Cover photo by Dede Hatch of her dog Ruby
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A Message from the Director

The Baker Institute celebrated its 40th anniversary this year. A milestone of that significance calls for reflection and renewed commitment. Founded on hope and enthusiasm, the Institute was given a specific mission: to improve the health and quality of life for animals through research aimed at finding the causes of infectious diseases and developing practical strategies for their diagnosis, treatment, and control. Much of the initial work targeted diseases of cattle and swine, although dogs and horses also benefited from research undertaken in the 1950s and 1960s. Prominent early accomplishments included the development of vaccines for the prevention of bovine viral diarrhea, hog cholera, and canine infectious hepatitis.

The Cornell Research Laboratory for Diseases of Dogs, which was established as a section of the Institute during its first year, has set down a history of remarkable achievement. Among the Laboratory’s many accomplishments have been the identification of infectious agents causing hepatitis, enteritis, reproductive failure and puppy losses, the development of accurate diagnostic tests for those diseases, and the development of vaccines for the prevention of hepatitis, parvovirus, distemper, and kennel cough.

The Institute’s association with the Veterinary College at Cornell created an environment where research could be coupled with scholarly activities and teaching. Institute faculty participate in the teaching programs of the College and have in addition provided advanced training for nearly 200 health professionals during the past 40 years. Institute staff have published their research findings in more than 800 scientific papers and organized conferences to discuss health problems of dogs, cattle, swine, and horses. Institute trainees often achieve distinction in their own right. Our alumni include the president of a major university, six deans of veterinary medicine, and many individuals who occupy leadership positions in academic institutions, government, and industry.

From a modest beginning, the Institute has grown into a modern, well-equipped research enterprise. During the past decade alone, the Institute’s facilities have doubled in size; the original buildings have been refurbished or expanded, and an impressive array of scientific instruments has been acquired. These improvements have fostered research at the cutting edge of veterinary science and provide a solid foundation for further growth.

A major decision was made this year that will benefit our canine programs while simultaneously broadening the research undertaken in Professor Antczak’s laboratory. Our parent institution, the College of Veterinary Medicine, has given high priority to a basic research initiative that will explore processes associated with early development of the embryo. The Institute will have a major role. Research conducted at the Institute will make use of recent advances in biotechnology to identify, manipulate, and transfer genes that govern the expression of important phenotypic and functional traits. Besides adding to our basic knowledge of life processes, these efforts also will have practical benefits, including improvements in the reproductive efficiency of animals, a deeper understanding of the genetic basis of diseases, and, ultimately, the capacity to improve the quality of life by rendering animals more resistant to disease.

Since its founding 40 years ago, the Institute has had only two directors. Continuity has its advantages, but it is not the preferred arrangement in a rapidly changing research environment where success depends on flexibility and innovation. In mid-1991 I will step down as Institute director. I do so secure in the knowledge that we are now financially strong, that our scientific programs are prospering, and that a consensus has been forged on the Institute’s future course. It would be premature to define that course precisely, for it will be determined in large measure by the new director. Nevertheless, there is agreement on two major issues: first, the Institute will retain its species-oriented focus on the dog; and second, it will build on its current strengths in cell and molecular biology. These decisions combined with a renewed commitment to excellence augur well for the future.

The goals before us are ambitious, but so too were the challenges that confronted the Institute 40 years ago. An institution that aspires to leadership and excellence must be willing to move in new directions and to probe the unknown. The challenge is to do so ever conscious of our mission, our earlier achievements, and also our corporate and individual responsibilities to society and to the countless numbers of men and women whose efforts, generosity, and encouragement have enabled the Institute to grow and prosper. The Institute is richly endowed with a talented staff and an Advisory Council of distinguished scientists and community leaders. All are determined that the Institute will continue to operate at the forefront of veterinary medical research. These are remarkable assets for any institution, and they will ensure the Institute’s success in the years ahead.
As part of the celebration of our 40th anniversary, we had the pleasure this year of honoring our beloved friend Dorothy Donnelley with the Arthur F. North, Jr. Canine Service Award. An Institute founder, Dorothy served seven years as chairwoman of the Advisory Council. She stands out even among her exemplary peers as an exceptional advocate of animal health and welfare. Our recognition of her numberless contributions to the progress of Institute research is long overdue.

The support and endorsement of clinical practitioners has always been critical to the success of this institution. This year’s anniversary celebration provided a fitting occasion on which to present the first annual Founders’ Award to Charles E. Fletcher, D.V.M., who is himself a founder of the Institute and a distinguished Cornell alumnus. The award recognizes his outstanding contributions to the veterinary profession through enlightened practice and the advancement of Institute research.

Two veteran staff members retired from the Institute this year. Jean Joubert, a research technician, began work in Leland Carmichael’s laboratory in 1968. She made lasting contributions to the Institute and to canine health as a member of the task force formed in 1978 to study canine parvovirus. Florence Huth came to the Institute in 1971 and worked for many years as Dr. Carmichael’s secretary. In that role, she was responsible for the in memoriam program and made many friends with dog owners and our veterinary contributors. We shall remember Florence and Jean with affection and gratitude for their combined service of over 41 years.

William Rockefeller, the chairman of our Advisory Council and a true friend, died earlier this year. He inspired us for many years with his commitment to animal welfare and his staunch support of our educational programs, and we miss him very much. Dr. Robert E. Shope, a founding member of the Advisory Council, has succeeded Bill as chairman. A distinguished scientist and administrator, Bob has had a major role in guiding the development of the Institute since 1977. His insight and counsel will ensure a steady course during the present period of transition.

Other changes to the Advisory Council took place during the past year. Another trusted friend and advisor, Robert Winthrop, retired from the Council after eight years of invaluable service to the Institute. We are extremely grateful to have received the benefit of his good counsel. Bob’s nephew Robert Winthrop II, a veteran member of the Council, was joined this year by two new second-generation members—Strachan Donnelley, son of Dorothy and Gaylord Donnelley, and Sarah R. Bogdanovitch, Bill and Molly Rockefeller’s daughter. As the renewal of the Council illustrates, change is a necessary element of successful continuity.
Minute Virus of Canines  
(Canine Parovirus-1)

Minute virus of canines (MVC) was isolated in 1967, but no disease role in dogs was discovered until recently. Three years ago we recovered this virus from intestinal contents collected from field cases of dead pups. Subsequent serological evidence suggested that MVC might have a role in early embryonic deaths, or fetal resorptions. Among the causes of canine reproductive failure, only brucellosis, bacterial infections of the uterus, and canine herpesvirus have so far been identified. We have therefore continued to focus on the effects of this virus on the developing fetus.

