sheffy, Marvin Goff, and me, working closely with Laboratory director Daouda Sylla, who had received advanced training at Cornell. Over three million doses of good rinderpest vaccine were produced within the first year. Twenty-seven years later, the Laboratory is still producing good vaccines.

I'm tempted to chronicle my major flops, but those would then surely be remembered most. There is a plentiful supply of problems in canine infectious diseases and immunology that remain to be studied, but it would be presumptuous of me to suggest them. Investigators must find their own problems, ideally settling on those that are important, of interest, and fundable. Microbes never cease to try to outwit those who seek to control them.

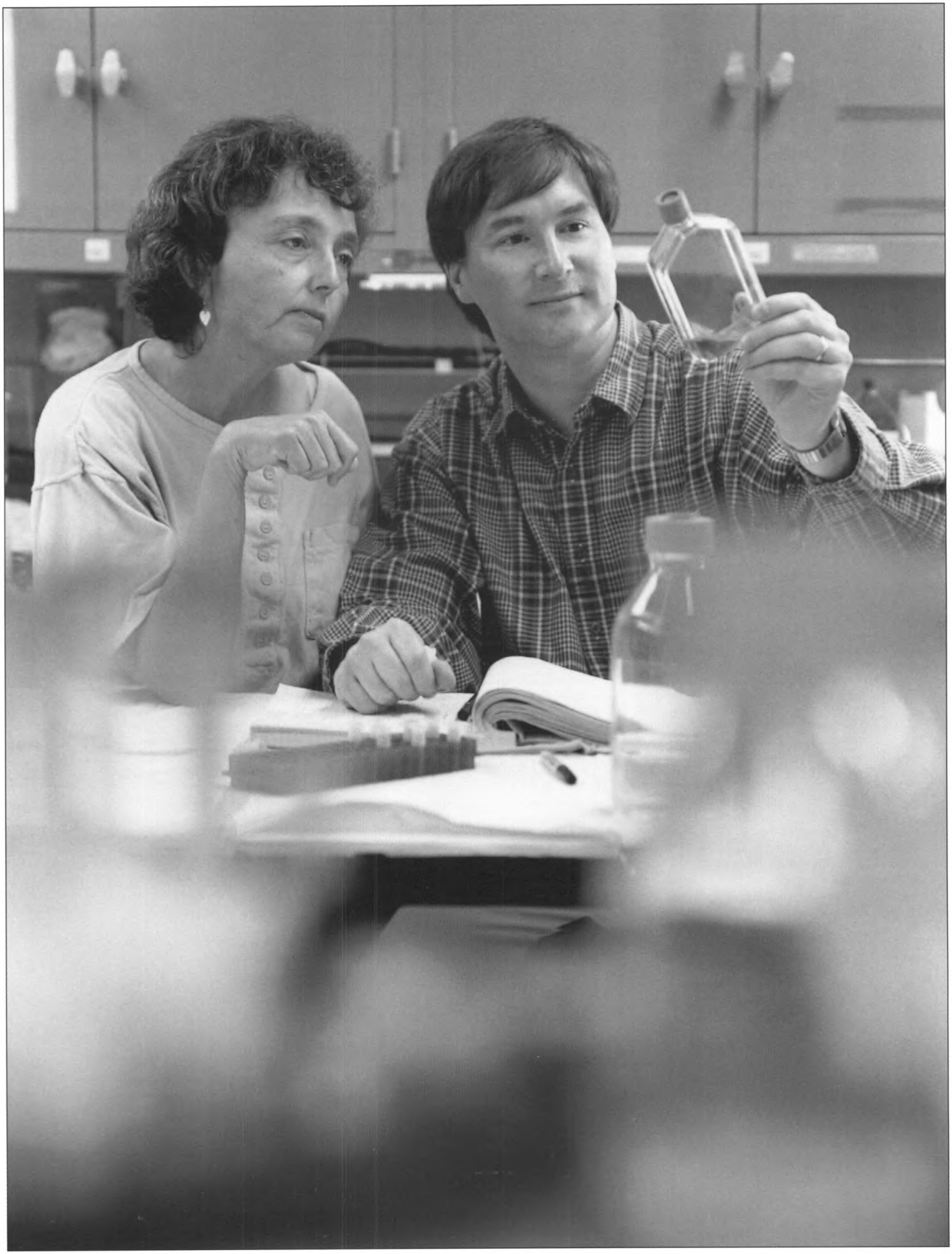
There is a plentiful supply of problems in canine infectious diseases and immunology remaining to be studied. Microbes never cease to try to outwit those who seek to control them.



Professor Scott Ellege, a distinguished professor of English at Cornell from 1962 to 1984, is quoted to have modestly stated on his retirement: *"It is time I stepped aside for a less experienced and less able man."* I look forward to being replaced by an individual who, though perhaps less experienced, will be better able to address contemporary problems through the tools now available to modern biomedical scientists.

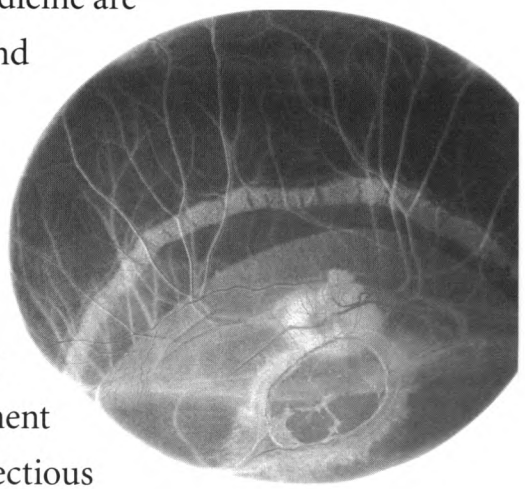
I close with a statement that Dr. Baker often recited to his colleagues and students: "Only the lead sled dog is able to enjoy the scenery." For those of us who've had the good fortune to work up here on Snyder Hill, the scenery has been marvelous.

—Leland E. Carmichael



Genetics and Development

DEVELOPMENT OF METHODS for the identification, characterization, and manipulation of genes is perhaps the most influential technical advance of the past quarter century in the biomedical sciences. Biology, agriculture, and human and veterinary medicine are being transformed by our new-found ability to decipher the code of life.



