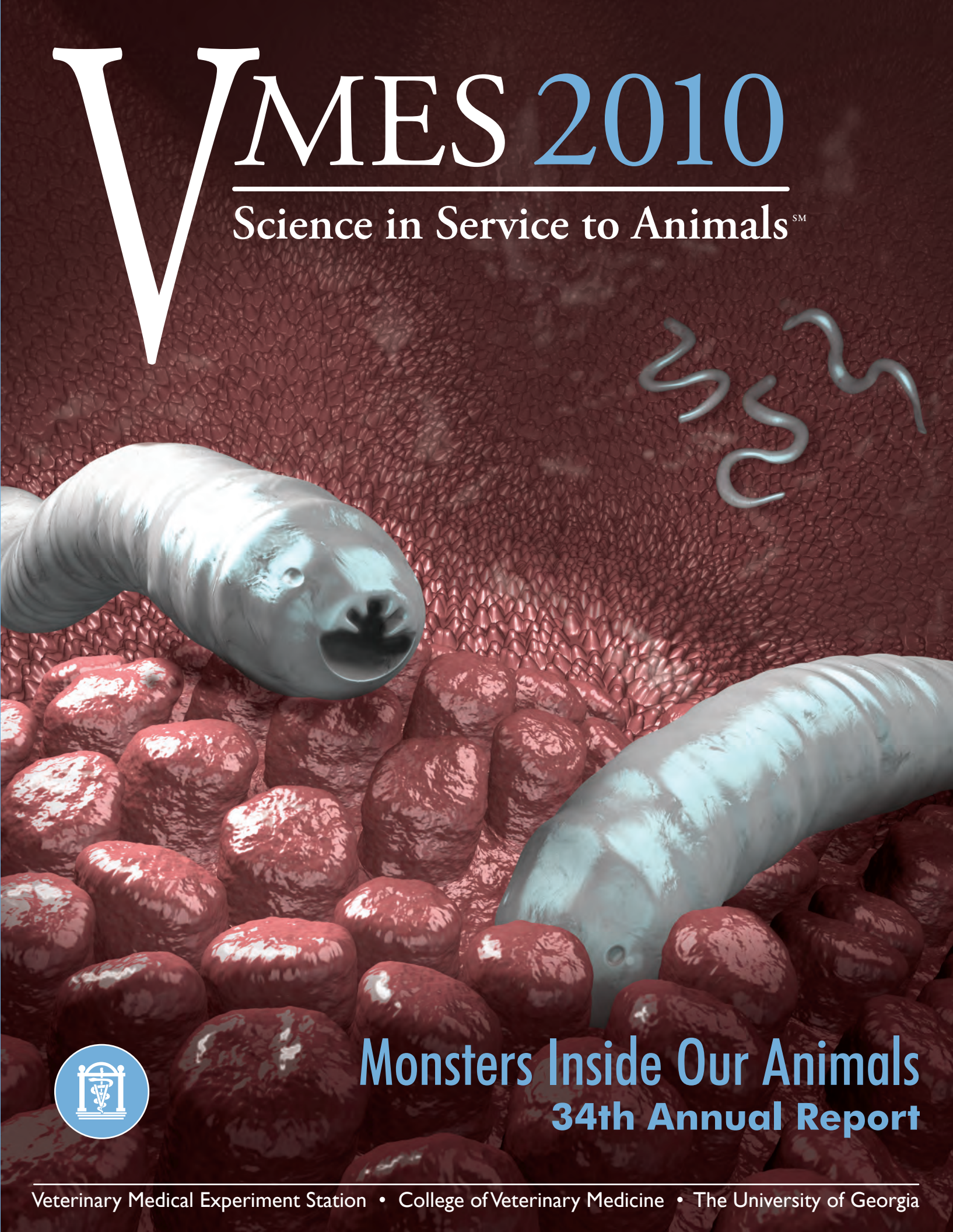
VMES 2010

Science in Service to Animals℠



Monsters Inside Our Animals

34th Annual Report

Veterinary Medical Experiment Station • College of Veterinary Medicine • The University of Georgia

Contents

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The University of Georgia

VMES 2010

Science in Service to Animals℠

34th Annual Report

July 1, 2009 to June 30, 2010

Overview, Mission, & Objectives

The VMES mission is to coordinate research on animal disease problems of present and potential concern to Georgia's livestock and poultry industries.

The Veterinary Medical Experiment Station (VMES) was established as a budgetary entity by the state legislature in July 1976 following approval by the University of Georgia Board of Regents in 1973.