We have now established experimentally that MVC is capable of causing transplacental infections. These seem to occur principally in dams exposed to the virus prior to the 35th day of pregnancy. Pregnant females inoculated intravenously with MVC between gestational days 25 and 35 all resorbed their embryos within ten days following inoculation. However, infections occurring later in gestation only rarely resulted in fetal deaths. Direct exposures of fetuses to the virus via the intra-amniotic sac resulted in fetal deaths when inoculations were done prior to gestational day 50; after that time the only indications of infection were active immune responses in the inoculated fetuses. On the other hand, oral-nasal exposures of dams to MVC gave variable results. Fewer than half of the exposed dams had abnormal fetuses. At birth those pups were weak or dead; some suffered from hydroptic fetais.

As in the field cases studied, fetal infections were characterized by extensive infection of the lung and intestinal tract. Heart and lymphatic...
tissues were also sites of viral disease in some fetuses. We were interested to note that bitches that had carried infected fetuses had higher levels of antibody to MVC than did infected dams that had normal fetuses or pups. MVC could be isolated from fetal tissues only at the time of death, or prior to the appearance of MVC antibodies in fetal circulation. It would therefore be difficult to diagnose this virus in field cases by current methods. More-sensitive nucleic acid hybridization methods are now being developed for viral diagnosis and to determine the prevalence of MVC as a cause of natural reproductive disease.

**Canine Parvovirus Vaccines**

On another front, we have continued to examine various commercial vaccines for their capacity to immunize dogs against canine parvovirus Type 2 (CPV-2), especially newer products that claim exceptional efficacy. The recent introduction of a CPV vaccine containing the most prevalent virus type (CPV-2a, which was originally characterized by Colin Parrish) has prompted many more veterinarians and breeders to ask whether CPV-2 vaccines that contain only one strain protect against all types. Results have been unequivocal—all vaccines tested, regardless of the CPV strain incorporated into the vaccine and regardless of whether the vaccine contained living or inactivated virus, protected dogs against challenge with virulent CPV-2a. Advertising claims that certain vaccines “break through” maternal antibody levels and immunize pups at the time when they first become susceptible to infection are false. We and others have substantiated that pups become susceptible to infection with virulent CPV some two-to-five weeks before they can be successfully vaccinated. Vaccines rarely alter the course of infection in kennels once an outbreak has occurred. Recognition of that fact is important, since prevention of parvovirus infection in young pups depends mainly on management—only the strictest isolation possible and rigorous attention to hygiene will prevent transmission of this exceptionally stable parvovirus. The function of a vaccine is to protect susceptible animals prior to exposure, and most current vaccines do that very well. A recent brochure from the Institute that discusses canine parvovirus vaccination “myths and realities” is available to veterinarians and breeders on request.

**CONFRONTING AN UNKNOWN ENEMY: CANINE PARVOVIRUS**

Brian Summers, a Cornell pathologist, was one of the first to telephone Institute virologist Max Appel in August of 1978 to report finding a panleukopenia-like enteritis where he had never seen it before—in a dog. Within a week of that and other reports, the Institute had formed a task force to study the new canine disease, which was to erupt on four continents within one year. The remarkable speed and skill with which those investigators moved to quell the emerging catastrophe spared untold thousands of dogs and gave the Institute perhaps its greatest success story to date.

Electron microscopy of fecal specimens submitted to the Institute in August of 1978 soon began to reveal a very small parvo-like virus. In September Dr. Appel isolated the virus for the first time after propagating it in kidney cell culture. Fred Scott of Cornell’s Feline Health Center repeated that success within a few days. After serological comparison established its difference from “minute virus of canines” (CPV-1), the previously unknown virus received tentative designation as canine parvovirus Type 2. Cultures of CPV-2 stained with fluorescent antibody reagents prepared against feline panleukopenia virus (FPV) revealed strong cross-reactivity.

That was an important finding. It suggested a possible short-term vaccination strategy, using live attenuated feline panleukopenia virus to protect dogs against CPV-2. Once he had successfully reproduced clinical disease in the laboratory, Dr. Appel made rapid progress. By the end of December, he was able to demonstrate that the heterotypic vaccination concept worked, provided that high enough virus titers could be reached. However, commercial FPV vaccines only contain sufficient concentrations of virus to immunize 70-80% of all dogs.

The second approach successfully demonstrated during the same period was experimental vaccination with killed CPV-2. The effectiveness of that method was also limited. The killed vaccine protected dogs from disease for several months, but not from infection. Dogs that showed no signs of illness could still shed virus and infect other dogs. Nevertheless, the two vaccines submitted for patenting in January, 1979 are credited with saving countless lives.

Leland Carmichael of the Institute ultimately produced the first successful attenuated live CPV-2 vaccine. Attenuation of the new pathogen, which was begun in the fall of 1978, took nearly two years and 100 passages in tissue culture to complete. Canine parvovirus infection reached panzootic proportions during that time.

Ten years later, the attenuated live vaccine that Dr. Carmichael perfected is still the dominant vaccine used in the field. It is perhaps surprising to realize that most of the canine parvovirus vaccines in use today were developed at the Institute before there had been time to learn very much about the disease itself. The task force’s intensive efforts to define the clinical disease and determine its pathogenesis and means of transmission continued well into the 1980s.
Lyme Disease

Our efforts this year have concentrated on Lyme disease in dogs. Lyme disease has received extensive media coverage in the last decade. It was recognized as a new disease entity in humans on this continent in 1975, when an unexplained cluster of cases of joint disease resembling juvenile rheumatoid arthritis suddenly appeared in children in Lyme, Connecticut. The deer tick, *Ixodes dammini*, was found to be the vector of the pathogen, *Borrelia burgdorferi*, a spirochete related to *Leptospira* and *Treponema* (the causative agent of syphilis).

Since first being observed in humans, the disease has been seen at an increasing rate in dogs in the northeastern United States, in the Midwest, and in California. In contrast to human Lyme disease, which usually appears in three different stages, the canine disease most often appears to be acute. Affected dogs demonstrate sudden lameness and evidence of pain when touched. Other signs include severe depression, total reluctance to move, fever, and swollen lymph nodes. Treatment with antibiotics usually cures the disease. (For a more comprehensive review of the disease, see Appel: *Compendium on Continuing Education for the Practicing Veterinarian*, Vol. 12(5), pp. 617-626, 1990.)

A vaccine for the prevention of Lyme disease in dogs that was produced by Fort Dodge Laboratories was licensed conditionally in the spring of 1990. Use of the vaccine in the field for a year or more should produce sufficient data for a good evaluation.

We began our study of the pathogenesis of Lyme disease in dogs in collaboration with several other Cornell scientists. In addition to investigators from our laboratory, the team includes
Sandra Allan, Richard Jacobson, Eric Shaw, and Sang Shin. Dr. Allan, an entomologist, established a colony of white-footed mice \((Peromyscus leucopus)\), the carrier of \(B. burgdorferi\), and collected infected ticks from Westchester County, an endemic area near New York City. After exposing pathogen-free dogs to infected ticks as well as to the isolated agent, we began serial attempts to isolate the agent, serological studies, and clinical observations. We have now reproduced the disease in dogs; however, further studies are needed to explore its pathogenesis and to evaluate vaccines.