Nearly ten years ago the faculty of the Baker Institute made a decision to expand the size and scope of our research activities by adding programs in genetics and development to the long-standing projects in infectious diseases. The new programs encompass traditional genetic studies of diseases caused by single gene defects, such as the various forms of progressive retinal atrophy; more complicated forms of inheritance, such as that responsible for canine hip dysplasia; and studies of normal and abnormal development of tissues and organs. Included in this third category are studies of joint biology and osteoarthritis, defects in the development of the reproductive tract, and eye disease that is manifested primarily in later life.

In making such a commitment to the new Genetics and Development programs, the Baker Institute staff have reaffirmed the founding mission of the Institute to improve the health of animals through basic and applied research. As veterinary medicine and biomedical science continue to advance, we will continue to reinterpret our mission to meet the needs of future generations of animals and their owners.

Opposite: **Nancy Burton-Wurster and Jamie MacLeod**

Right: **A well-developed early placenta surrounds and protects this horse embryo at day 33 of gestation.**

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

Canine hip dysplasia has proven a challenging problem to researchers attempting to determine its causes and a reliable means to predict, and therefore prevent, its development. Multiple factors appear to influence the development and severity of hip dysplasia in dogs, among them diet, physical stresses, and an undefined combination of genetic abnormalities. Together with Dr. Rory Todhunter from the Department of Clinical Sciences and graduate student Michael Olivier, and with financial support from the Ralston-Purina Company, we have continued our studies to identify a marker in the DNA of dogs that can be associated with the inheritance of hip dysplasia. Such a finding is a necessary first step toward discovering the genes themselves that control the development of this important disease. Toward that end, we are currently developing a genetically informative pedigree by crossing dysplastic Labrador retrievers with greyhounds that are free of hip dysplasia. More information about this research will be described in future reports.

In the course of searching for a molecular genetic marker for hip dysplasia, Michael Olivier made an interesting, unrelated observation. He discovered a new marker in the DNA of male dogs that is not present in female DNA. He made this observation while scanning canine DNA for random polymorphisms. DNA was obtained from 107 dogs from seven breeds and the new test correctly identified all 51 male dogs in the group.

Currently Mr. Olivier is attempting to localize the male-specific DNA sequence to the male sex chromosome Y. He has already found a one-to-one correlation between the new marker and a gene on the Y chromosome that is known to play a role in male sex determination, Sry.

We are currently developing a genetically informative pedigree by crossing dysplastic Labrador retrievers with greyhounds that are free of hip dysplasia.

George Lust and Nancy Burton-Wurster

Other preliminary data suggested that the nucleotide sequence identified in male dogs also is present in the DNA of men and male rodents.

INTERNATIONAL HIP DYSPLASIA MEETING

The Baker Institute inaugurated an annual conference series in August 1996 with an international symposium on hip dysplasia and osteoarthritis in dogs. The conference was the first in more than two decades to be dedicated to this subject. More than 50 scientists gathered to share the latest research on the diagnosis, pathogenesis, genetics, and treatment of hip dysplasia and osteoarthritis.

The major recommendations presented were to breed only dogs with the best, normal hip joint conformation and the lowest degree of hip laxity, and to restrict the growth rate of young dogs by feeding smaller amounts of food. Recent studies have established that if dogs are fed 25 percent less food than they normally would eat during their rapid growth phase in the first year, the appearance of hip dysplasia and hip osteoarthritis can be lowered by 65 percent.

—George Lust

Healthy cartilage is a smooth, resilient tissue that resists compression and minimizes friction from joint movement. Our goal is to understand the biochemical and mechanical changes that occur in cartilage very early in the development of osteoarthritis, a progressive disease that invariably accompanies hip dysplasia in dogs, in order to intervene early and stem the degenerative process.

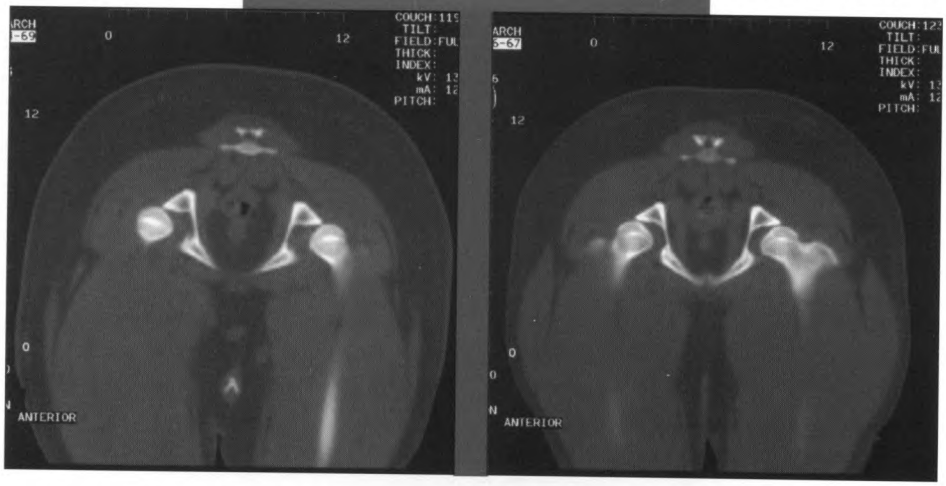
Sorting out the complex genetics of hip dysplasia is made more difficult by the effects of environmental influences on the expression of the disease. In the absence of a DNA-based screening method, it is difficult to identify those puppies that will develop hip dysplasia. To address this problem, we have been working with colleagues in the Departments of Clinical Sciences, Anatomy, and Mechanical and Aerospace Engineering to develop a non-invasive imaging technique involving quantitative computerized tomography. This technique will allow us to correlate changes that occur over time in joint geometry and the distribution of mechanical loads with the onset of early cartilage lesions while a dog is still without symptoms and appears normal by standard radiographs.

One aspect of our studies of cartilage biology that has proved very exciting for us involves the adhesive glycoprotein fibronectin. Fibronectin is an important protein present in a variety of tissues in the body, and we have known for several years that fibronectins from different cell types vary in structure at three different regions of the molecule. Recently, however, we and our colleagues in Jamie MacLeod's laboratory discovered yet a fourth variant region of the fibronectin gene—one that is uniquely expressed in cartilage. In cartilage, 50 to 80 percent of the fibronectins lack both this newly found region (which we have designated as "C", for "cartilage") as well as one of those previously identified (the "V" region). We postulate that fibronectin with this unique structure may have some crucial role in cartilage function.