Specific VMES objectives are:

- To improve the health and productivity of domestic livestock, poultry, fish, and other income-producing animals and wildlife through research;
- To assist in preventing disease epidemics by providing laboratory resources and highly skilled scientific personnel;
- To assist in protecting human health through the control of animal diseases transmissible to man;
- To improve the health of companion animals, which serve to enrich the lives of humankind;
- To train new scientists in animal health research in order to provide continuity and growth in this vital area of veterinary medicine.

The Veterinary Medical Experiment Station is committed to enhancing animal production, profitability, and well-being by improving animal health.

All programs and activities of the Veterinary Medical Experiment Station are conducted without regard to race, color, national origin, age, sex, or handicap.

From the Director

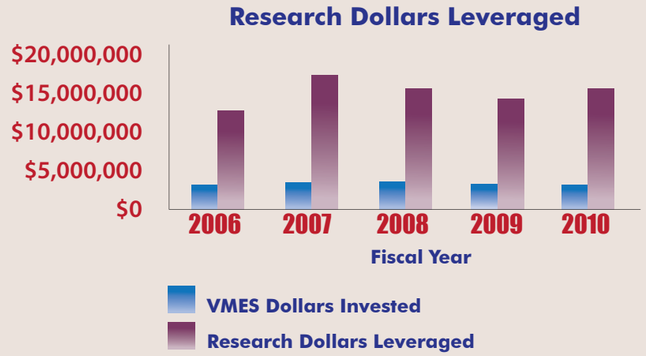
These are challenging times for veterinary research. Despite the importance of animal agriculture to the State of Georgia and our nation, funding for both basic and applied research related to animal health and food safety is diminished, or at best remains flat. Nevertheless, research expenditures have continued to rise at the College of Veterinary Medicine over the last 5 years. How is this possible? Research at the University of Georgia College of Veterinary Medicine (CVM) is supported by funding from various sources. The state appropriations for the Veterinary Medical Experiment Station (VMES) is an integral component of this research support with its major function aimed at investigation and elimination of animal diseases affecting the citizens of Georgia and Georgia's economically essential livestock and poultry industries. The majority of research projects are supported by federal, and private sector competitive grants and contracts. The diversity of our faculty's research combined with our investigators' excellent research training, dedication and hard work have allowed the research enterprise of the CVM to develop and progress even in these tough economic times where garnering of extramural research funds is highly competitive. For example, many of our clinical scientists have expertise that is sought after by private industry and whose research is recognized and supported by private donations. Likewise, federal agencies such as USDA and NIH often put out requests for proposals in specific areas of interest or importance to that agency. Our research faculty is well-poised to take advantage of these opportunities and our researchers have had success in getting these research grants. Research Budgets for VMES research projects are on file in the College of Veterinary Medicine and expenditures are made in accordance with these budgets and University of Georgia fiscal policies.

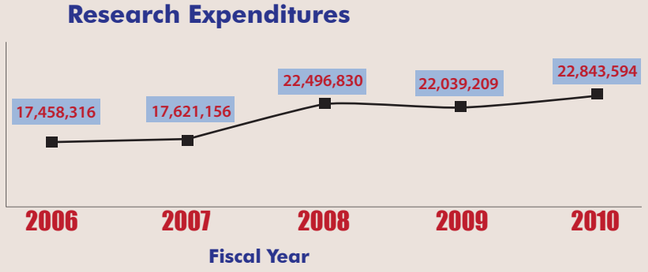
The cover story of this year's annual report highlights the research of Dr. Ray Kaplan and Dr. Adrian Wolstenholme on the protection of livestock against parasitic worms. As they state in the article: "…the threat that gastro-intestinal nematodes (roundworms) pose to the health and productivity of livestock such as cattle, horses, sheep and goats, is greater than it has been for more than fifty years since the first modern dewormers, or anthelmintics, were introduced." The basic and applied research carried out by the Wolstenholme and Kaplan labs is a great example of the relevance and importance of veterinary research to our state and the nation as a whole.

This 34th VMES Annual Report provides an overview of peer-reviewed, competitive projects and new faculty start-up projects conducted during fiscal year 2010 (July 1, 2009 – June 30, 2010). Additional information on any of these projects can be requested by contacting the VMES office by phone, email or website, or directly from the investigators themselves. A list of publications is provided as well. These peer-reviewed papers represent a selection of VMES supported work and other scholarly research originating at the College of Veterinary Medicine.