**Canine Distemper Virus**

Our research efforts with canine distemper virus (CDV) resulted in an unusual finding this year. During the winter months of 1990, Arizona Fish and Game Department representatives found signs of encephalitis in eight adult javelinas \((Tayassu tajacu, a relative of the pig also known as the collared peccary)\). Although tissue tests conducted in Arizona yielded negative results for a variety of agents, histopathology and electron microscopy did indicate the presence of a morbillivirus. Tissues were sent to our laboratory, and we isolated CDV from the brains of three javelinas. Because a CDV-like virus that was different from classical CDV had been isolated from North Sea seals with distemper-like lesions, we were interested in comparing the two viruses. By use of monoclonal antibodies we found the javelina isolates to be identical to CDV.

*Max J. G. Appel*

In 1950 infectious canine hepatitis, distemper, and rabies were the only known viral diseases of dogs. Infectious canine hepatitis, which had been identified only a few years earlier, was already so widespread that nearly one dog in two was infected with it. Mortality in young dogs was especially high. Concerned by its severity, James Baker made it a primary focus of study at the newly created Cornell Research Laboratory for Diseases of Dogs.

Dr. Baker and his colleagues were also interested in distemper virus, and in a phenomenon previously observed only in laboratory mice, that of dual infection. Institute studies had turned up cases where distemper virus was preceded, accompanied, or followed by infectious canine hepatitis. Those studies had found that the incubation period for simultaneous infections was half that for either virus alone, and disease was especially severe in those cases.

Dr. Baker reasoned that, if dual infection was possible, dual protection should also be possible. Laboratory staff took little time to produce the first vaccine for animals that combined two different live viruses for simultaneous protection against two different diseases. Live distemper virus could be attenuated in embryonated eggs, but infectious hepatitis virus, being host specific, could not. Therefore, the earliest vaccine used unattenuated virus combined with antiserum to temper its virulence. Five years later, though, Institute researchers had succeeded in adapting tissue culture methods so that infectious canine hepatitis could be grown and attenuated in pig kidney cells.

The principles of viral attenuation and dual vaccination helped form the basis of the canine disease control program that Dr. Baker and his staff worked for ten years to define. They were exploring largely uncharted territory. In addition to studying the transmission and the pathogenesis of infectious diseases, the Institute set industry standards for vaccine production and diagnostic testing. Immune responses to vaccination were studied in great detail.

Those painstaking analyses yielded a crucial finding that explained many vaccination failures: the role of maternal antibody. It was found that protection conferred upon nursing pups through colostrum and milk interfered with vaccination attempts for periods of time that varied according to the dams’ antibody titers. The Distemper Nomograph, a chart of those precise relationships developed by Douglas Robson and James Gillespie, represented a major step forward. It made it possible to determine the proper age for vaccination before a litter of puppies had even been born. Use of the Distemper Nomograph in field trials of the dual distemper-hepatitis vaccine conducted during 1959 and 1960 produced an unprecedented vaccination success rate of 98.8%.
Although a great deal of effort has been expended on the study of hip dysplasia, both in our laboratory and by other investigators, to date the cause of hip dysplasia has not been identified. We continue to seek answers to the fundamental questions of how and why the joint abnormality occurs. A further goal of our laboratory is the development of an accurate method for diagnosing the disease in young dogs.

Previously we reported on a long-term study designed to assess the possible role of excess capsular laxity in the development of hip dysplasia. We observed that the dysplastic adult dogs studied exhibited greater joint laxity than their dysplasia-free counterparts. At four months of age, many of the pups we examined appeared to have laxity in their hip joints as well. We are re-evaluating those dogs at two years of age in an effort to determine whether the apparent early laxity is predictive of hip dysplasia. Two years is the youngest age at which conventional X-ray diagnosis can be used to identify the presence or absence of hip dysplasia. We expect to reach a conclusion within the next year on the relationship between the laxity measured in pups at four months and their hip joint conformation at two years.

In another but related study we obtained tentative evidence suggesting a link between hip dysplasia and relaxin, a maternal hormone present during pregnancy. In most mammalian species relaxin cannot be detected in the mother's blood after the delivery of the young. However, we observed that blood concentrations of relaxin were elevated for up to six weeks in lactating Labrador Retriever bitches with hip dysplasia, whereas relaxin was not
detectable after the second week of lactation in the blood of non-dysplastic Retrievers or dysplasia-resistant Beagle bitches. We also detected high concentrations of relaxin in the colostrum and milk of dysplastic bitches during the entire lactation period. Milk of non-dysplastic bitches did contain relaxin, but at lower levels and only during the first week of lactation.

We recently determined that relaxin, ingested in the milk during suckling or injected orally, is absorbed into the circulation of newborn pups. It is now important to determine the glandular source of milk-borne relaxin. Using Northern blot analysis, we intend to look for the messenger RNA molecule specific for relaxin in an effort to determine if the mammary glands and/or other tissues such as the ovary and placenta are sources of relaxin in dysplastic and non-dysplastic bitches. Other future studies will test whether the administration of relaxin causes hip dysplasia in hand-reared and milk-deprived pups born to non-dysplastic bitches, and also if antirelaxin antiserum can be used to passively immunize, and thus protect, predisposed pups that are nursing on dysplastic bitches.

George Lust

Osteoarthritis

Pathogenesis

The normal environment of healthy articular cartilage is influenced by mechanical as well as biochemical factors. One goal in our laboratory is to maintain healthy cartilage in explant culture for an extended time and then, through manipulation of biochemical and mechanical parameters, to mimic in vitro the metabolic changes that we observe in tissue from osteoarthritic joints. The loss of proteoglycan and the accumulation of fibronectin are well documented characteristics of degenerated cartilage. We are interested in learning how culture conditions, including the application of mechanical stress, alter the biosynthetic pattern of those macromolecules.

We have completed studies showing that we can maintain and manipulate proteoglycan and fibronectin synthesis in cartilage explants cultured in a defined medium supplemented with insulin, calcium, and TGF-B. Our report on those studies will be published this year. We also presented a preliminary report to the Orthopaedic Research Society in February suggesting that cartilage will respond to cyclic mechanical stress by synthesizing less fibronectin.

The experiments investigating the effect of mechanical stress on cartilage used a prototype machine designed by Dr. Peter Torzilli, a biomechanical engineer at Cornell Medical College. Our studies have so far been limited by the fact that the single-chamber design of the prototype apparatus does not allow replicate incubations. Dr. Torzilli and his engineering colleagues have therefore developed and tested a multi-chambered apparatus, which should soon be available for further experiments. In the interim we have constructed four more single-chambered devices, modeled after the prototype but designed for the application of static loads, to compare the results we obtained by applying the load cyclically to the responses that a static load of comparable magnitude would elicit. Our goals are to systematically relate the magnitude and frequency of applied load to metabolic response and to ascertain what mechanical parameters, either alone or in concert with biochemical factors, might be related to a pathological response.