We have been developing a non-invasive imaging technique involving quantitative computerized tomography to correlate changes that occur over time in joint geometry.

We have continued to learn more about this cartilage-specific fibronectin and have answered the question of how it is secreted. Most fibronectins are formed and secreted as heterodimers, meaning that their molecules are compounds of two unlike, smaller molecules. The cartilage-specific isoform, however, does not like to form hetero- (mixed) dimers. Instead, it dimerizes only with a subunit like itself or remains a simple molecule, also unusual for fibronectins. In the cartilage-specific fibronectin, there is no V or C region at all, and whatever the functions of these regions, they cannot be compensated for as they might be in a heterodimer. The knowledge that these regions have been implicated in matrix assembly and in expression of enzymes that degrade the matrix may offer a clue to the importance of the cartilage-specific fibronectin to cartilage biology.

—Nancy Burton-Wurster



These quantitative CAT scans illustrate the dynamics of hip joint laxity as it might appear in a dog as it walks. The anesthetized dog is placed in a "kneeling" position, with its weight on the stifle joints to simulate the natural forces acting on the ambulating hip joint. The use of this position for diagnostic purposes is being explored by colleagues in the Department of Clinical Sciences of the College of Veterinary Medicine.

The dog on the left, diagnosed previously as having moderate laxity, did not appear dysplastic on standard radiographs. Note how the rim of the acetabulum, or joint socket, is pressing on the fovea, a depression at the top of the femoral head where the round ligament attaches to the femur. The fovea is the classic site for the development of joint lesions in osteoarthritis. In contrast, the dog on the right does not show the dramatic subluxation present in her littermate.

Laboratory of Cellular Growth and Differentiation

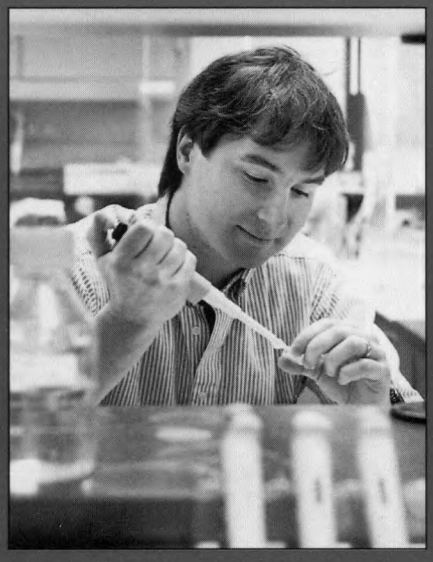
Diseases involving joint cartilage, such as osteoarthritis and osteochondritis, are common in dogs and horses. They are often progressive and potentially debilitating. Research questions in our laboratory focus on the molecular mechanisms of these diseases and how those mechanisms relate to basic functional properties of cartilage cells.

Cartilage is a unique tissue, both structurally and functionally. Most other tissues share some common cell types, like those that make up blood vessels, nerves, or fibrous tissue. In contrast, cartilage contains only a single cell type, called chondrocytes, embedded in an extracellular matrix. The chondrocytes manufacture and maintain this matrix, which provides cartilage with its specialized biomechanical and functional properties. Having no internal blood vessels or nerves, injured cartilage does not bleed, and any pain that is felt originates from nerve endings in the surrounding bone or joint capsule.

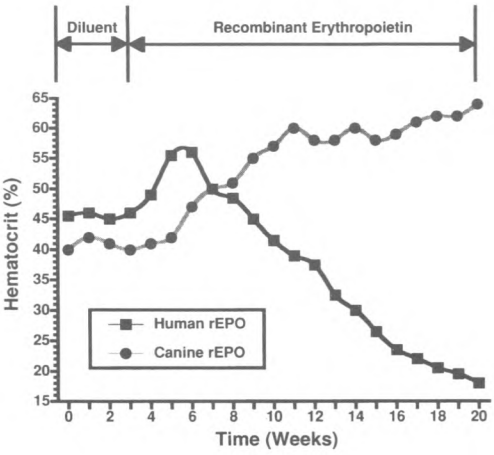
The biomechanical properties of cartilage matrix are central to the tissue's normal function, so it is important to understand how matrix components change in diseases and in response to different therapies. In collaboration with Nancy Burton-Wurster and others in her laboratory, we have discovered that cartilage contains a unique variant of a protein called fibronectin. Although cartilage fibronectin levels are high and increase further during the development of osteoarthritis, scientists who study cartilage biology have tended to focus their attention on other major matrix components, such as type II collagen, proteoglycans, and metalloproteinase enzymes. In other areas of biomedical research, however, fibronectin has generated great interest.

Physical contact with the surrounding environment is one of the most important regulators of coordinated cellular function in any tissue. The loss of cell regulation results rapidly in abnormal function and the development of disease. Fibronectin has been shown to be one of the most important molecules through which cells interact with their surrounding environment. Our discovery of a new fibronectin variant is especially exciting because it appears to be present at high levels only in cartilage tissue, unlike other forms of fibronectin, which have a wide tissue distribution throughout the body. This distinction strongly suggests that the newly found fibronectin variant has an important role in cartilage function.

Our discovery of a new fibronectin variant is especially exciting because, unlike other forms of fibronectin, it appears to be present at high levels only in cartilage tissue.



Jamie MacLeod



One of the projects we are pursuing is not related to cartilage or chondrocytes, but to the ability to synthesize red blood cells. In certain diseases, particularly those involving renal failure, anemia develops due to a deficiency of a hormone called erythropoietin (EPO). During the past decade humans with this form of anemia have been successfully treated with human EPO prepared using recombinant DNA technology. Dogs treated with this product show initial improvement, but the treatment ultimately fails when the immune system recognizes human EPO as a foreign protein and develops antibodies. We have now completed the preparation of canine-specific erythropoietin to address this problem. As illustrated above, canine EPO works well in dogs without the immune-mediated problems associated with human EPO. This therapy is now available to privately owned dogs through a clinical trial I am conducting in collaboration with Dr. John Randolph in Cornell's Companion Animal Hospital.

