Annual Report 1994—95

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Broomhilda and Alma Jo Williams
ORTY-FIVE YEARS and still growing! The James A. Baker Institute for Animal Health has been a center for veterinary medical research for nearly half a century. Founded in 1950 as a unit of the College of Veterinary Medicine at Cornell, the Institute has established a worldwide reputation for practical advances in infectious disease control and immunology, particularly in vaccine production for dogs.

In the 1980s a strong research program in equine immunology was begun, and in 1990 the Institute expanded in size and scope by adding to the staff several scientists with interests and expertise in medical genetics. Today, with a scientific staff of 55, the Institute is in the most productive period of its history.

In the year 2000 the Institute will celebrate the 50th anniversary of its founding. In anticipation of that event, we have developed a strategic plan to help us meet the challenges of the coming century. Major goals of our Campaign for the Twenty-First Century include:

1. establishing permanent new programs that will enhance the Institute's research capabilities,
2. purchasing the new scientific equipment needed for research at the cutting edge of technology, and
3. strengthening the academic training programs of the Institute.

Such ambitious goals will be accomplished only with the continued assistance of those individuals whose dedication to animal health and welfare has made possible the establishment and growth of the James A. Baker Institute for Animal Health. As the following examples illustrate, several key gifts and grants made during the past year are furthering our progress in all three areas.

The Ralston-Purina Company, Nutramax Laboratories, Pfizer Animal Health, and the Orthopedic Foundation for Animals have agreed to help underwrite the first meeting of the new Baker Institute Scientific Conference Series. This series will highlight the research activities of Institute faculty and their colleagues with a yearly meeting held at Cornell. The initial meeting, organized by Dr. George Lust and scheduled for August of 1996, will be the first international symposium on hip dysplasia and osteoarthritis in dogs held in over 20 years. This and future meetings will serve to stimulate new research and to disseminate practical advances to veterinarians and dog owners and breeders.

Also this summer, the Institute will establish a new core laboratory for cell and molecular biology. Purchase of a sophisticated gene analyzer for this facil-
In the year 2000, the James A. Baker Institute for Animal Health will celebrate the 50th anniversary of its founding. We invite you to join us in:

The Campaign for the Twenty-First Century

Our vision is to serve the animals that so faithfully serve mankind.

Our mission is to improve animal health through basic and applied research.

Our goal is to be the leading academic institution in animal health research worldwide.

Thank you for helping us achieve this goal.

The James A. Baker Institute for Animal Health holds a special place in veterinary medicine by virtue of its distinguished history of practical accomplishments. Today the Institute carries on that tradition proudly through the efforts of its dedicated, capable, and hard-working staff. It is my great pleasure to serve them as director and to bring you this annual report. Good reading!

—Douglas F. Antczak
The Institute in Perspective

HE JAMES A. BAKER INSTITUTE FOR ANIMAL HEALTH will turn 50 at the start of the next century. Far from settling into any middle-aged complacency, however, the Institute is moving toward this milestone with the same vigor and enthusiasm that have characterized its research efforts since the days of James Baker.

If anything, the importance of its continuing contributions to animal health may grow as the medical research community tries to anticipate the unknown but major challenges that the future will almost certainly bring to both human and veterinary medicine. The Baker Institute is prepared to play a major role in meeting those challenges.

Today, the Institute boasts excellent faculty in two important areas: infectious diseases and immunology, and genetics and development. The former group, led by veteran scientists Skip Carmichael and Max Appel, has been responsible for the majority of research in canine infectious diseases and vaccine production worldwide during the past 40 years. The advent of antibiotic therapy seemed to predict the end of the threat of infectious diseases within our lifetimes, but our perspective has changed during the past decade. The emergence of new diseases such as AIDS, the resurgence of old ones such as tuberculosis, the evolution of drug-resistant strains of bacteria, and rapid mutation of viruses to virulent new strains have provided a sobering view of the future. New infectious diseases will continue to emerge, and we must be ready to meet those challenges. The Baker Institute is one of very few research facilities worldwide with the technology and expertise to lead the fight against veterinary infectious diseases. Max Appel's research on canine Lyme disease and Colin Parrish's studies of canine parvovirus are examples of efforts already underway.

The Institute's programs in genetics and development, though relatively newer than the infectious disease work, are also anchored by experts of long standing. The major projects on inherited eye disease, led by Gus Aguirre, and canine hip dysplasia, directed by George Lust, address two of the most important categories of genetic and developmental diseases of dogs.

As is true in the study of infectious diseases, basic research is also fundamental to progress in more applied aspects of veterinary medical genetics. New technologies have greatly increased the speed with which this groundwork can be laid. For example, the task of sequencing the entire human genome, which once seemed insurmountable, is now within reach. It is gratifying to see Baker Institute scientists contributing to the gene mapping efforts in dogs and horses.
From my perspective as a human medical researcher, I see the Baker Institute as a unique resource in veterinary medicine. Research at the Baker Institute begins with the health needs of dogs, horses, and other animals, yet its relevance to human health has been recognized many times over at the national level. The awards to Institute scientists of grants from the National Institutes of Health are a measure of the excellence of their programs. Those awards also reinforce the concept of 'one medicine', and the two-way connection between human and animal health. What better testimony could there be to the quality of the programs at the Baker Institute?

The Institute's director, Doug Antczak, has the full support and encouragement of the Advisory Council in his undertakings. I have known Doug for over 20 years, and we share many friends in the world of human and veterinary medicine. While still a veterinary student Doug apprenticed himself to some of the great figures in infectious disease research of the past half century. With his background in immunology and his knowledge and interest in genetics, Doug is superbly positioned to lead the Institute into the next century. Please continue to give him and the Institute your support.

—Robert E. Shope, MD

**ADVISORY COUNCIL**

**Robert E. Shope, M.D., Chairman**
Professor of Pathology, Microbiology, Immunology, Preventive Medicine, and Community Health; Center for Tropical Diseases, University of Texas

**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, Laboratory of Retinal Cell and Molecular Biology, National Eye Institute of the National Institutes of Health, Bethesda, Maryland

**Strachan Donnelley, Ph.D.**
Director of Education, The Hastings Center, Briarcliff Manor, New York

**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

Dean Emeritus, School of Veterinary Medicine, University of Pennsylvania

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

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

**Judith Wilpon**
Locust Valley, New York

**FORMER ADVISORY COUNCIL MEMBERS**

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

**Dorothy R. Donnelley**
1980–1992
Chairwoman, 1982–1988

**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

**Frederick A. Murphy, D.V.M., Ph.D.**
1984–1994

**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

*Deceased 1994
THE ARTHUR F. NORTH, JR. CANINE SERVICE AWARD

The North Award was established in 1982 to honor the memory of Dr. North, a 1935 graduate of Cornell’s College of Veterinary Medicine. Dr. North was a skilled and innovative practitioner and an enthusiastic friend of the Baker Institute. The North Award recognizes those whose contributions to canine health and well-being reflect his spirit of concern for all dogs. The women we honored in 1994 and 1995 manifested that spirit through hard work... and dogged determination.

1994 ♦ Dolly Trauner

At the dinner following the Annual Meeting of the Advisory Council, Gustavo Aguirre presented the North Award to Dolly B. Trauner, a longtime dog breeder and show judge and past president of the Pomeranian Club of America. Mrs. Trauner was honored for her selfless and persistent 25-year effort to promote recognition of the problem of inherited eye diseases in dogs.

In 1974 Mrs. Trauner established the Canine Eye Registration Foundation (CERF). Mrs. Trauner worked full-time, on a volunteer basis, to run the organization from her home before finally entrusting CERF and her considerable responsibilities to Purdue University in 1988.

Mrs. Trauner has worked closely with the American College of Veterinary Ophthalmologists and with breed clubs to encourage the organization of clinics for large-scale screening of dogs by a board-certified ophthalmologist. The results of those examinations are entered anonymously in CERF’s national database and used to compile statistics by breed, type of disease, and other factors relating to affected dogs. The information that has been gathered and analyzed since CERF’s founding has provided breeders with the first reliable tool to help slow the proliferation of inherited eye diseases of dogs. CERF’s profits were donated to fund canine eye research at the Institute.

In 1985 Mrs. Trauner alerted the veterinary ophthalmology community to the observation that PRA in the Siberian husky affected males in overwhelmingly disproportionate numbers. Mrs. Trauner wrote, “Just off the top of our heads, it
would appear that PRA in Siberian huskies is sex-linked, and would therefore be genetically different from any other type of PRA seen in other breeds.” That letter prompted Dr. Aguirre and colleague Gregory Acland to begin studying the disease. Their work, which was recently completed, proved the validity of Mrs. Trauner’s observation.