Harry W. Dickerson

Financial Tables





Expenditures by Category

Budget Category	Amount	% of Budget
Personnel - Researchers/Techs/Research Staff	$1,962,704	68.8%
Research Materials & Equipment	$533,882	18.7%
Personnel – Research Administration & Accounting	$354,315	12.4%
Travel	$2,240	0.1%

A summary of the College's research funding is provided above. Over the past year approximately five research dollars were leveraged for each VMES dollar invested. Expenditures are from all sources including State Appropriations, Extramural Research Funding, Donations - Includes all expenditures including personnel costs.

Monsters Inside Our Animals

Millions of people have been entertained and informed about parasites that infect humans by the TV show, 'Monsters Inside Me'. Part of the vicarious pleasure of watching the show is the knowledge that these infections are mostly very rare in the United States, and the chance that any one of us will fall victim to these horrible diseases is remote. However, the situation is very different for our companion and livestock animals and infection with one or more parasites is a daily threat that we all - owners, veterinarians and researchers - need to be aware of and to guard against. In particular, the threat that gastro-intestinal nematodes (roundworms) pose to the health and productivity of livestock such as cattle, horses, sheep and goats, is greater than it has been for more than fifty years since the first modern dewormers, or anthelmintics, were introduced. The familiar and somewhat depressing reason for this increased threat is the emergence and spread of resistance to these drugs, caused by overuse and misuse. With only limited prospects that affordable new compounds will reach the US market in the short to medium term, it is essential that farmers, veterinarians and agricultural advisors are aware of the current best practice in parasite management and how to use the drugs we have available today.

Adrian Wolstenholme (pictured above)

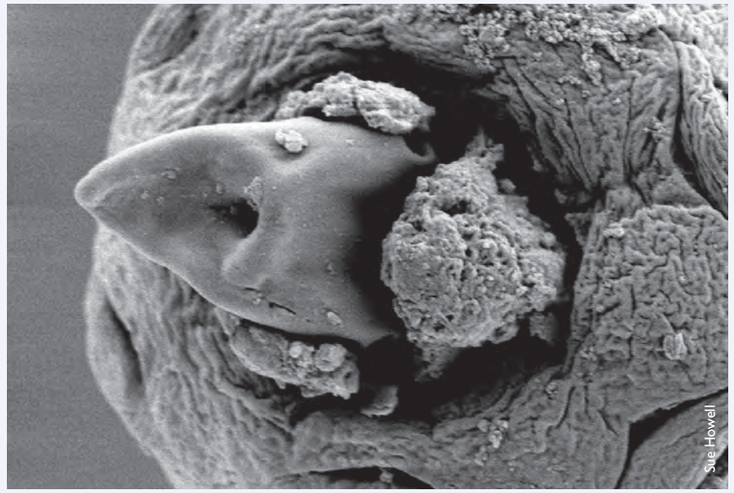
It is estimated that, left untreated, GI nematodes will steal more than 20% of the productivity of infected animals, and the more pathogenic species like *Haemonchus contortus* (goats & sheep), *Ostertagia ostertagi* (cattle) and the small strongyles (horses) can cause serious health problems and even death. For the last fifty years we have been able to control these infections using excellent anthelmintics such as thiabendazole (introduced in the early '60s), levamisole (introduced in the '70s) and ivermectin (introduced in the '80s). These drugs all had better than 90% efficacy against the common parasites, sometimes 100%, and their enormous success essentially prevented development of any new compounds for decades. In agriculture, nearly all farm animals were treated several times a year, sometimes even once a month. The consequences were, in hindsight, predictable and inevitable – the worms evolved to become resistant and the drugs became less effective. Resistance to thiabendazole was first reported in 1964 and by the mid-70s resistance to benzimidazole anthelmintics was common in nematode parasites of both sheep and horses throughout the world. The same pattern was repeated after the introduction of levamisole and ivermectin, and by the early 1990s worms resistant to all three drug classes were appearing in small ruminants.

Today, resistance is widespread to all anthelmintics on the market. Locally, a survey of resistance we performed from 2002-2006 in the southern US found resistance to benzimidazole, levamisole, ivermectin, and moxidectin on 98%, 54%, 76%, and 24%, respectively, of sheep and goat farms. Resistance to all 3 major classes of anthelmintics was detected on half the farms, and resistance to all 3 classes plus moxidectin (total anthelmintic failure) was detected on 17% of farms. Furthermore, ongoing surveillance indicates that the problem continues to worsen; 97% of goat farms we tested from 2007-2009 had resistance to ivermectin and 55% to moxidectin; 6 of 6 large goat farms in Georgia (>100 goats) tested in 2010 had resistance to both ivermectin and moxidectin. Stated boldly, anthelmintics can no longer be relied upon to control parasites of goats and this situation threatens the viability of the goat industry in the southern US.