—James N. MacLeod

Laboratory for the Study of Inherited Canine Reproductive Diseases

In August 1996 I began a sabbatical leave, the first half of which was spent in the laboratory of Peter Goodfellow in the Department of Genetics at the University of Cambridge in England. Dr. Goodfellow is a world-recognized expert in the genetics of development of the human reproductive system. In his laboratory I continued my studies of canine XX sex reversal, an autosomally inherited disorder that causes testicular tissue to develop in individuals that are chromosomally female. This problem, which can also occur in humans and domestic animals, is already a well-documented cause of infertility and sterility in at least seven breeds of dog—the American and English cocker spaniel, German shorthaired pointer, weimaraner, beagle, Kerry blue terrier, and pug. In addition, the problem has been diagnosed in the West Highland white terrier, basset hound, Doberman pinscher, Pomeranian, vizsla, soft-coated wheaten terrier, and Walker hound. The condition may occur in other breeds as well.

Our studies have focused on the inheritance of XX sex reversal in the American cocker spaniel and the German shorthaired pointer, two breeds with a relatively high prevalence of the disorder. Although the problem may be caused by the same gene mutation in breeds that are closely related, such as the American and English cocker spaniels or the German shorthaired pointer and the weimaraner, the disorder may also result from different mutations in more distantly related breeds.

We are examining the genes that normally control testicular development to understand how testis tissue can develop in animals with female sex chromosomes. The goal of this research is to identify the gene that causes the problem and determine how the mutation induces testis development during embryonic development. It is likely that this mutation resides in an as-yet undiscovered gene in the testis differentiation pathway. Thus these studies should provide new information about the genetic control of normal testis differentiation. Once the gene is identified it will be possible to design a practical DNA test to detect carriers and affected dogs.

We are examining the genes that normally control testicular development to understand how testis tissue can develop in animals with female sex chromosomes.

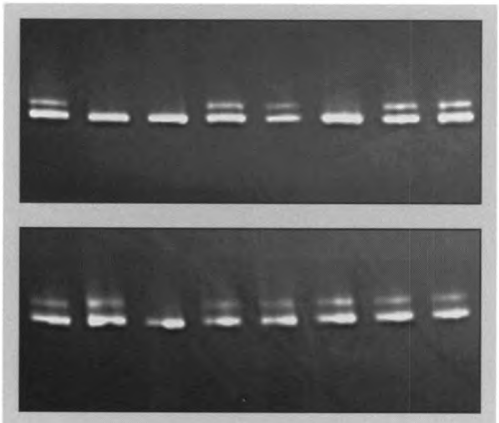
We have examined DNA from affected American cocker spaniels and German shorthaired pointers for the presence of Sry, a gene that is responsible for initiating testis development in males of several species. This gene is normally located on the Y chromosome, but might theoretically be passed from father to genetic daughter as a result of translocation. This phenomenon occurs when the part of the Y chromosome containing the Sry gene breaks off during meiosis and attaches to a different chromosome. XX offspring that inherit this "attachment" chromosome therefore inherit the translocated Sry gene, and the ability to develop testes, without having a Y chromosome. Translocation of the Sry gene has been shown to occur in other species, but has not been demonstrated in dogs.

—Vicki N. Meyers-Wallen

Vicki Meyers-Wallen

Donnelley Laboratory of Gene Regulation and Expression

Research in our laboratory focuses primarily on a group of inherited diseases of dogs, cats, humans, and other mammals known as mucopolysaccharidosis, or MPS. This unwieldy name describes a serious and in some forms fatal accumulation of mucopolysaccharides in the cells of many tissues throughout the body, including bones, joints, eyes, skin, the liver, the spleen, and the cardiovascular, respiratory, and central nervous systems.



Representative pedigree analysis demonstrating co-inheritance of the GUSB mutation with the MPS VII disease locus.

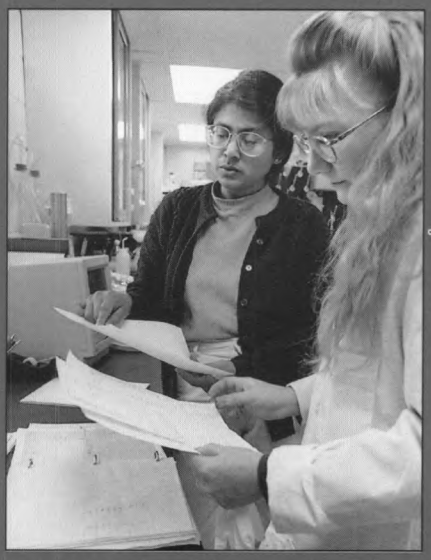
A DSCP (double-stranded conformational polymorphism) analysis of PCR products obtained from genomic DNA. The heterozygous carrier animals show a heteroduplex formed from one normal band and one affected band, while homozygous normal and affected animals show only one or the other of the two bands.

B DSCP analysis of PCR products after mixing with known normal sample from genomic DNA. This step causes the formation of a heteroduplex for affected dogs as well as for carriers, thus distinguishing affected from normal dogs.

In one form of the disease that we are currently studying, MPS VII, this accumulation results from a deficiency of beta-glucuronidase (GUSB), an enzyme with the essential function of breaking down mucopolysaccharides, a by-product of cell membranes and extracellular matrices. In individuals who lack sufficient levels of this enzyme, mucopolysaccharides build up in the lysosomes, tiny compartments that normally aid in digestion within many types of cells. The build-up of mucopolysaccharides in MPS VII leads to deformity and death, first on the level of the cell, and finally for the body as a whole.

This year we completed the work begun last year to determine the nucleic acid sequence of the GUSB gene in dogs with MPS VII. In our subsequent work to characterize the GUSB gene, we have detected a single nucleotide change in its coding sequence that renders it non-functional in the diseased dog. The discovery of this mutation has allowed us to develop a blood test capable of identifying carriers and affected dogs, as shown in the figure at far left. This test can now be routinely used to screen dogs for the GUSB mutation, providing breeders with MPS VII in their lines with the information they need to determine future breeding strategies.

Our experimental attempts to treat diseased canine retinal pigment epithelial cells in culture have yielded extremely encouraging results.



Jharna Ray and Maria Verdugo

We have recently begun cloning the feline GUSB gene. This work will enable us to characterize the molecular defect in cats, again with the aim of developing a DNA-based diagnostic test for use in preventing the inheritance of the disease.