1995 • Barbara J. Hartsig
Barbara Hartsig first heard of the Institute when veterinarians John Garman and Greg Brzoska made a gift in memory of her German shepherd, Tanya. She responded with a gift of her own. When her collie, Butch, died the following year, she made another gift, and continued to remember her pets every year. Although Mrs. Hartsig’s own means were limited, it was her position as bookkeeper for a law firm that ultimately enabled her to influence the endowment of a scholarship fund for graduate research at the Institute.

Mrs. Hartsig’s employer also happened to be the president of the Carl J. Herzog Foundation. In 1988 she ventured to submit a modest proposal to the Herzog Foundation board on behalf of the Baker Institute. The trustees honored her request, and continue to do so every year.

Carl J. Herzog’s late daughter, Johanna-Maria Fraenkel, strongly supported the cause of animal health and welfare. When Mrs. Hartsig learned that her trust fund was to be dissolved, she lobbied for the Baker Institute to receive a portion of the trust. The Johanna-Maria Fraenkel Scholarship Endowment was established at the Institute in 1995 as a result of her efforts.

THE FOUNDERS’ AWARD
The Founders’ Award was established in celebration of the Institute’s 40th anniversary in 1990. This recognition 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.

1994 • Dr. Clayton Dudley
The Baker Institute was pleased to honor G. Clayton Dudley, DVM ’64 with the 1994 Founders’ Award. Dr. Dudley was joined for the presentation by his daughter, Wynne, and his wife, Georgia, who manages his veterinary practice in Woodbury, Connecticut.

Dr. Dudley is an active civic volunteer and community mentor, promoting responsible pet care in the schools and in the community and helping to prepare students for careers in veterinary medicine and other fields. He and Mrs. Dudley have been dedicated and innovative contributors to the Baker Institute for many years. They have twice undertaken the daunting task of organizing an art show...
at their hospital as a benefit for the Institute. *Art for the Animals* has been a resounding success, and is on its way to becoming a Woodbury tradition.

1995 • Dr. John Ward

John A. Ward, DVM '36 has been a Research Partner since 1963. At that time he had a practice on Staten Island, where he and his wife, Mary Clare, had gone after graduation to take a job at the newly built Staten Island Zoo. For two years Dr. Ward worked at the zoo during the day and built a practice at night. His was a mixed practice, as livestock herds—and backyard pigs—were still a common sight on the island in those days. The Wards also built a family, raising their three daughters above the office. In 1995 they celebrated their 60th wedding anniversary.

Dr. Ward was unable to make the trip to Ithaca to receive the Founders' Award, but we were honored to have Mrs. Ward and daughter Lynn in attendance. Mrs. Ward brought a videotaped acceptance speech from Dr. Ward and read additional remarks that he had prepared. It was another highlight of a very moving evening.

1995 • The Irish Setter Club of America

The Irish Setter Club of America has lent years of vital assistance to Dr. Aguirre’s efforts to identify the genetic basis of progressive retinal atrophy in the Irish setter and in other breeds of dogs. His group’s recent development of a blood test to determine the PRA carrier status of Irish setters owes much to the Club’s loyal support. Club director Connie Vanacore has been an especially important advocate of the PRA project, and Dr. Aguirre had the pleasure of presenting the award to her and to one of her fellow directors, Mr. Craig Hackenberg, in Morristown, New Jersey.
Dr. Edward Grano, Jr.

Dr. Grano, a 35-year supporter of the Baker Institute, died on April 6, 1994 at age 68. Dr. Grano came to Cornell's College of Veterinary Medicine after his World War II Army service, during which time he participated in the Battle of the Bulge. He graduated with a degree in veterinary medicine in 1952, then practiced in Briarcliff Manor, New York for nearly 40 years. He was active on the state and local levels of the SPCA and the New York State Veterinary Medical Society.

The magnitude of the tribute made by the donors listed below testifies to the depth of admiration and affection that Dr. Grano inspired in his community and his colleagues. As Elizabeth Grano wrote of her husband: "Dr. Grano was very proud of his profession, and firmly believed that knowledge, service, kindness, and respect were the hallmarks of a successful practitioner. His whole life reflected these qualities."

Gifts received in memory of Edward Grano, Jr., DVM '52

Jean K. Acker
Victoria T. Allen
Mary Lou and Edward Anderson
Robert J. Arminio
Bette Ann Arnold, DVM
Lorraine M. Barstow
Thomas A. Beale
Gloria Beasley
Clare and Gregory Bivona
Mary Borrelli
Mary F. Brown
Carolyn V. Bruehn
Burke Rehabilitation Hospital, Physical Therapy Department
Mr. and Mrs. Joseph M. Callahan, Jr.
Rocco Cambareri
Jane Cane
Edward F. and Carol Carini
Paula Chadwick
Bobby and Nancy Checchi
Lois Jean and Jim Colgan
Ann W. Cooper
Joyce and Broward Craig
William and Dolores Creeden
Dennis and Cindy Curran
Peter and Kathy DiPrima
Carla and Peter Di Yorio
John O. and Alice Doern
Bill and Jo Ann Doran
Stephen and Linda Egbert
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Alan and Judy Duke
Esther and Louis Fischer
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John L. Frasca
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Gracelane Kennels
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Ann Hoffman and Ira Rosenfeld
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Leslie and Sandra Jenkins
Anne and Bob Klami
Gloria and Joseph Kurilecz and family
Elizabeth and Richard Leins
Carolyn and Herrick Lengers
Cynthia Livingston
Mario and Ann Lopez
Elaine Lorson
Debra A. Lovecky and family
Jean Luongo
Max and Alice Marxreiter
Barbara and Robert Mavian, DVM
Charles and Janet Maxwell and family
Deb and Carl Mehne
Peter and Rosalie Menegas and family
Anne and Louis Meszaros
Marion S. Miller
Anne and Bob Morales
Margaret Naughton
Allen E. Nichols
Cindy Nolting
Sandra and G. Richard Parrino
Helen H. Pearson
Amy G. Price
Angelina Psaras
Katherine P. Reilly
Dr. Arnold M. Remson
Clark and Mary Sue Robson
Jack and Joanne Rodwell
Barbara and Robert Rohrbach
Filomena Schechter
Joy Seaman
Carole J. Seiden and family
Marilyn and Jerry Slater
Mark and Patricia Sofranko
Marilyn Sole
Barbara Stewart
Marian Tavolacci
Mrs. Edwin W. Underhill
Erika Rindler Urbach
Joan and Peter Vlahakos
Myra Yosburgh
Dr. and Mrs. Robert Wainwright
Elizabeth L. Wiltse
Charles and Susanne Ziegler
Barbara Zinke

Dr. William C. Beck, surgeon, researcher, inventor, essayist, and twelve-year member of the Baker Institute's Advisory Council, died on August 11, 1994 at the age of 86. Dr. Beck was president emeritus of the Donald Guthrie Foundation for Education and Research and retired chairman of the Department of Surgery of the Robert Packer Hospital and Guthrie Clinic in Sayre, Pennsylvania. He authored three books, more than 450 articles, seven book chapters, and his memoirs, which he completed only hours before his death. They have since been published in book form.

In addition to a distinguished career as a surgeon, Dr. Beck was known internationally as a research scientist and a noted inventor who held patents on a number of widely used devices designed to protect surgical patients and staff from contamination with infectious agents. In the year before his death he gave the rights to two of those patents to the Guthrie Foundation, explaining, "I want the Foundation to prosper, so that it can do the work it was set up to do." For his contributions to improving operating room and hospital lighting, he was named a fellow of the Illuminating Engineering Society, the only physician to be so honored. He was also a fellow of the American College of Surgeons and an active member of several other professional associations. In 1991 the Guthrie Medical Center named the William C. Beck, M.D. Health Science Library and Resource Center in his honor.

Dr. Beck's experience, enthusiasm, humanity, probing intellect, and dedication to excellence served the Institute well and lent an important perspective to the deliberations of the Advisory Council. He will be sorely missed.
Honorable Mentions

FACULTY HONORS

Gustavo Aguirre was named in 1995 to the newly established Science Advisory Committee of the American Kennel Club's Canine Health Foundation. Dr. Aguirre also co-organized the AKC-sponsored Conference on Molecular Genetics and Canine Genetic Health held in October of 1994.