The situation in horses is also very serious. A study we led in 2001 found extremely high levels of anthelmintic resistance on horse farms in the southern US. Small strongyles (Cyathostomins) resistant to fenbendazole, oxibendazole and pyrantel were present on 100%, 54%, and 41% of farms, respectively. Ivermectin resistance was not detected, but recent reports suggest that it is now emerging. Furthermore, resistance to ivermectin and moxidectin in roundworms (*Parascaris equorum*) of foals is highly prevalent, and reports of resistance in pinworms (*Oxyuris equi*) are increasingly common. Anthelmintic resistance in cattle parasites is not as severe, but evidence suggests the problem is rapidly escalating.

Given this rather bleak scenario, what alternatives for worm control on farms do we have? Despite considerable effort, no vaccines are available for GI nematodes, nor can we expect any in the near future. No new anthelmintics have been developed for the livestock market in the US in the last 30 years, thus we are left trying to preserve the effectiveness of a very few drugs that remain efficacious. Some new drugs are being commercialized, but these are not licensed for livestock in the US, and their approval is not guaranteed. For some years at least, we will need to use the drugs we have now in a more focused and 'smarter' way than in the past, integrating their use with other novel, non-chemical methods of control. In addition, because resistance is so prevalent, it is essential for each farm to know which drugs will be effective against the parasites present in their animals. This can be tested 'on-farm' by measuring the number of worm eggs in the feces of animals both before and after treatment, but this is very laborious. An alternative is laboratory tests for measuring drug resistance, but these are technically demanding. The Kaplan lab at UGA CVM is the only US laboratory currently offering this service (contact Sue Howell for more information).

Parasite infections are overdispersed, meaning that a small proportion of the infected animals harbor most of the worms – the majority carries a relatively light infection that has little impact on welfare or productivity. This can be exploited by using targeted selective treatments (TST) of only those animals that actually need treating. This strategy has several huge benefits; fewer treatments mean reduced drug and labor costs, plus the worms in the untreated animals are not exposed to the drugs and are not selected for resistance. These untreated parasites form a refugium of susceptible parasites, which greatly dilutes any resistant worms that are present. Thus, when animals become infected, the infecting worms are less likely to carry genes for drug resistance, and any treatment needed is more likely to be successful. A method for implementing a TST strategy in sheep and goats called FAMACHA© was developed in South Africa, and the validation and use of FAMACHA© in the US has been pioneered by the Kaplan Lab at UGA. Distribution of FAMACHA© to farmers is linked with educational programs on integrated parasite management. Since 2003, farmers in 47 states have received this training and more than 20,000 FAMACHA© charts have been distributed. The success of this program has been immense; 94% of farmers surveyed felt that it improved their ability to control parasites, 74% reported reduced parasite problems, and 88% felt that they had saved money (For more information contact Bob Storey in the Kaplan lab, Manager of the US FAMACHA© Program).



Ray Kaplan (pictured above)

Research efforts in the Wolstenholme lab are concentrating on understanding genetic changes causing parasites to become resistant. These changes can be used to develop rapid and sensitive molecular tests for resistance and, potentially, ways of reversing it. Drug resistance is often associated with a change in the drug target, or in the biochemical processes that destroy or remove it from the target animal. In parasitic nematodes, many drugs affect components of the nervous system, causing paralysis, so we are studying the receptors that regulate worm movement. Recently, we have identified the receptors targeted by levamisole in *Ascaris suum*, an important parasite of pigs with close relatives that infect humans and horses. We also identified a receptor from *H. contortus* that is directly stimulated by low concentrations of ivermectin, and is likely to be a key target of this very important drug. Knowing how the drugs work can be a first step to understanding resistance, but changes in target molecules are not the only possible mechanism. Changes in the way that parasites metabolize or transport drugs within their tissues may also cause resistance – if the drug never reaches its target then it cannot paralyze the worm. We are also looking for changes in the expression of proteins that protect parasites from toxic substances; these changes may be constitutive, present continuously, or may be induced by the drug itself. One family of drugs for which we know which genetic changes cause resistance is the benzimidazoles, the oldest of the major anthelmintic families. Unlike other anthelmintics, these drugs do not affect neurotransmission, but microtubules, part of the internal 'skeleton' inside all cells. They specifically affect worm microtubules and resistance is associated with a change in the β-tubulin protein (a major component of microtubules) that makes it more like the mammalian form and less able to bind the drugs. We have produced a molecular test for this change and shown that we can detect the resistance mutation when it is at a very low frequency within the population, before resistance is obvious as treatment or control failures. We aim to develop such tests for the other drugs on the market, with the hope that they will form an important part of future integrated control strategies for parasitic worms of livestock.