In collaboration with Dr. Mark Haskins of the University of Pennsylvania, Dr. Philippe Moullier of the Pasteur Institute, and Baker Institute colleague Gustavo Aguirre, my laboratory is continuing research to develop a gene therapy method to treat MPS VII. Our experimental attempts to treat diseased canine retinal pigment epithelial cells in culture have yielded extremely encouraging results. We will soon begin experimental gene therapy in dogs suffering from blindness as a result of MPS VII.

AGING STUDIES

Macular degeneration is the leading cause of blindness in primates. Unlike the MPS group of diseases, which develop in early life, the changes observed in macular degeneration stem from aging. With grant support from the American Federation for Aging Research, Maria Verdugo and Virginia Scarpino have shown that several lysosomal enzyme activities increase in aging humans, and that this increase is due to alterations in gene regulation. We have begun looking for genes that are critically susceptible to aging and disease.

—Jharna Ray

Inherited Eye Disease Studies Unit

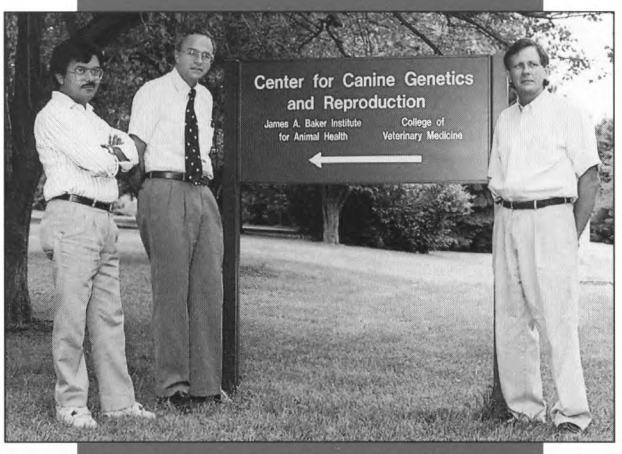
Progressive retinal atrophy (PRA) in dogs is not one disease, but seven separately inherited forms that are prevalent in different breeds. In the two forms of PRA affecting collies and Irish setters, disease is characterized by an early disruption in the development of the retinal cells, a condition known as rod-cone dysplasia. This type of PRA results in blindness by the age of one year. In other forms, blindness results from a later and sometimes gradual degeneration of the photoreceptor cells, beginning with the rods and progressing to the cones. Since the rods are the cells responsible for vision in dim illumination, the earliest sign of retinal degeneration is night blindness.

The retinal dysplasias found in collies and in Irish setters are unlike any other forms of PRA in that each has a biochemically definable abnormality. These abnormalities involve an inactivity of the enzyme cyclic GMP phosphodiesterase (PDE). Several years ago we announced the development of a mutation-based diagnostic test for the Irish setter disease, which we had found to be caused by an inactivity of the beta subunit of the PDE gene. Since the disease of collies is clinically identical but genetically distinct from the disease of Irish setters, we have concentrated our search for its cause on the other subunits of PDE—alpha and gamma—and the proteins involved in its activation. With support from the Collie Club of America Foundation, Weiquan Wang and others in our laboratory have cloned the alpha and gamma subunits of the PDE gene as well as the alpha subunit of the transducin gene, which is also involved in phototransduction. In the collie, we have excluded transducin-α, and we have found no sequence abnormality for any of the three subunits of PDE. This may mean that the mutation is elsewhere in other genes involved directly or indirectly in phototransduction. With progress in genome mapping in dogs, we anticipate rapid progress in this area.

Photoreceptor dysplasia (*pd*), a type of PRA inherited only in miniature schnauzers, appears to be a unique disease in terms of its effect as well. Examination of affected schnauzers by electroretinography shows a possible problem with the generation of an electrical signal in the retina. This finding led us to question whether the gene for the channel protein, the last effector in the phototransduction pathway, was working or not. Xi Zhang in our laboratory has cloned the channel protein gene, and we are now examining this gene for mutations in the schnauzer.

Our progress this year brings us close to achieving two goals: the development of a DNA-based linkage test and the identification of the gene responsible for* prcd *and any breed-specific mutations.

ALEXIS WENSKI-ROBERTS



Kunal Ray, Gus Aguirre, and Greg Acland

Progressive rod-cone degeneration *(prcd)* is the most prevalent form of PRA that occurs in the dog. We know that it is involved in five breeds: poodles, American and English cocker spaniels, Labrador retrievers, and Portuguese water dogs. We also suspect its involvement in the disease of basenjis, Nova Scotia duck tolling retrievers, Akitas, mastiffs, Australian cattle dogs, papillons, Chesapeake Bay retrievers, and English springer spaniels.

In the past year, working with Dr. Elaine Ostrander of the Fred Hutchinson Cancer Research Center and Dr. Jasper Rine of the University of California, Berkeley, we identified a marker linked to the *prcd* locus. This information allowed us to restrict our search for the causative gene defect to a single chromosome. From there we were able to identify and clone one gene that we place quite close to the *prcd* gene. In addition, one of the research fellows in our laboratory, Weikuan Gu, has identified another marker that is linked to the disease. This information has localized the gene to a very specific region. Our progress this year has been dramatic and it brings us close to achieving two goals. The first is the development of a DNA-based linkage test that can be used to identify affected, carrier, and normal dogs with an exceptionally high degree of accuracy. The second is to identify the gene responsible for *prcd* and any breed-specific mutations. The aim for this year is to move along these two fronts very rapidly. As a direct result of these studies, it will then be possible to establish one-by-one the other breeds of dogs that possess the *prcd* defect.