Back in the United States, Dr. Aguirre lectured on "Inherited Disorders of the Eye" at the American Veterinary Medical Association meeting on Molecular Therapeutics in Veterinary Medicine in Pittsburgh. He also spoke on "Mechanisms of Photoreceptor Disease and Degeneration" at the Second Great Basin Visual Science Symposium at the University of Utah School of Medicine in Salt Lake City.

Douglas Antczak was invited to speak in 1994 at the International Symposium on Genetics and Disease in the Horse held in Interlaken, Switzerland. The title of his talk was "Major Histocompatibility Complex in the Horse: Structure and Function." In 1995 Dr. Antczak gave an invited lecture, "Maternal Immunological Response to the Equine Conceptus," at the Workshop on Reproductive Immunology sponsored by the Society for the Study of Reproduction and held in Davis, California.

Max Appel presented papers in 1994 at the Sixth International Conference on Lyme Borreliosis and at the International Symposium on Morbillivirus Infections, which was held at the Hannover Institute of Virology and Immunology in Germany. Dr. Appel also spoke at the Centennial Anniversary Conference at Cornell's College of Veterinary Medicine and at the annual meeting of the American Association of Zoo Veterinarians.

Judith Appleton accepted an invitation to serve as a member of the National Institutes of Health's Tropical Medicine and Parasitology Study Section, Division of Research Grants, for the term beginning July 1, 1995 and ending June 30, 1999.

Robin Bell was appointed director of the Immunology Unit of the Department of Microbiology and Immunology of the College of Veterinary Medicine. He is taking a six-month sabbatical leave at the John Curtin School for Medical Research at the Australian National University in Canberra.

Leland Carmichael was awarded the degree of Doctor honoris causa by the Veterinary Faculty of the University of Liège, Belgium in October 1994. In that same year, Dr. Carmichael was named the first recipient of the American Kennel Club Career Achievement Award, presented by the American Veterinary Medical Association's Council on Research at the AVMA's annual meeting. Dr. Carmichael delivered the keynote AKC lecture, "Canine Parvovirus Type-2: New Realities and Old Myths Revisited", at the meeting's Advances in Veterinary Medicine Symposium.

In 1995 the National Veterinary School in Lyons, France invited Dr. Carmichael to present a lecture at the 15th annual Entretiens de Bourgelat. Dr. Carmichael spoke on "Vaccines and Vaccine Research—100 Years After Pasteur". Also in 1995 Dr. Carmichael was appointed Adjunct Professor at the Institute of Infectious and Parasitic Diseases of Animals in the School of Veterinary Medicine of the University of Bari in Rome, Italy.

Vicki Meyers-Wallen was promoted to associate professor in 1995. In recognition of her expertise in canine fertility and infertility, she was elected to serve on the Diplomate Examination Committee of the American College of Theriogenologists for a term ending in 1998.

Dr. Meyers-Wallen spoke by invitation at the annual meeting of the American Veterinary Medical Association in San Francisco, giving talks on breeding management, elective Caesarean section, male infertility, and mismating options.

Colin Parrish was promoted in 1994 from assistant to associate professor. He addressed the Annual Meeting of the American Society for Microbiology on "Canine Parvovirus and its Relatives—Understanding the Mechanisms Involved in the Emergence and Evolution of a New DNA Virus".

Jharna Ray was invited to lecture in 1995 at the National Eye Institute in Bethesda, Maryland, where she spoke on "Retinal
The Veterinary Faculty of the University of Liège in Belgium conferred the degree of Doctor Honoris Causa on Leland E. Carmichael, the Baker Institute’s John M. Olin Professor of Virology, in September 1994. Dr. Carmichael and his wife, Mary Margaret, are shown standing between Professor and Mrs. Paul-Pierre Pastore, left, and fellow honoree Dr. Francisco Bolivar Zapata and his wife, right.

Pigment Epithelium—A Model System to Study Disease Expression and Correction,” and at the University of Rochester Medical Center, where her talk was entitled “The \textit{in vitro} RPE—An Experimental Model to Examine Lysosomal Function and Dysfunction”.

**STUDENT HONORS**

Philip Peters and Allan Tsung received undergraduate honors in 1995 for their thesis work in the Appleton laboratory. The title of Philip Peters’s thesis was “Dominance of an IgG Isotope in the Immune Response to \textit{Trichinella spiralis}”. Mr. Peters graduated \textit{magna cum laude} from the College of Arts and Sciences and has moved on to a research fellowship at the Harvard School of Public Health. He has spent several months of that time in Brazil studying schistosomiasis. He will enter Cornell Medical College in the fall of 1996.

Allan Tsung’s thesis title was “Characterization of Surface Antigens of \textit{Trichinella spiralis}”. Mr. Tsung graduated \textit{cum laude} from the College of Agriculture and Life Sciences and is attending medical school at the State University of New York Downstate campus in Brooklyn.

Christopher DeSanto received undergraduate honors from the College of Agriculture and Life Sciences for his research on mucopolysaccharidosis (MPS) in the laboratory of Jharna Ray. Mr. DeSanto worked on the project from spring, 1994 through summer, 1995 and was awarded a Howard Hughes Summer Fellowship for 1995. He is now enrolled in medical school at the State University of New York at Brooklyn.

Derek Wilson, a 1995 Summer Leadership Program student in Jharna Ray’s laboratory, received the Best Program award for his work with MPS.

Summer Leadership Program student Maria Lara Tejero was honored for the best presentation of a research project for her work with Kunal Ray on canine progressive retinal atrophy.

**DEGREES CONFERRED**

Lauri Ellis, Ph.D. 1994: “Analysis of \textit{Trichinella spiralis} Surface and Secreted Glycoproteins that Bind Protective Antibodies”. Dr. Ellis is now a postdoctoral fellow at the Palo Alto Medical Foundation of Stanford University.

Gabriele Grünig, Ph.D. 1994: “The Maternal Leukocyte Response to the Invasive Trophoblast in the Horse”. Dr. Grünig, who is also a veterinarian, is working as a postdoctoral fellow in the Department of Immunology of the DNAX Research Institute of Molecular and Cellular Biology in Palo Alto, California.

Juli K. Maher, Ph.D. 1994: “Control of Expression of Major Histocompatibility Complex Class I Molecules in the Equine Trophoblast”. Dr. Maher, who also holds a D.V.M. degree, currently works as a postdoctoral fellow in the Department of Microbiology and Immunology of the UCLA School of Medicine.

Dai-wei Zhang, Ph.D. 1994: “The Expression of Fibronectin in Normal and Osteoarthritic Canine Articular Cartilage.” In recognition of his work as a graduate student, Dr. Zhang was awarded the Liu Memorial Award for Chinese graduate students. He is continuing his training as a resident in radiology at the Nassau Medical Center on Long Island.

Clarissa L. Santos, M.S. 1995: “RT-PCR Analysis of Cytokine Production During the First 72 Hours of \textit{Trichinella spiralis} Infection”. Ms. Santos is currently working as a research assistant at the University of Seattle.

Dina Barbis Tresnan, Ph.D. 1995: “Analysis of Canine Parvovirus Cell Interactions”. Dr. Tresnan is now a research assistant professor in the Department of Microbiology at the University of Colorado Health Sciences Center. Dr. Tresnan’s last year of study in Colin Parrish’s laboratory was funded by an individual Physician and Scientist Award from the NIH. Dr. Tresnan also holds a veterinary degree.
Staff of the
Baker Institute

ADMINISTRATION

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

Susan Howell Hamlin
Administrative 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

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. Shefky
Casparcy 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; Ph.D., Cornell; Ph.D(hc); Diplomate, American College of Veterinary Microbiologists

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

Amy L. Kloster
Laboratory technician: B.S., William Smith College

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

Vesna Novosel
Postdoctoral associate: D.V.M., U. of Zagreb

Alix F. Straubinger
Postdoctoral associate: D.V.M., U. of Munich

Reinhard Straubinger
Graduate research assistant: D.V.M., U. of Munich

Mary Beth Matychak
Research technician: U. of Evansville

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

A. T. M. Wahid
Postdoctoral associate: M.D., Chittagong Medical College

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

Dina B. Tresnan
Graduate research assistant: B.S., Stanford U.; D.V.M., U. of California

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

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

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., Ph.D., New York U.

Anthony R. C. Farquhar
Postdoctoral associate: B.S., U. of Massachusetts; M.S., Ph.D., Cornell

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

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

Dai-Wei Zhang
Graduate research assistant: M.D., Beijing Medical U.

Caroline F. Borden
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

Margaret S. Vernier-Singer
Laboratory technician: B.S., Ohio State U.