Diagnosis

Our laboratory continues to have a strong interest in developing a blood
test for osteoarthritis. Early detection would allow clinicians to monitor progression of the disease, and such a test for the osteoarthritis that accompanies the hip dysplasia model would be of great value to dog breeders and owners. Recent reports indicate that people with osteoarthritis develop autoantibodies to a protein present in the plasma membrane of the chondrocyte, or cartilage cell. With the help of Susan Schaefer, a third-year veterinary student at the University of Wisconsin, we began studies last summer to determine whether such an autoantibody might be present in dogs with osteoarthritis. During her short stay in our laboratory as a Dodge Foundation Summer Fellow, Ms. Schaefer was able to isolate and purify the chondrocyte membrane protein that is the putative antigen for the autoantibody response. Technician Margaret Peery is continuing those studies. She plans to use the purified protein to set up an ELISA assay to determine if antibodies to this protein can be detected in dogs with osteoarthritis and hip dysplasia.

Just as the development of the very sensitive ELISA (enzyme-linked immunosorbent assay) has made it attractive to look for biochemical markers of disease, so the recent advances in molecular biology have made it possible to look for genetic markers of disease. This fall we welcomed to our laboratory Dr. Dai-Wei Zhang, an orthopedic surgeon from Beijing, China, who plans to study for the Ph.D. He is isolating DNA from the white blood cells of normal and dysplastic dogs and their progeny and initiating experiments to look for restriction fragment length polymorphisms, genetic markers that may be associated with the disease.
Concern. It is the Type II collagen that gives articular cartilage its tensile strength, but repair tissue often contains the less desirable (for cartilage) Type I collagen. In collaboration with collagen biochemists Dr. Joyce Wooten and Dr. Ronald Minor of the College of Veterinary Medicine’s Department of Pathology, Dr. Todhunter has developed methods to determine the ratios of Type I and Type II collagens in small quantities of repair tissue. This information will be useful to guide clinicians in providing the most favorable conditions for cartilage regeneration.

Nancy Burton-Wurster
We reported two years ago that the intestine produces populations of cells with protective properties against *Trichinella spiralis* infection. That provocative finding has potential significance for concepts of intestinal immunity and for novel vaccine and immunization strategies, and we have committed substantial effort to exploring the mechanisms and site of production of those protective cells.

A previous report outlined the approaches we had used to localize the site of production to the intestine. Those experiments analyzed cell activation based on the location of dividing cells, since one of the important early steps of an immune response is the division of a very small number of cells with immune specificity for the target pathogen into a large and effective population. We observed that the lymphocyte population of the intestinal epithelium, where *T. spiralis* takes up residence, responded very rapidly to infection with the parasite, completing division within twelve hours. A similar degree of cell activation was also evident in the immediately adjacent lymphoid cell population of the lamina propria. A third cell population, that of the Peyer's patches, the most structurally organized lymphoid tissue of the gut, was noticeably quiescent with no increase in cell division apparent until four or five days after infection had begun.

Another measure of recognition of a foreign pathogen is the capacity of lymphocytes to undergo a second wave of division when confronted with the target antigen in tissue culture. We examined the *in vitro* response of cells from the Peyer’s patches, the mesenteric lymph node, and the lymph draining the gut. Cells in lymph draining the gut were reactive to
antigen two days after infection and
cells in the mesenteric lymph node by
day three, but cells from the Peyer's
patch were unreactive until nine days
after the infection had begun.
That evidence suggested quite
strongly that the organized tissue of the
Peyer's patches was not the source of
the protective cells. Only two
possibilities remained, the lymphoid
cells of either the intestinal epithelium
or the lamina propria. Both
populations are very distinct in terms of
the cell types present in them, and
nothing was known of the possible
interrelationships between the two
populations. We decided to examine
both of them, but the difficulty of
isolating lamina propria cells has meant
that most of our work so far has focused
on the epithelial lymphocyte. Studies
conducted by Anthony Vella, a
graduate student in our laboratory,
showed that the first 24 hours of
infection produced a series of distinct
changes in the epithelium and the
overall villus. Two effects were quite
pronounced: a loss of some 50% of the
total intraepithelial lymphocyte pool
and a shortening of overall villus
length. Other changes included
modifications to the basement
membrane that could not be
quantified. Those changes were specific
to the areas of the intestine that the
parasite had invaded and were not
found in areas the parasite had not
invaded. By doing a series of intestinal
washouts at various time points after
infection, Mr. Vella showed that the cell
loss was most likely due to migration of
the lymphocytes away from the
epithelium into the lamina propria.
The use of monoclonal antibodies
directed against surface markers of the
remaining lymphocytes demonstrated
that all of the major phenotypes lost
proportionate numbers of cells,
therefore indicating that migration was
not selective. We concluded further
that the major cell type moving out of
the epithelium was CD8⁺, since these
cells comprise 60-70% of the epithelial
lymphocyte pool. Our work with the
lymphocytes obtained from thoracic
duct lymph had shown that the
protective cells were CD4⁺, not CD8⁺.
Therefore, it appeared unlikely that the
epithelium was the principal source of
the protective CD4⁺ cells important in
this form of immunity.

Although this work has for the time
being ruled out consideration of the
epithelium as the source of protective
cells and instead focused attention on
the lamina propria, it has not lessened
our interest in the epithelial cells. We
are investigating the possibility that the
movement of epithelial cells has a role
in immune cell activation in the lamina
propria. Our preliminary findings
suggest that the epithelium normally
secretes factors that prevent the
epithelial lymphocyte population from
dividing or responding to antigen.
Such a mechanism would explain the
negligible response of most individuals
to the potentially life-threatening daily
influx of foreign proteins in food. But
this poses a conundrum—how does the
body distinguish between a food
antigen and a parasite antigen? We
theorize that the division of cells and
their abnormal migration during the
first 24 to 48 hours of a *Trichinella*
infection may represent the triggering
of an active form of immunity in
response to a pathogen rather than a
benign substance such as a food
antigen. We hope that the next year’s
work by graduate student Ted Liana
may help illuminate this problem.

Robin G. Bell
We have continued our studies of mucosal immunity against the early stages of infection by Trichinella spiralis larvae. We had found previously that, in suckling rat pups, parasite-specific antibodies are able to cause the expulsion of larvae from their niche in the intestinal epithelium. Melissa Carlisle, formerly a graduate student and now a post-doctoral associate in our laboratory, confirmed that the entrapment of the parasites in intestinal mucus follows their expulsion from the epithelium and thus promotes their transit through and removal from the intestine. We now have evidence that an antibody that does no more than cause mucus entrapment will not ultimately protect the animal from infestation with the parasite.