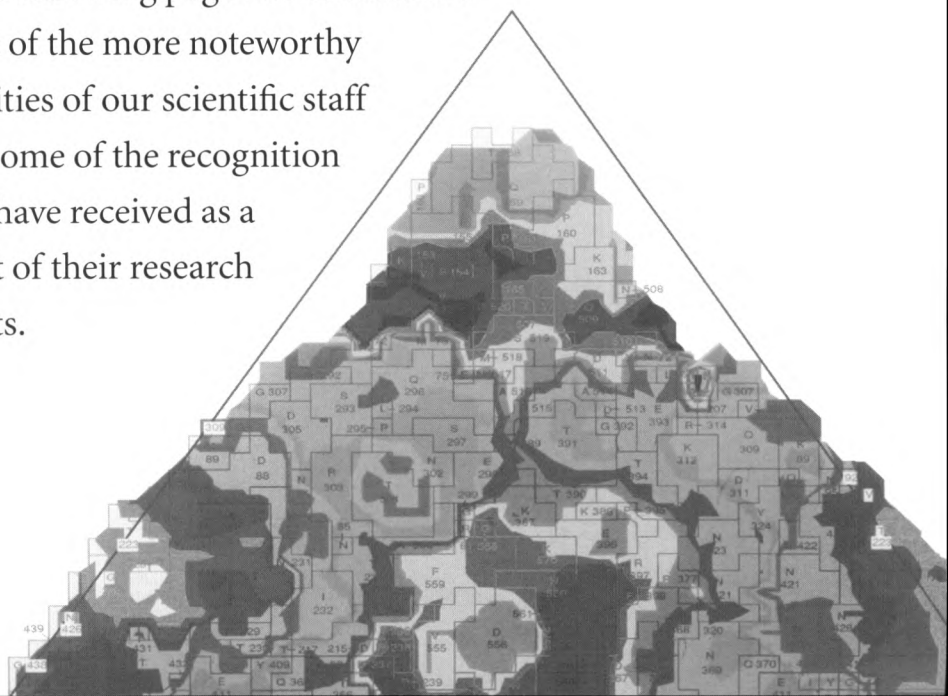
—Gustavo D. Aguirre

Beyond the Laboratory

SCIENTIFIC RESEARCH may involve many long hours of solitary experimentation in the laboratory, but it is by no means a solitary pursuit. For knowledge to advance, it must be shared. The faculty and students of the Baker Institute engage in frequent scientific interaction with their colleagues in the broader scientific community.

They do this first by teaching and by publishing their findings in peer-reviewed journals. They also travel throughout the country and abroad to lecture and confer with their colleagues in other universities, public health laboratories, and industry. A variety of seminar series at the College of Veterinary Medicine and elsewhere within the University ensure that a steady stream of experts from other institutions are brought to Cornell to share their findings with their colleagues here. The number and quality of these exchanges give a true measure of the value of a research program or institution.

In the following pages we summarize some of the more noteworthy activities of our scientific staff and some of the recognition they have received as a result of their research efforts.



Opposite: **John Parker and Wen Yuan**

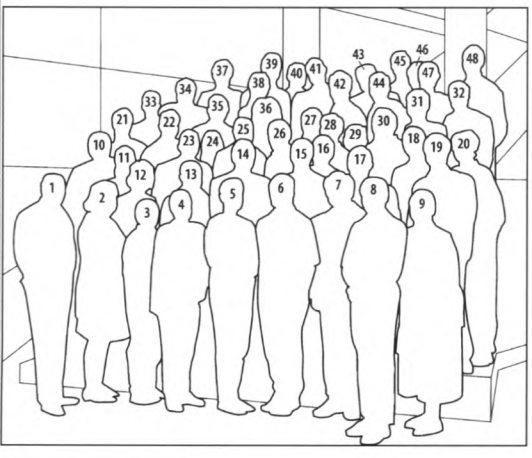
Right: **A two-dimensional map of surface residues within one unit of the 20-sided parvoviral structure**

Baker Institute Scientific Conference Series

The Baker Institute in 1996 launched a series of annual scientific conferences to address subjects of importance to the health of companion animals. The International Symposium on Hip Dysplasia and Osteoarthritis in Dogs, hosted by the Baker Institute in August, was the first meeting convened in a quarter century to assess international progress toward understanding, preventing, and treating these widespread, closely related diseases. Many of the papers presented at the conference were accepted for publication in the May 15, 1997 issue of the *Journal of the American Veterinary Medical Association*. The conference was supported by the Ralston-Purina Company, Nutramax Laboratories, Ciba-Geigy Corporation, the Orthopedic Foundation for Animals, Pfizer, and the American Kennel Club Canine Health Foundation. We would also like to extend our thanks to Clare and Eugene Thaw for germinating the notion of a symposium series through their endowment of the Thaw Seminar Fund.

ADRIANA ROVERS / CU

INTERNATIONAL SYMPOSIUM ON HIP DYSPLASIA AND OSTEOARTHRITIS IN DOGS



Participant identification:

1 George Cardinet (speaker),
2 Deborah Lynch,
3 Margaret Pough,
4 Dorte Hald Nielsen,
5 Jane Miller,
6 Reinhard Straubinger,
7 George Lust (speaker),
8 Ron Kettenacker,
9 Elizabeth O'Byrne (speaker),
10 Gail Smith (speaker),
11 Pamela McKelvie-Smith,
12 Nancy Burton-Wurster (speaker),
13 George Brewer (speaker),
14 Gregory Acland,
15 Spencer Johnston (speaker),
16 Eldin Leighton (speaker),
17 Greg Keller,
18 Doug Antczak,
19 Kiyoshi Kawase,
20 Nelly Farnum,
21 Stephen Peterson,
22 Tom Gregor,
23 Bernard Steinetz (speaker),
24 Elizabeth LaFond,
25 Darryl Biery,
26 Richard Kealy (speaker),
27 Bonnie Thomson,
28 Charlie Rodi,
29 Stephen Fox (speaker),
30 James Edwards,
31 Fuliang Du,
32 Tony Farquhar (speaker),
33 Charles DeCamp (speaker),
34 Jens Sønderup,
35 Larry Wallace (speaker),
36 Pat Fay,
37 Malcolm Willis (speaker),
38 Kurt Matushek,
39 Michael Olivier (speaker),
40 E. Al Corley (speaker),
41 Marvin Olmstead (speaker),
42 Dan Richardson (speaker),
43 Gerry Pijanowski (speaker),
44 Jeffrey Wortman,
45 Jens Madsen (speaker),
46 Phil Toll,
47 Alma Williams,
48 John Bertram

Not in photograph: Avi Deshmukh, Nathan Dykes (speaker), Allan Lepine, Kathy Linn, Sheryl Morgan (speaker), Victor Rendano, Rory Todhunter (speaker), Xuning Wang, Gary White (speaker), and Zachary Wood

Honorable Mentions

FACULTY HONORS

Gustavo Aguirre has been awarded honorary membership in two professional organizations, the Italian Society of Veterinary Ophthalmology and the Brazilian College of Veterinary Ophthalmology.