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

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
Postdoctoral 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

**Hsi Liu**  
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BRUCELLA CANIS WAS FIRST RECOGNIZED as a cause of abortion and sterility in dogs in 1966, when it was isolated from field trial beagles at the Baker Institute. Since its discovery, the extent of brucellosis in field trial dogs and breeding kennels has never been defined.

However, the number of serum samples submitted to Cornell for canine brucellosis testing in 1994 jumped more than 75 percent over the previous year, while the proportion of seropositive samples remained constant at 5.6 percent. In 1994 our laboratory and colleagues in Cornell's Diagnostic Laboratory diagnosed the disease in dogs from 25 states, with the highest rates found in the South. However, reports from veterinarians in the Midwest also suggest an increase in the number of infections in that region. Several large kennels that breed pups for the wholesale market have been identified as infected.

In addition to causing irreversible sterility in males, canine brucellosis is responsible for infections of the prostate gland and testes and commonly leads to testicular atrophy. Infected dams may abort several litters in succession. Both sexes may develop uveitis in the eye or diskospondylitis, a bone disease. The infection occasionally spreads to humans, though with less serious consequences.

Although successful treatment has been achieved in dogs infected for durations of less than three months, antibiotic therapy has been generally unsuccessful in dogs with unknown infection histories. Dogs that are found to be infected must be neutered. After neutering, treatment may be attempted in exceptional circumstances, but the cost will be great and the results uncertain. Our experience indicates that dogs with any opportunity to spread the disease should be euthanized, especially in breeding kennels.

With so much at stake, accurate diagnosis is essential. The commercial slide-agglutination tests and the tube-agglutination test used in the majority of veterinary diagnostic laboratories commonly have a false-positive rate of about 50 percent. That means that as many as half of the dogs diagnosed with canine brucellosis (and often subsequently euthanized) are in fact not infected. Our laboratory maintains a close link with Cornell's Diagnostic Laboratory, where the use of tests developed at the Baker Institute (using a mutant B. canis M-strain) has reduced the rate of false-positive results to less than twelve percent. If an animal's serum reacts with the M-antigen, the serum is then tested by an agar-gel immunodiffusion (AGID) test, using a cytoplasmic protein antigen of very high specificity for brucella antibodies. If both tests are positive, blood cultures are then requested to confirm the serodiagnosis.

Before introducing a dog into a kennel, it is essential to obtain negative results
from two tests performed one month apart. This testing is critical because it is often difficult to recognize that a dog is infected with brucellosis, and poor reproductive performance may be the only indication that brucellosis is present in a kennel. Stronger prevention measures are needed, especially in commercial breeding kennels. The American Rabbit Hound Association has been progressive in ruling that all dogs entering pack competition must first present a negative brucellosis test result. Widespread emulation of this policy, which is unfortunately a rare one, would have great significance for the control of canine brucellosis.

The Baker Institute has a pamphlet on canine brucellosis that can be obtained by writing to the Office of the Director.

— Leland E. Carmichael
IT IS ALWAYS DANGEROUS to become complacent in the battle against infectious diseases. Old diseases that seemed to be vanquished can appear in new hosts, as in the case of canine distemper virus in African lions. Our involvement in the recent epidemic is described below. In addition, new diseases continue to emerge, threatening both humans and animals. Thus it is with Lyme disease.

LYME BORRELIOSIS IN DOGS

One of the main problems for patients with Lyme disease, whether canine or human, is the acute and chronic arthritis that is seen months to years after infection. There is little information available about the pathogenesis of this syndrome.

Because the clinical signs are somewhat similar to those of rheumatoid arthritis, it has been speculated that immune complexes may be involved in Lyme arthritis. Another explanation may be that the migration of borrelia (the disease-causing organisms) into the joints provokes an inflammatory process. After infection by tick bite, borrelia are known to persist in dogs as well as in humans, perhaps for a lifetime if not treated. We have initiated studies to address that question.

Reinhard Straubinger, a veterinarian who is a graduate student in this laboratory, has accumulated data in an in vitro model and from synovial fluids collected from dogs with acute arthritis. In synovial fluids, as well as in cultured joint tissue, a factor was found that attracted the invasion of a specialized form of white blood cells called neutrophils, which in turn initiated a sequence of events leading to arthritis. The triggering factor appears to be interleukin-8 (IL-8), a powerful cytokine that attracts neutrophils. In earlier reports on Lyme disease it was suggested that tumor necrosis factor (TNF), interleukin-1 (IL-1), or interleukin-6 (IL-6) might be responsible. The direct involvement of TNF could be ruled out by our data, and tests for IL-1 and IL-6 are in progress.

Another important question in Lyme disease is whether borrelia survive and persist in tissues even after antibiotic treatment or whether they are successfully eliminated. We have initiated a study in which dogs are treated for a 30-day period with either amoxicillin or doxycycline. Three and six months later the dogs will be tested for persistent borrelia.

CANINE DISTEMPER IN LIONS IN THE SERENGETI

In 1992 we reported a fatal outbreak of canine distemper in large cats (lions, tigers, leopards, and a jaguar) in a zoological park in California. In 1994 a similar outbreak occurred in lions in the Serengeti in Tanzania that killed an estimated 30 percent of the lion population.
Material and information were provided by Drs. Melody Roelke-Parker, Craig Packer, and Linda Munson, who suspected canine distemper from histopathological studies. We were able to confirm the diagnosis by immunocytochemistry, which was performed by pathologist Brian Summers, and by virus isolation. In studies with monospecific antibodies, we found the isolated viruses to be identical to virulent canine distemper virus.

During the last two years we were pleased to welcome Vesna Novosel, a veterinarian from Croatia, and Alix Straubinger, a veterinarian from Germany, who joined our laboratory as postdoctoral associates.

— Max J. G. Appel
The question of how viruses or bacteria evolve and adapt to different hosts and environmental conditions has become an important public health issue over the past few years. Many microbial pathogens are changing to adapt to altered environmental conditions and to changes in their hosts' behavior.

There is greater recognition of the potential for the selection and emergence of new and better adapted pathogens. Although much attention has been given to recently emerging viruses infecting humans—such as human immunodeficiency virus, Ebola virus, or hantavirus infection in the American Southwest—similar emergence of viruses infecting other animals can be of veterinary importance, and can also provide basic information about the processes that result in the emergence and selection of new infectious agents.

Canine parvovirus (CPV) is a recently emerged canine virus that appears not to have existed in dogs in the USA or most other nations prior to 1978. In that year canine parvoviral disease was recognized in many countries around the world, and the virus was isolated in tissue culture. The development of safe and effective vaccines, initially at the Baker Institute, has allowed the disease to be largely controlled. However, mysteries remain concerning the origin of CPV, the reason it arose when it did, the properties of the virus that affect host range, and whether CPV has varied or remained the same since it began circulating in dogs.

Viruses similar to CPV have long been known to infect cats, mink, and raccoons, although those viruses have never been associated with disease occurring in dogs prior to 1978. To try to understand where CPV came from and how it has been changing, we have been comparing CPV isolates collected at different times, comparing CPV to the other viruses, and defining the true host range of CPV.

The genome of each parvovirus is made up of a specific sequence of DNA bases, and the relatedness of viruses can be determined by comparing the sequences of their genomes. In general, the more similar the genomes of two viruses (or other organisms) are, the more closely they are related. Comparing the DNA sequences of viruses that cause disease in cats, mink, raccoons, and foxes reveals that all of the viruses are very similar, suggesting that those viruses can spread between the different host animals. In contrast, CPV strains were distinct, and were all clearly derived from a single ancestral virus. This is consistent with the idea that CPV is a host-range variant that emerged once and then spread around the world as a new virus of dogs.

As it seemed likely that CPV derived as a variant of a cat virus, and as it also replicated in cat cells in tissue culture, we
expected that it would also be able to infect cats. The early strains of CPV—those collected from sick dogs prior to 1980—did not replicate in cats, but we have recently discovered that the newer strains of CPV naturally infect cats, and can be isolated from cats with clinical disease. The evolution of the host ranges of these viruses appears to be a model for how new viruses can emerge and vary in important functions such as host range through only a small number of mutations in their coat protein genes.

—Colin R. Parrish
HIP DYSPLASIA is a major orthopedic disease of dogs that results in pain and osteoarthritis. It is characterized by hip joint laxity and subluxation, or partial dislocation, of the femoral head. The diagnosis of dysplasia is based on hip radiography of dogs at two years of age. The genetic basis of hip dysplasia is well known, but the exact cause has not been established. The odds that pups born to parents with radiographically normal hip joints might become dysplastic are about one in four.