In a recent study, Dr. Carlisle challenged the supposition that, after the antibody binds with the parasite, the Fc region of the antibody molecule mediates rapid expulsion by binding with some other cell or molecule in the intestine, thereby triggering expulsion. The Fc region confers upon an antibody its subclass-specific properties, including the ability to bind to certain cells or to complement. Dr. Carlisle discovered last year that destruction of the Fc portion through chemical cleavage does not impair the ability of the antibody to expel the parasite. We speculate that the binding of antibody itself is interfering with some essential function of the parasite in order to cause its expulsion from the epithelial niche.

Dr. Carlisle has now found that entrapment of parasites in mucus is also not dependent on the Fc portion of the antibody molecule. This was somewhat surprising, in that other researchers have postulated that
antibodies interact with mucus in a specific way. Our results suggest that antibodies in some way affect the ability of the parasite to maintain itself free from mucus.

Dr. Carlisle and Oti Otubu, another graduate student in our laboratory, have also discovered that the antibodies that protect infant rats have less effect in older animals. While they are able to compromise the mobility of larvae in the epithelium, this functional compromise does not result in parasite expulsion.

Lynn Usack, a research technician in our laboratory, has studied the parasite proteins that are expressed in the secretions and also on the surface of the parasite. Graduate student Laurie Ellis is currently defining the composition of the antigen, a glycoprotein. By treating the antigen with glycosidases, she is cleaving off the carbohydrate portion of the molecule in order to determine whether antibodies will still bind to the remaining protein.

Dr. Prema Arasukavalar, a post-doctoral fellow working in our laboratory, is approaching the problem from another direction. Dr. Arasukavalar has been preparing a recombinant DNA library from the parasite's messenger RNA. She is using antibodies protective against *T. spiralis* to identify the parasite protein targets of those antibodies among the cloned gene products in the library.

**Equine Influenza**

In our continuing work with equine influenza virus, research technician Lucy Gagliardo has applied an *in vitro* lymphocyte assay toward the comparison of different types of vaccines for their ability to stimulate protective immunity against influenza. In addition, we have continued to monitor variation in the surface glycoproteins of the virus using monoclonal antibodies. We have applied a neutralization assay to those studies, as we have found that the conventional hemagglutination inhibition test failed to detect antibodies that were capable of neutralizing the infectivity of one of the newer viruses. This recent isolate is more closely related to viruses isolated in 1956 than to viruses isolated in 1987.

Finally, graduate student Laura Hanson is pursuing studies of the mechanism of virus neutralization. We have preliminary evidence that antibodies that bind to different regions of a virus protein mediate neutralization of infectivity by different mechanisms that may vary in their effectiveness. These findings have important implications for the design of subunit vaccines, which contain single proteins ("pieces" of viruses) chosen for their ability to provoke a strong antibody response. Natural mutations in the influenza virus might alter an epitope, or binding site, so that a formerly protective antibody would no longer bind to it. A vaccine containing only the affected protein might in this way be rendered ineffective.

*Judith A. Appleton*
Pregnancy and Histocompatibility

Our study of the immunological relationships between mother and fetus in the horse has three major components, and progress was made in each area in 1990.

The first area concerns the antigenic targets that the fetus presents to the mother during pregnancy. In the horse the primary targets appear to be the paternal class I Major Histocompatibility Complex (MHC) antigens. These MHC antigens are expressed in a temporally and physically restricted manner. They are found at high density only on the rapidly dividing trophoblast cells of the chorionic girdle and the young endometrial cups that derive from the girdle cells between days 30 and 45 of gestation. Juli Maher, a post-D.V.M. graduate student in the laboratory, has undertaken a study of the regulation of expression of these class I MHC genes using molecular biological techniques.

The second area of focus is the maternal immune response to the fetal antigenic targets. We are concentrating on the maternal white blood cells that cluster around and appear to destroy the invasive trophoblast cells of the endometrial cups. The remainder (and major portion) of the fetal-maternal interface, the allantochorion, does not attract substantial populations of maternal lymphocytes. We wish to understand the composition and function of these lymphocyte populations and their relationship to the expression of class I MHC antigens by the invasive trophoblast cells.

Several variants of normal horse pregnancy can be established by embryo transfer. The interspecies pregnancies are marked by a more intense lymphocyte response to the endometrial cups in an otherwise normal gestation. In the extraspecies pregnancy, the donkey chorionic girdle cells fail to invade the horse endometrium. This results in the absence of endometrial cups in these pregnancies. A high proportion of donkey-in-horse pregnancies end in abortion between days 80 and 95 that is marked by the accumulation of maternal lymphocytes all along the border of the allantochorion.

The benefits of these various types of pregnancy are counterbalanced by the lack of good markers for distinguishing horse lymphocyte populations. Mr. Chonghui Zhang, a graduate student in our laboratory, has spent the past four years creating monoclonal antibodies against horse lymphocytes and characterizing their reactivities. This work has been quite fruitful, and, in addition, the reagents have proved useful in the identification of several new cases of immunodeficiency disease and leukemias in horses from the Cornell Large Animal Clinic and from other veterinary schools in the United States. Dr. Gabriele Grunig, a veterinarian from Switzerland who recently joined our group, will continue work on this project. Those efforts should be aided by our participation in an International Workshop on Equine Lymphocyte Differentiation Antigens, which will be held in July of 1991 in Cambridge, England.

The third part of our study involves the cell biology of the trophoblast cells themselves. It is important for us to obtain pure populations of trophoblast cells, both for the molecular characterization of the antigenic targets they express and for use as targets in in vitro tests of the functional capacity of the maternal lymphocytes that cluster around the endometrial cups in vivo. Fortunately, the slow development of the equine placenta and its late attachment to the uterus allow us to recover horse conceptuses using nonsurgical techniques until day 36 of pregnancy. At this stage of gestation the trophoblast cells are available in relatively large quantities, and it is possible to isolate quite pure populations. One of our principal experimental goals in these studies is to develop an in vitro model for endometrial cup development that closely parallels the situation in vivo.

Equine Tumor Biology

Our studies of equine sarcoid, an important skin tumor of horses, were marked by two major events during the past year. In April the first international scientific meeting devoted to this condition was held in Interlaken, Switzerland. The meeting was organized by our laboratory and that of Professor Sandor Lazary of the Veterinary School of the University of Berne and was sponsored by the Dorothy Russell Havemeyer Foundation. That meeting brought together equine clinicians, immunologists, geneticists, and virologists. An important result of the meeting was the formation of a continuing study group of clinicians and scientists who will cooperate in a three-year investigation of sarcoid. This project will also be sponsored by the Havemeyer Foundation. The goal of the project is to study the relationship between papilloma viruses, equine genetics, and the clinical course of the disease. There is strong evidence that a virus identical or very similar to bovine papilloma virus causes sarcoid, and that horses of certain MHC types have increased susceptibility to tumor
formation. Finally, some cases of sarcoid can be cured by standard surgical techniques, while others continue to recur irrespective of the treatments administered. The study group hopes to identify critical host and environmental factors that influence the development of the tumors and the ability of horses to overcome them.