Douglas Antczak was invited to participate in a workshop on "Fetal Origins of Adult Diseases" held in London, England and sponsored by the Wellcome Trust. In 1996 he also presented the Passenger Memorial Lecture in Lexington, Kentucky. Dr. Antczak addressed the topic of heritability of racing performance in Thoroughbreds, with special reference to the bloodlines of Secretariat.

Dr. Antczak was appointed to a three-year term as a member of the Veterinary Immunology Committee of the American Association of Immunologists.

Max Appel was awarded a plaque and a certificate of appreciation for "sustained support and contributions to the study of infectious diseases of zoo and wildlife species" by the American Association of Zoo Veterinarians in November. The Association cited Dr. Appel's "meritorious research and contributions for the prevention of morbillivirus infections in exotic carnivores."

Dr. Appel and graduate student **Reinhard Straubinger** presented a paper and a poster at the 7th International Congress on Lyme Disease held in San Francisco. Dr. Appel also gave an invited lecture at the School of Veterinary Medicine of the University of California, Davis on the pathogenesis and treatment of Lyme arthritis in dogs.

Judith Appleton continues to serve as a member of Tropical Medicine and Parasitology Study Section of the National Institutes of Health.

Leland Carmichael gave a series of lectures on canine infectious diseases to veterinary students and veterinarians at the University of Bari and in Naples, Italy in May. In April he was invited by the government of South Korea to consult with Choong-Ang Laboratories on the production of canine vaccines. Dr. Carmichael also delivered lectures to veterinarians and vaccine specialists in Taejow, South Korea.

Drs. Carmichael and Appel were also invited to attend an international vaccinology meeting sponsored by the Fondation Mérieux and held in Annecy, France. Together with several European and American colleagues, they discussed the current status of companion animal vaccines.

Vicki Meyers-Wallen began a year-long sabbatical leave in England in August as a Fogarty Senior International Fellow. Dr. Meyers-Wallen spent the first half of her leave in the laboratory of Dr. Peter Goodfellow of the Department of Genetics, University of Cambridge. Dr. Goodfellow is a world-renowned expert in the genetics of development of the reproductive system.

Colin Parrish was a featured speaker at the annual meeting of the American Society for Microbiology in New Orleans. His work was also the subject of a feature article in the Microbiology Society newsletter.

Jharna Ray was presented with a plaque for excellence in aging research at the annual meeting of the American Federation for Aging Research. The award recognized her work on macular degeneration, an age-related, inherited, blinding disease.

STUDENT HONORS

Jessica Baker was awarded a travel bursary by the Rochester Trophoblast Conference to help her attend the Thomas G. Wegman Memorial Symposium on Reproductive Immunology in Banff, Canada. There she presented the results of her research on alterations of maternal immune responses during equine pregnancy.

Reinhard Straubinger won first prize among the faculty and graduate students who participated in the annual poster competition sponsored by Cornell's College of Veterinary Medicine. Dr. Straubinger's exhibit described his research to determine the possible role of interleukin-8 in initiating and maintaining the development of acute arthritis in dogs infected with Lyme disease.

DEGREES CONFERRED

David Peters, Ph.D.: "Pathogenesis of the Most Recent Variant of Canine Parvovirus: CPV-2b." Dr. Peters, who also holds a D.V.M. degree, is now a research scientist at Intervet, Inc. in Millsboro, Delaware.

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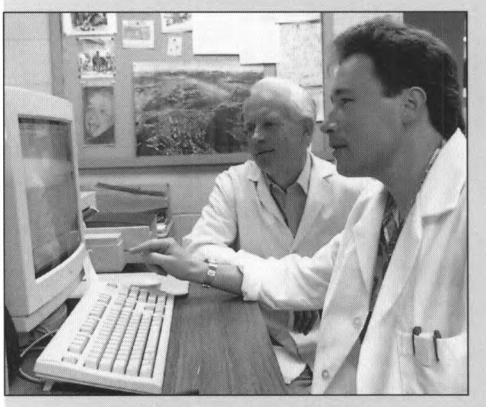
"Dissemination of *Borrelia burgdorferi* After Experimental Infection in Dogs", with Institute researchers Dr. Reinhard Straubinger and Dr. Max Appel as co-authors, was awarded second place as one of the best original scientific articles published in the *Journal of Spirochetal and Tick-borne Diseases* in 1996. Dr. Willy Burgdorfer, discoverer of the Lyme disease organism, is deputy editor of the journal.



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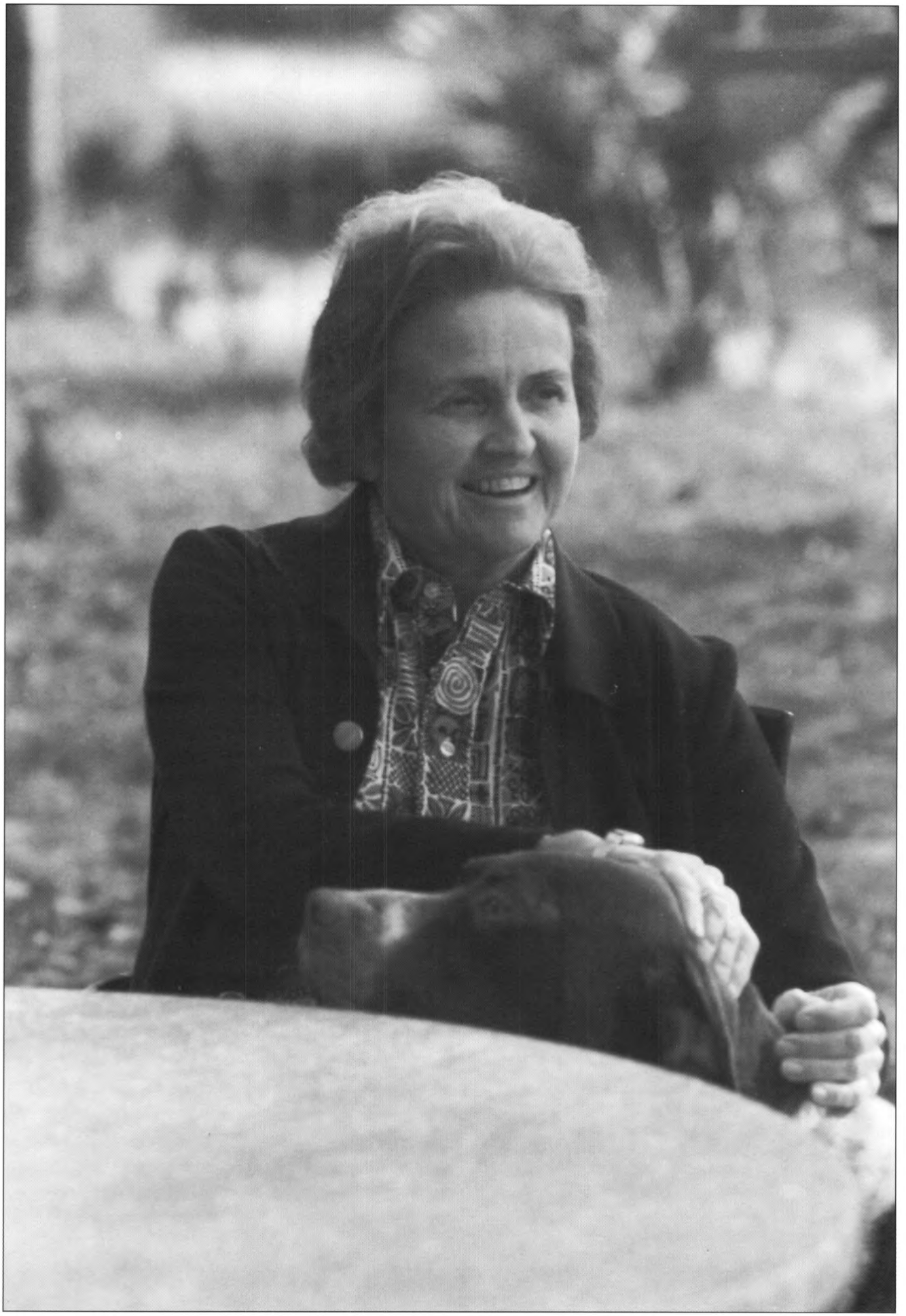