THE SEARCH FOR A GENETIC MARKER

Canine hip dysplasia is a quantitative inherited condition, meaning that it appears to be controlled by a combination of sites, or loci, in the DNA of the dog. Each gene locus controls the expression of a different trait that is inherited in Mendelian fashion; the disease, and perhaps also the degree of severity of the disease, results from some combination(s) of abnormal traits. We are using genetic linkage analysis in an effort to identify one or more of the genetic abnormalities that have a role in the development of hip dysplasia. Success in this work depends upon: a) the identification of variable DNA sequences (polymorphisms) that have a high degree of co-inheritance (linkage) with the loci involved in the inheritance of hip dysplasia; b) the development of a pedigree in which the genotype of the parents is known to be either dysplastic or non-dysplastic; and c) an accurate determination of the phenotype, i.e., precise physical definitions of dysplastic and normal.

With the support of the Ralston-Purina Company, we have begun to look for DNA polymorphisms in our study pedigree of Labrador retrievers, and Michael Olivier, a graduate student in our laboratory, has generated some interesting leads. Our approach to finding polymorphisms is to purify genomic DNA isolated from canine white blood cells and then use random nucleotide sequences as primers that bind to the DNA. The segments of the DNA that are randomly selected in this way are amplified using the polymerase chain reaction (PCR). By using ten-nucleotide primers, this method can span the whole genome to detect possible markers linked to a quantitative trait locus. We have so far identified thirty polymorphic DNA sequences that follow a Mendelian inheritance pattern in this family of dogs. We are testing to determine whether one or more of these polymorphic sequences shows linkage with hip dysplasia in our
Above: George Lust

Left: DNA was purified from white blood cells from eight Labrador retrievers, three greyhounds, and one beagle, and reacted with random nucleotide primer OPT-14, followed by PCR. The PCR reaction generated the DNA sequences shown in the figure. After staining with ethidium bromide the bands were visible under ultraviolet light. The RAPD sequence, indicated by the white arrow, was identified in five Labrador retrievers, all of which had hip dysplasia on radiographic examination. Three other Labradors, three greyhounds, and one beagle had disease-free hip joints.

pedigree. A high degree of association between the inheritance of a polymorphism and the development of hip dysplasia would help us pinpoint the location of the gene defect and develop a diagnostic blood test to predict the development of the disease.
—George Lust
OSTEOARTHRITIS
Healthy cartilage is smooth, resilient tissue that resists compression and minimizes friction from joint movement. Osteoarthritis, the often painful consequence of canine hip dysplasia, involves damage to the cartilage of the hip or other joints. We are interested in understanding the changes that occur in cartilage in very early disease and then modeling these responses in laboratory culture. Our goal is to learn how best to interrupt the progression of these earliest changes to more serious consequences.

A UNIQUE FORM OF FIBRONECTIN
We have focused much of our effort on learning more about fibronectin, an adhesive glycoprotein found in blood plasma and connective tissues throughout the body. In the hips and other joints, fibronectin is important to the organization and function of the matrix surrounding the cartilage cells. With the help of Daiwei Zhang, who completed his Ph.D. degree in our laboratory in 1994, we made significant progress in our studies of cartilage fibronectin. Dr. Zhang cloned and sequenced the "ED-B" segment of the canine fibronectin gene, a region that is of particular interest to us because it is found with some frequency in the RNA of cartilage fibronectin but only rarely in the RNA of the fibronectins that compose other adult tissues. Dr. Zhang showed conclusively that the form of fibronectin found in cartilage is distinct: 30 percent of the RNA that synthesizes fibronectin in adult cartilage contains the ED-B positive sequence.

When cartilage becomes osteoarthritic, its fibronectin content increases dramatically. The question that had challenged us for a long time was whether this increase represented active fibronectin synthesis by the cartilage or passive penetration of joint fluid proteins into a damaged cell matrix. In an effort to answer this question, Dr. Zhang developed an antibody that recognizes only the ED-B segment of fibronectin. He used this antibody to make two important observations: both ED-B positive and ED-B negative fibronectin increased in marked and equal proportion in osteoarthritic cartilage, but very little or no ED-B positive fibronectin could be detected in the synovial fluid of the joint. These results clearly showed that fibronectin accumulation reflects a primary change in the biosynthetic patterns of cartilage cells in the disease process. We were delighted to learn that observations made about human tissue in the laboratory of Vickie Bennett, our colleague in Philadelphia, both confirmed and complemented our own results.

In collaboration with Baker Institute colleagues James MacLeod and Da-Nian Gu, we recently made another important discovery about the structure of cartilage fibronectin. Fibronectins from different cell types were known to vary in structure at three different regions of the molecule: the ED-B segment, which was the focus of Dr. Zhang's work; the ED-A segment, which is barely detectable in cartilage; and the V segment. In our work
with Dr. MacLeod and Dr. Gu, we found that all of the V region plus two other RNA segments, III-15 and I-10, are deleted from approximately 80 percent of the fibronectin RNA messages expressed by cartilage cells. Our discovery of this novel splicing pattern, which we have designated (V+C), is exciting for several reasons. First, it very nicely explains our earlier finding that some cartilage fibronectin subunits are smaller than expected and cannot react with monoclonal antibody that recognizes the III-15 segment. Second, it is an unexpected result for investigators interested in fibronectin research and may have important conceptual implications for the field in general. Finally, since there is strong evidence that the (V+C) fibronectin is associated almost exclusively with cartilage, its study promises to further our understanding of cartilage structure and function in particular.

MODELING EARLY OSTEOARTHRITIS IN VITRO
Our experimental efforts to model some of the early changes in osteoartritic cartilage also produced promising results. We have approached this work from two aspects, attempting to simulate in culture both the biochemical and the mechanical environmental factors that trigger osteoarthritic changes in healthy joint cartilage. Through chemical alteration of the culture medium in which we maintained samples of normal cartilage, we succeeded in eliciting two behaviors characteristic of osteoarthritis: increased synthesis and retention of fibronectin; and increased turnover of proteoglycans. We accomplished this by adding TGF-β1, a biologically important molecule involved in signaling cells to alter their behavior, and fucoidan, a polysulfated sugar polymer, to cartilage cultures.

Through the work of postdoctoral associate Tony Farquhar, we continue to study the effect of mechanical load on joint cartilage. After applying high rates of repeated impact loading to the cores of cultured cartilage discs, we examined the cartilage by several techniques. By means of proton magnetic resonance imaging performed by Yang Xia, a member of Lynn Jelinski’s laboratory in Cornell’s Biotechnology Program, we were able to detect subtle signs of swelling and weakening in the collagen matrix where the cartilage had been subjected to load. Ten days later, increases in fibronectin accumulation and water content, markers of early osteoarthritis, were evident in the damaged tissue.

Further studies are needed to determine the relationship of our findings about TGF-β1 and high-impact loading to the initiation of the osteoarthritic process in active joints. We will also continue to explore the potential of non-invasive technology such as magnetic resonance imaging, both for use in research and for early diagnosis of osteoarthritis in dogs and other animals.

—Nancy Burton-Wurster
Almost all of the cells in an animal have the same DNA, the nucleic acid that encodes hereditary information for normal growth, differentiation, and function. The unique biological characteristics of the different tissues in the body are set by the particular combinations of genes that are “switched on,” or expressed, in the cells of those tissues.

When a pattern of gene expression changes, it alters the biology of the tissue. Many changes in gene expression are normal and necessary, but abnormal changes may result in the onset of disease. The way that unique patterns of gene expression are regulated, both in health and disease, remains a central question for biomedical research.

Gene expression is often regulated at the level of transcription. This step takes place in the cell nucleus, where the “switched on” portions of the DNA template are copied, or synthesized, as RNA. After transcription, messenger RNA is transported to the cell’s cytoplasm, where its gene-specific instructions are translated into proteins. Our laboratory is looking at changes in transcription that take place following joint inflammation and corticosteroid therapy in racehorses in order to gain insight into the disease process that leads to cartilage degeneration and osteoarthritis.

TRAUMATIC JOINT DISEASE

The physical exertion and musculoskeletal stress of racing predispose horses to traumatic joint disease. The initial pathology often develops in the synovial membrane, a thin layer of cells lining the joint capsule. The specific molecular mechanisms by which synovial inflammation progresses to cartilage degeneration are not well understood. There is also continued controversy over the value and safety of treating joint inflammation in horses with corticosteroids. We are studying four specific cartilage proteins—type II collagen, aggrecan, link protein, and fibronectin—to determine if their expression is changed during the acute phase of synovitis or as a result of corticosteroid therapy.