The second event was the completion of a Master’s Degree by Mr. John Angelos, a third-year veterinary student. John made a study of bovine papilloma virus and sarcoid during the past three years. During the fall of 1990 a scientific poster representing his work was awarded first prize in a competition of research conducted by veterinary students at Cornell.

**Perestroika and Equine Reproduction**

In closing, it is a pleasure to mention a scientific collaboration undertaken in the autumn of 1990 with my colleague Dr. W. R. “Twink” Allen of Newmarket, England, and a group of veterinarians and animal scientists in Eastern Europe. The experiments involved freezing a group of horse embryos in England and transporting them to two equine reproduction centers, the Slatinany Stud near Pardubice, Czechoslovakia, and Poland’s Academy of Agriculture in Krakow. The embryos were thawed and transferred to local recipient horses using a surgical technique requiring general anesthesia. The successful transfer of frozen-thawed horse embryos has been accomplished very few times—probably no more than ten foals world-wide have been born as a result of this procedure. At the time of this writing we had learned that two of the three recipient mares in Czechoslovakia were pregnant, an excellent result for procedures carried out under less-than-optimal surgical conditions. During our stay the hospitality of our East European colleagues was tremendous. We hope that the dismantling of centralized political and economic structures in the East will allow us the opportunity to offer our laboratory as a site for advanced training of veterinarians and scientists from Eastern Europe.

*Douglas F. Antczak*
This year we have made substantial advances in our studies of canine parvovirus (CPV) and the related feline panleukopenia virus (FPV).

We have successfully cloned complete genomes of CPV and FPV into bacterial plasmids using recombinant DNA methods, and have shown that the viruses that were cloned can be recovered when those plasmids are added to tissue culture cells. We are using those plasmid clones to examine the differences between CPV and FPV that determine the unique properties of each virus. Analysis of viruses prepared from those clones has already shown that the gene that encodes the coat protein of the viruses determines the canine and feline host ranges.

To further examine how the protein coat acts to determine the host ranges and other functions of the viruses, we have been collaborating with X-ray crystallographers at Purdue University to determine how that protein is folded to make up the three-dimensional structures of the viruses. The data obtained have shown that the specific properties of CPV that distinguish it from the feline virus are located on the outside of the virus protein coat, suggesting that they are altering the ways in which the virus interacts with canine cells to allow the infectious process to occur.

Antigenic characterization of CPV strains isolated from canine clinical specimens has shown that the antigenic type of many CPV isolates now differs from that which we saw in viruses during the early 1980s. Although the variant viruses probably differ in only a single antigenic site, that one change has apparently enabled them to supersede the previous strain, CPV Type-2a, to the extent that the variant strains now comprise most CPV isolates from all parts of the United States. We
are studying the nature of the changes in CPV-2b, and are using DNA sequencing to characterize the genetic and evolutionary relationships between the CPV Type-2b strain and the earlier CPV Type-2 and CPV Type-2a strains.

Examination of viruses from hosts other than dogs and cats has shown that foxes are infected by a virus that is closely related to the cat viruses, while the Asiatic raccoon dog is infected by CPV. We hope to further examine those and other viruses from wild carnivores in order to fully define the host-virus relationships among this group of viruses.

This work has given us materials and information that allow us to use the most advanced techniques to examine the structures and functions of CPV and FPV. These comparative studies will answer fundamental questions about viral host range, pathogenesis, and evolution. In addition, such studies will allow us to ensure that we are fully informed of any changes in canine parvovirus, so that we can develop the best vaccines and other means to combat those diseases.

Colin R. Parrish

Molecular structure of one canine parvovirus protein subunit. Courtesy M.G. Rossmann, American Association for the Advancement of Science.
The following is a list of manuscripts published by staff members of the Baker Institute in 1990. Publications listed as “in press” in last year’s report are repeated this year, with their original numbers, to record their full bibliographic details.


The 40th anniversary of the Institute is a time of reflection and a time of anticipation. I am fortunate to have known both Institute directors. Drew Baker was a close family friend. He helped mold my own career and those of the many young veterinary scientists who passed through the Institute during his time here. Doug McGregor has been a dedicated leader. He has devoted the past 14 years to building and conserving the Institute’s resources while using them to catalyze the efforts of the Institute’s outstanding scientists to attack on a broad front the diseases of dogs and other animals.

When the Advisory Council met in September, I was asked to address the question of the Institute’s future course in a letter to the Dean of the College of Veterinary Medicine. Writing on behalf of the Council, I expressed to Dean Phemister our pride in being associated with such a prestigious Institute and Veterinary College. I also expressed the Council’s hope and expectation that, under its new director, the Baker Institute will continue to watch over the health needs of dogs and to serve their human companions. The search for a new director of the Institute offers a maximum opportunity for progressive change or for strengthening of existing disciplines within the broad focus of research on canine diseases. There are major research areas that could be explored—treatment of canine cancers, studies of the genetics of dogs, and experimentation on the antigens of canine white blood cells, for example—while maintaining the Institute’s strong programs in infectious diseases and hip dysplasia. The new director will assume leadership of a dynamic and vibrant program, as always in touch with dog owners, kennel clubs, and practitioners.

The Advisory Council continues to have a close relationship with the Institute’s scientists and administration. In this transition period the Council welcomes suggestions from friends of the Institute and will work with all toward our common goal of improved health for animals.

Robert E. Shope, M.D.
Chairman

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MR. ROBERT WINTHROP
1982-1990
Mr. William Rockefeller, a dedicated friend of the Institute, died on March 16, 1990 at the age of 71. At the time of his death, Bill was the chairman of the Institute’s Advisory Council, on which he had served since 1979. He was an imaginative and energetic advocate of our work on behalf of dogs and had also promoted canine concerns as president of the American Society for the Prevention of Cruelty to Animals, the Westminster Kennel Club, and the American Kennel Club. As chairman of the Geraldine Rockefeller Dodge Foundation, Bill guided the legacy of his great-aunt, a central figure in the founding of the Institute. In 1950, her generosity funded the establishment of the Giralda Division of the Cornell Research Laboratory for Diseases of Dogs. Ten years later, she provided the means to establish within that division the first tissue culture laboratory in any veterinary college in the world. Her wish to make available a superior and more humane technology saved lives in the laboratory, and countless more beyond.