Acknowledgements

WE WISH TO EXPRESS OUR GRATITUDE to all of you whose concern for the health of dogs and other animals inspired you to contribute to the Baker Institute in 1996. Your assistance is vital to the strength and productivity of the Institute's animal health programs. A surprising number of the people named in the following pages stand out for their long years of giving, their great generosity, or both, and no donor roster is adequate acknowledgement of their contributions to our efforts. We are particularly fortunate to have their committed support.

One gift we wish to celebrate is the Gaylord and Dorothy Donnelley Foundation's fulfillment in 1996 of a pledge to endow the Dorothy R. Donnelley Faculty Career Development Award. This endowment was conceived by Mrs. Donnelley's late husband, Gaylord, and their children, Strachan, Laura, and Elliott, as a means to honor the special relationship we have been privileged to enjoy with Dorothy Donnelley. Since 1950, she has devoted not only her financial resources but also a great deal of time to building and sustaining our research programs. She served twelve years on the Advisory Council, six of them as chairwoman, and came to symbolize for us the dignity and importance of working for the well-being of animals.

The Institute would be a very different place without Dorothy Donnelley. Thanks to the farsighted generosity of her family, she will belong not only to our history but to our future, as the Donnelley Award allows us to attract and support the work of the best and brightest scientists working in animal health research today—and tomorrow.

Opposite: **Dorothy Donnelley, a cherished friend and one of the Institute's foremost benefactors, relaxing at home in the 1970s.**

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Elizabeth and Frederic Graber (in honor of Judy Wilpon)
Gracelane Kennels: Robert D. Gatti
Kathryn Grahn
Hanne Grastorf (in memory of Missy)
Steven and Arlene Grey (in memory of Rex, Dusty, and Ginger)
Gertrude L. Gutekunst (in memory of Dr. John A. Ward)
Olga Hagarty (in memory of Midge)
Dr. and Mrs. George E. Hahn
B. Joan Haines and Linda Gerber (in memory of Rosie)
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Agnes and Everette Mayer (in memory of Brenden and Knic Knac)
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Gerda H. Sauerschell
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Charles and Margaret Schreiber
Gail and Alan B. Schreier, DVM (in memory of Muttley)
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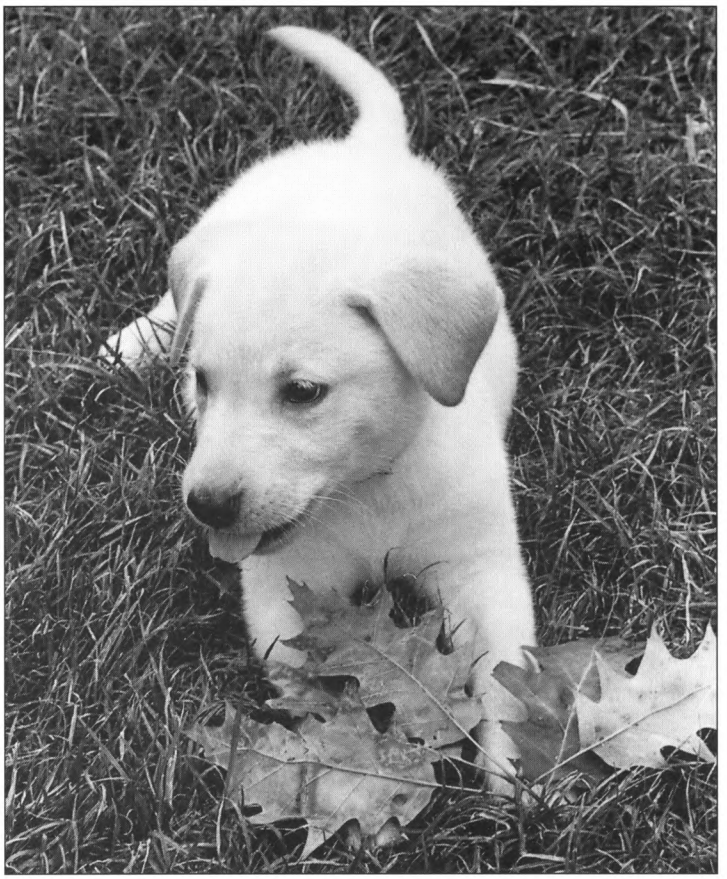

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Ruth and Jeffrey Grossman (in memory of Ernie and Betsey)
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Wendy Grudin
Raisa Gursky
Donna Gutbier
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Founders' Circle members have given $5,000 or more this year.

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Yorkshire Terrier Club of the Nation's Capital (in memory of Wilmer Webster)

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