We have recently demonstrated that both synovitis and the injection of corticosteroids into the joint induce rapid changes in the expression of some cartilage matrix proteins, most notably type II collagen, a protein that is very important for the molecular structure and tensile strength of cartilage. Synovitis increases type II collagen expression, while corticosteroids decrease it. These results provide strong additional evidence that very early transcriptional events play a critical role in the development of degenerative arthritis.
CANINE ERYTHROPOIETIN

The transcription of a gene can also be regulated in tissue or bacterial cultures by using recombinant DNA techniques to synthesize large amounts of a specific protein for therapeutic purposes. We are using this approach to produce canine erythropoietin, a hormone that should greatly improve a veterinarian's ability to treat anemia in dogs suffering from chronic kidney failure.

Anemia is a frequent secondary effect of chronic renal failure and contributes significantly to the clinical signs of lethargy, weakness, and poor appetite. The failure of the bone marrow to replace red blood cells is primarily due to a loss of functional kidney tissue and a drop in the production of a hormone called erythropoietin (EPO). To treat this problem in people, physicians use human EPO synthesized in tissue culture using recombinant DNA technology. The therapeutic application of human EPO in dogs works well initially, but then often stimulates the production of antibodies that recognize human EPO as a foreign protein and mount an immune response.

The antibodies not only block human EPO's activity, but also have the potential to cross-react with any residual EPO still made by the dog, leading to a life-threatening disease called erythroid hypoplasia. This problem severely limits the therapeutic potential of human EPO for veterinary applications.

Nevertheless, the concept of EPO replacement therapy is sound. We would expect synthesized canine EPO to be as safe and effective for use in dogs as human EPO has been for use in people.

To this end, we have isolated the gene encoding canine EPO and can now express this protein in a highly purified form in the laboratory. Our canine EPO preparation works well in laboratory tests, and we are now conducting safety and efficacy trials in dogs in collaboration with John Randolph, a veterinary clinician at Cornell. The availability of canine EPO should provide veterinarians and dog owners with a valuable therapeutic means to improve the quality of life for dogs suffering from the anemia of chronic renal failure.

—James N. MacLeod
T HAS LONG BEEN RECOGNIZED that immune responses can be categorized according to whether they are antibody-based or cell-mediated. Each type of response functions in a distinct way and acts against different types of infectious agents. The type of immune response produced depends on the type of immune system hormones, called cytokines, that are secreted by T helper (Th) cells.

If these Th cells secrete large amounts of interferon γ and IL-2, among others, they are called Th1 cells and produce the cell-mediated form of immunity. If the helper cells secrete IL-4 and IL-10, however, they are categorized as Th2 cells. Th2 cells work principally by promoting antibody responses, particularly involving immunoglobulin E (IgE), a specialized antibody.

In general, Th1 responses are thought to be desirable when intracellular parasites such as viruses and some protozoa infect animals. On the other hand, there is convincing evidence that Th2 cells produce responses that are particularly effective against large parasites, such as gastrointestinal helminths. Every nematode infection that has been studied so far has shown an important or essential role for Th2 cells. However, all of the infections studied have been intestinal and have been examined virtually exclusively in mice, with a mere handful of studies being conducted in rats and none in any other species. While we have already shown in this laboratory that rats mount a protective Th2 response to *Trichinella spiralis*, an intestinal nematode, we also knew that there was some evidence that *Nippostrongylus brasiliensis*, another intestinal nematode, was rejected by other mechanisms. Furthermore, there has been little analysis of immunity to helminths that live outside the intestine, such as canine heartworm and other filaria. We approached this issue by beginning an analysis of immunity to *N. brasiliensis*, and we also began experiments with *Brugia pahangi*, a filarial nematode that naturally infects cats and rodents.

We have based our approach on our previous work with *T. spiralis*, comparing immunity in a variety of rat strains to infections with each of these different parasites. We anticipated that some rat strains would prove unable to kill the parasites very effectively while others would be more effective. Comparison of their underlying immune responses would help us identify the functional elements of immunity most important in parasite killing. We have succeeded in defining two rat strains that vary widely in their ability to eliminate *N. brasiliensis* or *T. spiralis* parasites. One strain, designated LOU, mounts a highly effective immune response, while the other,
designated WKA, responds relatively weakly to infection with these parasites. Other rat strains exhibit immune responses that lie between these extremes, suggesting that there may be several genes that influence the strength of the host response. One strain surprised us, however. Lewis rats have been as effective as LOU rats in killing *T. spiralis* but as ineffective as WKA rats in killing *N. brasiliensis*. In other words, the immunity that this rat strain displays varies according to the parasite strain that infects it. Also like the WKA strain, Lewis rats respond weakly to *B. pahangi* infections. This interesting finding suggests an unexpected diversity in parasite-killing mechanisms. We are currently examining various immune response parameters in an attempt to better define those that are most effective in controlling these infections.

The existence of a variety of response patterns in rats correlates well with known patterns of infection in humans and other animals. The use of rat strains to examine the biological basis for these differences in host response may help produce better vaccines, or farm and companion animals that are genetically resistant to parasites. Further analysis of the cellular and molecular bases for the differences between these rat strains will be a significant area of research over the next several years.

—Robin G. Bell
THE RELATIONSHIP BETWEEN a parasitic organism and its host is intricate and must be carefully balanced. A parasite must acquire physical and nutritional support from its host without damaging or enraging it to the point that the reproduction of the parasite is jeopardized.

*Trichinella spiralis* is a parasitic worm that has adapted to its host in such a way that the damage it does during any one of its life stages does not induce a host response strong enough to affect its survival in the next stage. In this way the parasite stays one jump ahead of the host response and ensures its own successful reproduction and survival.

The response of the host to the parasite includes the production of antibodies against molecules synthesized by the parasite. Phil Peters, an undergraduate honors student, discovered that responses may offer a new means of therapy or parasite control.

Interfering with the processes by which *Trichinella spiralis* controls host responses may offer a new means of therapy or parasite control.

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Despite the fact that both of these sugars stimulate strong host responses, the parasite controls very carefully the times at which it synthesizes and releases the sugars so that it avoids destruction and is able to reproduce. The means by which the parasite regulates these events is of considerable biological and practical interest. Interfering with these processes may offer a new means of therapy or parasite control.

—Judith A. Appleton
Above left: Judy Appleton

Above right: Postulated location of T. spiralis in the small intestine. The parasite occupies epithelial cells at the level of the crypt-villus junction. Exposure of worm at sites between crypts (as shown) has not been documented. (Illustration by Lucy Gagliardo derived from Appleton and Oblak, unpublished observations and Dunn and Wright31).

Left: Phil Peters and Lucy Gagliardo
FOR NEARLY TWO DECADES much of the scientific effort of the Equine Genetics Center has focused on the relationship between pregnancy and the immune defense system. In this relationship the challenge for mothers, whether equine, canine, or human, is to learn to live compatibly with the fetuses developing in their wombs.

The problem for the fetus is how to avoid detection and destruction by the mother's immune system.

During the course of evolution mammals have developed sophisticated defense and recognition systems to identify and reject foreign substances within the body. These defense mechanisms are known collectively as the immune system. To the immune system, however, 'foreign' includes not only the vast array of bacteria, viruses, and parasites that share our environment, but cells, tissues, and organs from other members of the same species. The immune system's tendency to reject and destroy foreign tissue greatly complicates organ transplantation, but seems not to prevent the majority of pregnancies from reaching a successful conclusion. What accounts for the difference?

On the fetal side, the placenta has developed mechanisms for controlling the expression of the cell surface proteins that are the main targets of the immune system in the graft rejection process. These major histocompatibility antigens (major, for their importance in graft rejection, histo-, meaning tissue, and antigen, meaning foreign substance) are expressed on virtually all cells of the body. The placenta, however, switches off the genes that produce the histocompatibility antigens in the cells of its outer layer. As a result the mother's immune system fails to detect these foreign molecules on the placental surface, and therefore ignores the presence of the fetus. Inside the placenta and in the fetus, the histocompatibility antigens are expressed normally. This is a wonderful example of biological modification on the part of the fetus.