In September the Geraldine R. Dodge Foundation made a grant to the Institute in memory of William Rockefeller. Bill’s widow Molly had requested the funds for exterior restoration of the 100-year-old farmhouse standing on the grounds of the Institute, where it has lately served as a caretaker’s residence. Thanks to Molly’s inspiration and to the generosity of the Dodge Foundation, Robert Winthrop, and Louise Humphrey, the house now has a cedar shake shingle roof with copper flashing, the chimney has been repaired, and new gutters and downspouts will be installed in the spring.

Once Molly had focused our attention on the house, we began to appreciate its value and potential. We realized that, with proper renovation, the building would make an excellent conference center. Such a facility would promote the Institute’s dual mission of education and research in a very meaningful way. We have had preliminary plans drawn up for renovating the interior to include a conference room, kitchen, library, and overnight accommodations for several guests. We hope to create a gathering place for visiting scientists and scholars that will be dedicated to the exchange of knowledge for the advancement of animal health science.

The restoration and renovation work we envision has been estimated to cost $150,000. The successful completion of this project will depend on our ability to raise that sum through gifts, large and small, designated specifically for the conference center. The names of all contributors of $1000 or more will be inscribed on a bronze plaque that will hang in the center’s conference room.
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<td>Dr. William P. Cadwaller, Jr.</td>
<td>Campus Veterinary Clinic</td>
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<td>Mr. and Mrs. James Cavanaugh (in memory of Fritz)</td>
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<td>Dr. Kenneth W. Chamberlain, Jr. §</td>
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<td>Charlestowne Veterinary Clinic</td>
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<td>Ms. Jane P. Cheatwood (in memory of Mandy)</td>
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<td>Checktowaga Veterinary Hospital</td>
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<td>Dr. and Mrs. James P. Childress, Jr.</td>
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<td>Mrs. Lewrie A. Close (in memory of Misty)</td>
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<td>Mrs. Patricia E. Dowling (in memory of Barney)</td>
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Mr. and Mrs. Eugene J. Callahan  
(in memory of Kelly)  
Mr. and Mrs. Carl J. Camelo, Jr.  
(in memory of Jenny)  
Ms. Carol B. Camelo  
(in memory of Jenny)  
Mrs. Lois F. Cameron  
Ms. Rachel Campanella  
(in memory of Penni)  
Mr. Sydney Campbell  
(in memory of Mack and Nini)  
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Mr. and Mrs. Scott Canfield  
(in memory of Daisy and Duke)  
Ms. Angie H. Caputo  
(in memory of Cindy)  
Carlson Veterinary Clinic  
Dr. David J. Carlson  
Mr. and Mrs. Paul Carlton  
(in memory of Heidi)  
Carmel Animal Hospital  
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Dr. and Mrs. Philip B. Carter  
Mr. and Mrs. Ronald A. Carubba  
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Mr. and Mrs. O. T. Carver  
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Dr. Robert F. Case  
The Cat Hospital  
Mrs. Santo Cataudella  
(in memory of Hershey)  
Cathedral Dog and Cat Hospital  
Ms. Gail Celone  
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Central Veterinary Hospital  
Centreville Animal Hospital  
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Dr. Dean J. Cerf  
Ms. Daphne Chandler  
Mrs. Elizabeth Chandler  
Mrs. Genevieve M. Chapin  
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(in memory of Raspy)  
Mr. and Mrs. Howard J. Chase, Jr.  
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Mr. and Mrs. Bobby V. Checchi  
(in memory of Kaiser and Phoenix)  
Mr. Stewart Chodosh  
Ms. Betsy Chouinard  
(in memory of Pepe)  
Ms. Marie L. Cirelli  
(in memory of Tasha)  
Mr. and Mrs. Robert Citron  
(in memory of Chivas)  
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Mr. and Mrs. Gerald W. Clark  
(in memory of Mitzie)  
Ms. Jeanne M. Clark  
(in memory of Ben and Samantha)  
Mr. and Mrs. John R. Clark  
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Cobleskill Veterinary Clinic  
Ms. Janice M. Cocks  
(in memory of Strider)  
Mr. and Mrs. Michael A. Cole  
Mrs. John H. Coleman  
Mr. and Mrs. Robert L. Coleman  
(in memory of Benny)  
Mr. and Mrs. Harold C. Colley  
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Dr. Linda L. Collier  
Colonial Animal Hospital  
Ms. Marcia C. Colp  
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Mr. and Mrs. James R. Colway  
Community Veterinary Hospital  
Ms. Polly Comolli  
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Companion Animal Hospital  
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(in memory of Ole)  
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Ms. Carolyn Ann Cone  
(in memory of Hyia)  
Dr. James F. Cone, Jr.  
Ms. Judith Miller Conlon  
(in memory of Tasha, Sasha and Mishka)  
Mrs. Eileen G. Connolly  
Mr. John W. Cook  
Miss Mary Lee Cooke  
Mr. and Mrs. H. James Corbett, Jr.  
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Corder Companion Animal Clinic  
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Mrs. Margery D. Corrigan  
Ms. Ruth Costin  
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Mrs. Joan H. Cottrill  
Coudersport Animal Health Center  
Ms. Peggy Coughnour-Fecher  
Country Animal Clinic  
Country Home Veterinary Clinic  
Country View Animal Hospital  
Countryside Animal Hospital  
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Countryside Animal Hospital  
(Houston, TX)  
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Mr. and Mrs. Norman A. Croft  
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Mr. Ted Cryer  
Mrs. Margaret Cullinan  
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Mr. and Mrs. F. Glenn Cummings  
(in memory of Chrysiss)  
Ms. Helen T. Cupper  
(in memory of Tristan)  
Ms. Dawn E. Currier  
(in memory of Mitzu)  
Mr. and Mrs. John J. Cybulski  
Mr. Frank D'Agostino  
(in memory of Tania)  
Mr. and Mrs. Morris Dagen  
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Mrs. Anthony Dalesandro  
(in memory of Starr)  
Mr. and Mrs. Joseph J. Daley  
(in memory of Sebastian, Nick, and Mark)  
Mr. and Mrs. John Dalrymple  
Mr. and Mrs. John R. Danielski  
(in memory of Christie)  
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Mr. Herbert Davis, Jr.  
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Ms. Paula S. Davis  
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Mr. and Mrs. Richard C. Davis  
Mr. and Mrs. Anthony DeBrino  
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Mrs. Leonora Decker  
(in memory of Bambi, Daisy and Beauregard)  
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(in memory of Booster)  
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Dr. Dianne De Lorenzo
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(in memory of Dutch)
Dr. Thomas DeVincentis
Mr. Eric DeVine
Ms. Dorothy E. DeYoung
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(in memory of Alley Cat)
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Mrs. Ellen S. Difantis
(in memory of Bo Ling)
Mrs. Sheree Lee Doggett
Mrs. Judith A. Dolan
(in memory of Shaggy)
Ms. Serena C. Dolecki
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Dr. J. F. Doles
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(in memory of Bubbles and Run)
Dr. Michael E. Doty
Mr. and Mrs. William F. Droese
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Mr. and Mrs. William E. Drury
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Mrs. Edith Wills Dubose
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East Side Animal Hospital
Easthampton Animal Hospital
Easton Veterinary Clinic
Edgefield Veterinary Hospital
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Mr. and Mrs. H. Burton Entrekin (in memory of Egbert)
Ms. Leona Epstein
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Dr. Gilbert J. Feldman
Mr. James C. Fennimore
Mrs. Finita C. Fensel
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Ms. Evelyn Fenston
Ms. Marianne M. Feraca
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Dr. and Mrs. Lincoln E. Field
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Ms. Mary D. Fine (in memory of Crunch)
Mr. and Mrs. Alan J. Finkel
(in memory of Taffy)
Mr. Herman Fisher
Ms. Sheila A. Fisher
Mrs. William W. Fisher
(in memory of Merl)
Dr. Jane Fishman
Mr. William G. Fladung
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Ms. Sarah J. Fletcher
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Mrs. Bernadette Fossler
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Dr. Lorraine A. Fournier
Mrs. Sue Foust
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Freedom Animal Hospital
Mr. and Mrs. William K. Freestate (in honor of Chipper)
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Ms. Theda Fuller (in memory of Mr. Cabb and in honor of Dr. and Mrs. George Hahn)
Mr. and Mrs. Marco Furino
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Mr. Ronald Fuydal
Ms. Virginia L. Gahan
Mr. and Mrs. Arthur Gaines
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Ms. Elizabeth A. Galloway
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Mr. and Mrs. Charles A. Gardiner
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Mr. and Mrs. Thomas J. Gentner (in memory of Emily)
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Gerber Small Animal Hospital
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Mrs. Ruth H. Geringer
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German Flatts Veterinary Clinic
Gerson Animal Hospital
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Mrs. Linda Gillespie
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Mrs. Sally Gilrain (in memory of Tashe and Kelly)
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Mr. and Mrs. Maxwell Gimple
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Mr. Gregory Giron
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Glenelg Animal Hospital
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Miss Evelyn Graham (in memory of Nancy Green Parker)
Mr. and Mrs. Joseph Graham
Miss Mildred Graham (in memory of Nancy Green Parker)
Mrs. Joan L. Grant
Mr. Stanley R. Grant
Mrs. Hannelore B. Grastorf
(in memory of Missy)
Ms. Isabel W. Gray
(in memory of Elsie T. Gray)
Ms. Vera M. Gray
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Dr. Mitchell H. Greenberg
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Mr. Martin Jonigan
Ms. Cathy M. Jordan (in memory of Crunch)
Ms. Ramona P. Jovenall (in memory of Heidi)
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Ms. Joann Julia (in memory of Muffin)
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Mr. Robert E. Kesel (in memory of Penney)
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Ms. Jean Kleinschmidt (in memory of Toby)
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Mr. and Mrs. Francis J. Koenig (in memory of Flicker)
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Mr. and Mrs. Marius S. Kominek
Mr. Victor Konig
Dr. Moe Kopp
Dr. Lloyd Kornblatt
Ms. Judith Kossin (in memory of Havel)
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Mrs. Kathleen S. Kraus (in memory of Buttons)
Mr. and Mrs. Leon Kraus (in memory of Princess)
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Ms. Bonnie S. Krieger (in memory of Tamara)
Dr. and Mrs. Arthur I. Kronfeld
Ms. Virginia B. Kuertz (in memory of Widegon)
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Miss Eleanor C. Lawhorne
Dr. and Mrs. Jock D. Lawrason
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Mr. Emmett W. Louis (in memory of Chevy)
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Dr. and Mrs. Harold Ludman (in memory of Jennie)
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Ms. Stephanie Lukens (in memory of Schweppes)
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Mr. and Mrs. Irving M. Lustig (in memory of Chris)
Mrs. Enid C. Lutz (in memory of Happy Susie)
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Mrs. Kathleen McAllister
Mrs. Patricia P. McCann
(in memory of Penny)
Mr. Jack H. McCarthy
(in memory of Toby)
Mr. and Mrs. Robert McCarthy (in memory of Whitney and Mike)
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Dr. Anne McComb
Fr. Francis McCormick
(in memory of Dr. Dana D. Ford)
Ms. Geraldine McCormick
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Mrs. Janet Mantineo
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Mr. and Mrs. Michael Mariano (in memory of Bandit)
Ms. Marianne Marks
Mrs. Sharon L. Martin
(in memory of Ginger)
Ms. M. Helen Martino
Mr. and Mrs. Matthew J. Martino
Mrs. Barbara K. Marx (in honor of Dr. Judith Johnessee and in memory of Kela and Kryssy)
Mr. Noruko Masai (in memory of Harry)
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Dr. James T. Meunier
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Mr. Jonathan Meyer
Mr. and Mrs. Albert Michaels
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Ms. Sonja Midgette
(in memory of Mr. T.)
Ms. Thelma Midgette
(in memory of Mr. T.)
Millburn Veterinary Hospital
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Mr. John C. Miller
(in memory of Holly)
Mr. and Mrs. Paul Miller
(in memory of Sadie)
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Mr. Martin Minor (in memory of Red and Sugah)
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Montrose Animal Health Center  
Dr. Calvin Moon