End of story? Not exactly. For reasons that are not well understood, in most species a small, but important, subset of placental cells takes the dangerous course of exposing foreign histocompatibility antigens to the maternal environment. In the horse, these cells induce a strong maternal antibody response against the developing equine fetus. For many years our laboratory has studied this immune response and the expression of histocompatibility antigens in the placenta of the horse. Our current efforts are designed to identify the genetic mechanisms that regulate this antigen expression.

Why doesn't the horse's maternal immune response destroy those placental cells that express histocompatibility antigens? In 1995 we began new studies of the reactivity during pregnancy of cytotoxic, or cell-killing, lymphocytes. We found an
unexpected and exciting result. The ability of a pregnant mare to produce a cytotoxic lymphocyte response against the foreign histocompatibility antigens of the mating stallion was greatly reduced during pregnancy, but not in the non-pregnant state. In a non-pregnant mare, a foreign skin graft results in the production of both antibodies and cytotoxic lymphocytes, and the graft is promptly rejected. During pregnancy, however, it appears that the cytotoxic lymphocyte response to the fetal “graft” is impaired, while the antibody response is normal. Could this be a clue to the accommodation that all mothers make during pregnancy?

The questions that remain are relevant not only to pregnancy and transplantation, but to protection against infectious diseases as well. For example, what effect might this pregnancy-induced alteration of immune reactivity have on the ability of a mare to respond to vaccination or to infection? The staff of the Equine Genetics Center will continue to dedicate its efforts and excellent resources to this important line of investigation.

—D. F. Antczak
X SEX REVERSAL, an inherited disorder that is common in several breeds of dogs, is an interesting puzzle. Despite having the chromosomes of females (78, XX), dogs affected with XX sex reversal develop testes, as if they were males, or ovotestes, which are a combination of both ovarian and testicular tissues that develops together as one organ.

We are examining the genes that normally control testicular development to understand how testis tissue can develop in animals with female sex chromosomes. Our goal is to identify the gene that is responsible for this disorder and then develop a DNA test that will detect both affected dogs and carriers easily and early in life.

Our studies have focused on the mode of inheritance of XX sex reversal in the American cocker spaniel and the German shorthaired pointer, two breeds with a relatively high incidence of the disorder. XX sex reversal has also been reported as a common problem in English cocker spaniels, weimaraners, beagles, Kerry blue terriers, and Chinese pugs, and has been diagnosed infrequently in other breeds, such as the soft-coated wheaten terrier and the basset hound. 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.

During the past year we 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 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.

Since the canine Sry gene had not yet been identified, we began by cloning and sequencing the part of the canine gene called the Sry HMG box. This is the “business end” of the Sry gene, as it allows Sry to turn on genes involved in testis organization. Institute colleagues Greg Acland and Ben Hershfield collaborated with Vicky Palmer and me on this project. After identifying the canine Sry HMG box
sequence, we placed it in GenBank, a computer registry of gene sequences, to make it available to other researchers.

Once we knew the sequence for the Sry HMG box, we were able to look for it in the DNA of affected American cocker spaniels. With the help of two veterinary students taking part in the Summer Leadership Program, Virginia Fajt of Auburn University and Larissa Bowman of North Carolina State University, we also looked for the Sry gene in affected German shorthaired pointers. We did not find the suspect gene in affected individuals of either breed. Since we had previously shown through breeding studies that the disorder is inherited as an autosomal recessive trait in American cocker spaniels, we have now ruled out the possibility that the gene mutation responsible for XX sex reversal could be located on the Y chromosome. In German shorthaired pointers, the mode of inheritance of this disorder has not been firmly established, but it may also prove to be an autosomal recessive disorder.

— Vicki N. Meyers-Wallen
MUCOPOLYSACCHARIDOSIS (MPS) is a descriptive term for a group of inherited lysosomal disorders that cause mild to severe clinical manifestations in dogs, cats, mice, rats, and humans. The MPS group of diseases are caused by deficiencies of various lysosomal enzymes needed to degrade glycosaminoglycans, which are residues of cell membranes and extracellular matrices.

These residues are normally present in all the tissues of the body. In severe forms of MPS, undegraded glycosaminoglycans accumulate in the lysosomal compartments of cells, ultimately resulting in liver enlargement, bone deformities, mental retardation, blindness, and early death.

While MPS is a system-wide disease, our research is concentrated on two of its effects, retinal dysfunction and abnormal bone development. Our studies focus on three forms, MPS VI, MPS VII, and MPS I, each of which is due to a different enzyme deficiency. Our goal is to discover the molecular defects that alter the biochemical functions underlying normal retinal and skeletal development.

We are also engaged in developing strategies for treating or curing these inherited disorders through gene therapy.

Our work this year with MPS VI illustrates the approach we are taking in studying this group of diseases. In MPS VI as in other forms of MPS, the storage of undegraded glycosaminoglycans causes severe enlargement, or hypertrophy, of the retinal pigment epithelium of the eye. In the case of MPS VI, this accumulation is due to a deficiency of arylsulfatase B (ASB). In studies to determine the pathology and biochemical properties of the disease in cats, we have observed that the level of the ASB enzyme in diseased retinal tissue falls to five-to-fifteen percent of normal, causing the buildup of dermatan sulfate, a glycosaminoglycan.

Our attempts to treat diseased retinal pigment epithelium in laboratory culture have been very encouraging. We inserted normal copies of the gene for the deficient enzyme into a harmless viral vector, then infected the retinal cells with the vector. In the infected cells production of the necessary enzyme increased, causing a reduction in the level of dermatan sulfate stored in the lysosomal compartments. These experiments are preliminary, but our success encourages us to believe that we will someday be able to treat the retinal manifestations of MPS in animals and humans. In addition to the contributions of research associate Yanggeng Wu and postdoctoral fellow Maria Verdugo, the gene therapy project is being done in collaboration with Mark Haskins of the University of Pennsylvania, J. M. Heard of the...
Pasteur Institute, and Gustavo Aguirre of the Baker Institute.

Dr. Wu and I also began in vitro studies of cartilage in order to understand the MPS VI disease process as it relates to skeletal abnormalities. Dr. Wu successfully established cell cultures of normal and diseased cartilage, and we have seen that the cartilage cells show a biochemical and cellular pathology similar to that observed in the retinal pigment epithelium: increased storage of glycosaminoglycans in the diseased cells with a remarkable decrease of enzyme activity. Our work to characterize this disease continues.

In our work on MPS VII in the dog, we have so far determined 70 percent of the nucleic acid sequence of β-glucuronidase (GUSB), the enzyme that is deficient in diseased animals. With the help of Wei Sun, a graduate student from China, we have compared the sequence in the DNA of normal and diseased dogs in an effort to detect any mutations. We are continuing to analyze the remaining portion of the canine GUSB sequence for mutations.

— Jharna Ray
PROGRESSIVE RETINAL ATROPHY (PRA) is one of the leading causes of blindness in dogs. At the time that Gustavo Aguirre began studying the disease at the Baker Institute in the early 1970s, the mechanisms that caused this incurable hereditary condition were unknown, and PRA was thought to be one, or possibly two, diseases.

In the intervening years we have found that PRA is a group of seven related but separately inherited diseases, all of them characterized by malformation or degeneration of the retinal visual cells. Our group has pioneered the way in establishing the genetic basis of all of these retinal disorders, which, taken together, are suspected to occur in over 80 different breeds of dogs. In some affected breeds, vision loss can be observed in puppies, and dogs may become blind before or soon after maturity. In other breeds, however, PRA can go undetected until the dog is several years old.

A DIAGNOSTIC BREAKTHROUGH
During the past two years we were successful in our efforts to develop a blood-based DNA test to identify carriers of rod-cone dysplasia 1, a form of PRA inherited in Irish setters. In this work, we were aided by the expert technical assistance of Vicki Baldwin and Sue Pearce-Kelling.

The new blood test can determine rcd-1 status shortly after birth and before the onset of symptoms. Most importantly, it is now possible for breeders to distinguish carriers, which carry one copy of the defective gene, from normal dogs, which carry two normal genes, without resorting to test breeding to identify and remove all carriers and affected dogs from their lines. Additionally, it allows breeders to continue to keep otherwise desirable dogs in their breeding programs, even if they are affected with rcd-1. An affected dog bred to a genetically normal dog will produce clinically normal progeny. In other words, although these offspring will carry one copy of the defective gene, they will not be affected by the disease. Breeders can now phase out the undesirable gene through informed matings without otherwise diminishing the genetic diversity of their lines. Accordingly, the Irish Setter Club of America is now recommending that all breeding setters be tested, and that data on PRA status be included in all AKC pedigrees.

The new DNA test identifies only rcd-1, the form of PRA specific to Irish setters. However, the development of the rcd-1 blood test demonstrates the potential for all forms of PRA, and indeed many other inherited diseases, to be found through DNA screening. Our success with rcd-1 has allowed us to step up our efforts to identify the gene defects responsible for several other forms of PRA; we hope to
make other tests available within the next few years. Our efforts are now concentrated on rod-cone dysplasia 2 in collies, retinal dysplasia in the miniature schnauzer, and progressive rod-cone degeneration. The latter is an especially widespread, late-onset form of PRA that is known to affect poodles, Labrador retrievers, English and American cocker spaniels, and Portuguese water dogs and is suspected to occur in as many as sixty other breeds. Our efforts to discover the gene defect of collies have been enhanced this year by the work of Weiquan Wang, an M.D. who has joined our laboratory as a graduate research assistant. Our other research interests include rod dysplasia and early retinal degeneration, both of which occur in Norwegian elkhounds, the X-linked PRA of Siberian huskies, and the cone degeneration that strikes Alaskan malamutes. These are very exciting times for our group, and we look forward to future reports where we can detail the progress and success of our endeavors.
—Gustavo Aguirre, Kunal Ray, and Gregory Acland
CANINE GENOME MAPPING

To the geneticist, dog populations in some respects resemble human populations. Like humans, dogs are afflicted with numerous genetic disorders such as hip dysplasia and osteoarthritis, muscular dystrophies, cancers, and progressive retinal degenerations.

Further, the incidence of these disorders varies in different breeds, much as the incidence of genetic disorders in man varies among different ethnic groups.

But while rapid progress is being made in mapping the human genome, genetic studies in the dog have been hampered by the difficult characteristics of canine chromosomes. Dogs have a large number of chromosomes, all of which, with the exception of the sex chromosomes, are relatively small and strongly resemble each other. There is a special and pressing need for studies on canine chromosomal mapping and the genetic relationship of dog breeds to one another.

Fluorescence in situ hybridization (FISH) is an extremely sensitive, rapid, and reproducible technique for marking gene locations on chromosomes. Through a further refinement of this technique, it is now possible to produce two- and three-color signals wherein each color represents a different probe. This allows rapid physical ordering of genes on the chromosomes and is a major advance over previous physical mapping methods. In collaboration with Dan Goldowitz of the University of Tennessee, I am using FISH in two areas: chromosome painting with repetitive probes, and demarcation of regions of canine chromosomes that have remained relatively unrearranged in the course of evolution, a state known as synteny.

In the painting experiments, DNA probes cloned from dogs are hybridized to canine chromosome spreads. We have found one particular probe that produces a hybridization pattern that unequivocally distinguishes specific pairs of canine chromosomes. In the synteny experiments, canine clones of sequences that are evolutionarily conserved among other species are mapped to the already defined, corresponding chromosomal regions in man or mouse. These clones are then hybridized to canine chromosomes for comparison. Simultaneous application of two probes known to hybridize to contiguous sites in the human or mouse genome can be used to determine if the same relative positioning exists in the canine chromosomes. If so, the synteny is preserved. Knowledge of syntenic relationships forms the basis for correlating the genetic map of the dog with the genetic map of humans.

To assess the level of genetic diversity within and between dog breeds, I have used DNA fingerprinting as a predictive measure. This technique identifies complex, individual-specific hybridization patterns that are inherited in a Mendelian fashion. I have found one probe that identifies more than 15 polymorphisms in a single study pedigree.

As an extension of this work, I have found that the same probe can determine which vertebrate species has contributed DNA to a given biological sample. When PCR was performed on DNA samples using the probe as a single primer, I noted
discrete patterns that were invariant in size among members of a single species while varying markedly between species. Each vertebrate species tested had a unique PCR pattern which was invariant within the given species. This invariance held true even for different human races, while the band pattern could appear markedly different between even such closely related species as chimpanzee, gorilla, and man.

Although it is possible that divergence in the banding patterns occurs before breeding barriers develop between nascent species, it seems more likely that the divergence in banding patterns occurs soon after species formation and represents rapid mutational change in the amplified sequences. If this is true, then populations undergoing genetic divergence, such as different breeds of dogs, should show differences in sequence composition. Thus, these sequences may be used to trace the ancestry and relatedness of the different dog breeds. I have cloned the amplification products to begin addressing these questions.

—Bennett G. Hershfield
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Joseph Ferris, DVM
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Mary E. and Homer W. Flippen
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George W. Frey (in memory of Brandywine of Morgan Farms)
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Mr. and Mrs. Douglas P. Fritz (in memory of Prudence, Oscar, and Maggie)
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Joseph E. Genewich (in memory of Sam)
H. William Ghriskey (in memory of Doris Beckham Fletcher)
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Dolores Bona
Anna Bonchick (in memory of Sparkle)
Nancy Stephenson Bond
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Mr. and Mrs. Bill Bryn (in memory of Katheryn Torda)
Mr. and Mrs. Robert M. Burns (in memory of Fritz)
Daniel E. Button (in memory of Gordie)
Mr. and Mrs. William C. Byrnes (in memory of Benji)
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Elinor M. Call (in memory of Gingerbread)
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Helen and Carol M. Canora (in memory of "Rusty", Ch. Charlieshope Ever So Easy)
Barbara J. Canter
Joseph E. Capuzzo
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Rachel Carren (in memory of Oscar)
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Beverly R. Young
Ways of Giving

The Cornell University Board of Trustees, in establishing the Baker Institute, authorized the Treasurer’s Office of Cornell to be custodian of all funds given in support of the Institute.

You are thus assured that your gift will have the maximum benefit. There are many ways you can give to advance the work of the Baker Institute. Some of these opportunities offer substantial income tax and estate tax benefits.

Checks All checks may be made payable to the Baker Institute and mailed to:

Office of the Director
James A. Baker Institute for Animal Health
College of Veterinary Medicine
Cornell University
Ithaca, New York 14853

Appreciated Stocks Selling appreciated stocks is almost certain to increase your taxes, but if you give the stocks to Cornell outright and deduct their full current market value as a charitable contribution, you can probably avoid all or most of the capital gains tax. To complete the transaction with maximum speed and at lowest cost:

1. take the certificate to your bank or broker;
2. inform your bank or broker that you want to make a gift of the shares or securities to Cornell University for the Baker Institute;
3. instruct your bank or broker to telephone the Office of Trusts and Estates at 607 277-0025; and
4. write a note to the Office of the Director, James A. Baker Institute for Animal Health, College of Veterinary Medicine, Cornell University, Ithaca, New York 14853, including the name of your bank or broker and the form and size of your gift.

Depreciated Stocks You receive the maximum benefit from a gift of depreciated stocks by selling the shares and then giving the proceeds to Cornell. In that way you may take advantage of the capital loss allowance and a charitable contribution deduction for the amount of the gift. Instruct your bank or broker to sell the shares or securities and send the proceeds as a gift to Cornell for the James A. Baker Institute for Animal Health.

Bequest Charitable bequests provide substantial estate tax benefits. They can be gifts of land or buildings, securities, personal property, or cash. Cornell University counsel suggests the following provision: I hereby give, devise, and bequeath [description of property] to Cornell University, an educational corporation located at Ithaca, New York, for the uses and purposes of the James A. Baker Institute for Animal Health.

Deferred Giving An income-producing trust enables you to make a gift to the Baker Institute, gain income for life, and derive tax benefits. A beneficiary may be named to receive the income, too. Cornell University offers three plans: the Pooled Life Income Fund, the Annuity Trust, and the Unitrust. You may also establish a Charitable Lead Trust, which will first pay income to Cornell and later transfer assets back to you or to a third party. Financial planning involving deferred gifts requires expert advice from your attorney and other specialists. If you are interested in deferred giving, please notify the Office of the Director, and we will make arrangements for you to receive more information.
Office of the Director
James A. Baker Institute
for Animal Health
College of Veterinary Medicine
Cornell University
Ithaca, New York 14853

☐ I enclose my check for $ ___________, payable to the Baker Institute.

☐ Send me periodic reports on the progress of Baker Institute research. I am especially interested in information on:
   ☐ canine parvovirus
   ☐ canine Lyme disease
   ☐ canine brucellosis
   ☐ progressive retinal atrophy
   ☐ hip dysplasia and osteoarthritis
   ☐ other ______________________________

☐ Send me information on how I can help canine health research and the Baker Institute in my will.

_________________________________________________________

name

________________________________________________________

street address

________________________________________________________

city state zip

Contributions are tax deductible.