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Ms. Dorothy S. Nadherny (in memory of Pepper)  
Dr. Jolene Nagakura  
Ms. Vinnie Natale (in memory of Dulcinea)  
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Mrs. Ruth L. Osterwald (in memory of Dutchess and Buffy)  
Ms. Anna M. Ostrosky (in memory of Tiko I and Tiko II)
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Ms. Cheryl H. Reynolds (in memory of Kam)
Mrs. Christine M. Reynolds (in memory of Benjamin J. Beagle)
Mrs. Regina Reynolds
Mrs. Elizabeth G. Rhoades (in memory of Cookie)
Mr. and Mrs. Ralph E. Rhule
Ms. Joan L. Richards (in memory of Fluffy)
Mr. and Mrs. Mark Richardson (in memory of Onyx and Georgie)
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Mrs. Marian Richter-Sage (in memory of Penny)
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Mrs. Mary D. Rider (in memory of Bridgett)
Dr. Steven B. Ringer
Dr. John F. Risickella
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Riverside Animal Hospital
Mr. and Mrs. Chris Rivest (in memory of Bridgett)
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Ms. Elizabeth N. Roberts
Ms. Martell Roberts
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Dr. Edward A. Rogoff
Mrs. Phyllis Roitsch (in memory of Connie and April)
Dr. Calvin B. Roper* (in honor of Dr. Jeffrey S. Hubsher and Dr. Arnold Leitner)
Ms. Sharon A. Rose (in memory of Sheba)
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Dr. James H. Rosenberger
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