On television, once the parasite is identified treatment with the correct drug is almost always successful, often very quickly – there is a happy ending to the story. In our fields and on our farms, drug resistance is threatening such positive outcomes and with them, the future prosperity of livestock farming. We are working to introduce practical and effective ways of combating this threat, and to educate the community in their use. Only by taking a rational evidence-based approach to parasite control with sustainability as a goal can the livestock industry ensure that the few anthelmintic drugs we have available will remain efficacious into the future.

Adrian Wolstenholme & Ray Kaplan

Epizootic Papillomatosis in the Bluegill Sunfish *Lepomis machrochirus*


Alvin Camus

The objective of this project was to investigate the potential role of a viral agent as the cause of an outbreak to skin tumors, benign papillomas, in the bluegill sunfish. A high incidence of proliferative skin growths was observed in a managed population of bluegill for a period of 3 years and served as the basis for this study. Tumors occurred most commonly on the lips and infrequently on the fins and body surface. Indigenous to eastern North America, this popular gamefish is produced by private, state and federal hatcheries for stocking purposes on public and private lands.

Skin tumors are well known in fish and have been recognized by laymen and scientists for centuries. Although the causes of epizootic neoplasia in fish skin are varied and debated, electron microscopy and virological methods have demonstrated virus or virus-like particles in over half of the outbreaks examined. Tumor associated virus particles tentatively identified by electron microscopy only, include herpesviruses, adenoviruses, papovaviruses, and retoviruses. However, oncogenicity, or the ability to produce tumors, has only been clearly demonstrated for herpesviruses, most notably *Herpesvirus salmonis* type 2 and *Herpesvirus cyprini*. These viruses cause mortalities in young masou salmon *Oncorhynchus masou* and common carp *Cyprinus carpio*, respectively. Survivors develop papillomas on the lips or skin 4-12 months later. Tumor induction has been demonstrated for additional herpesviruses and two retoviruses identified by molecular means, but not isolated in culture.

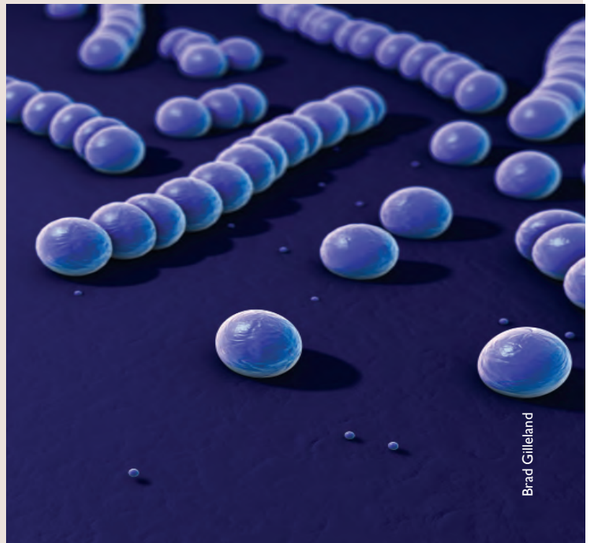
It was hypothesized that a herpesvirus was responsible for the outbreak of papillomatosis in these fish. Working with collaborators at the University of California, this hypothesis was investigated by: 1) transmission electron microscopy for the demonstration of viral particles in tumors, 2) attempted isolation in appropriate fish cell cultures, 3) use of generic herpesvirus and other viral primer sets in the polymerase chain reaction, and 4) transmission studies using cell free tumor filtrates potentially harboring infectious virus by injection and waterborne challenge in juvenile bluegills. To date, all attempts to visualize viral particles by electron microscopy or identify viral genetic sequences in tissue by molecular methods have been negative. While these initial results are disappointing, the project is on-going and fish exposed to tumor filtrates will be observed for tumor development for a period of 1 year. Identification of a viral agent would add significantly to the body of knowledge regarding oncogenic viruses in fish and potentially limit its spread in a gamefish distributed throughout Georgia.

Principal Ivestigator: Alvin C. Camus,
Co-Principal Investigators: Tom Waltzek, Ron Hedrick

Mycoplasma Research

M*ycoplasma synoviae* (MS) has been identified as the cause of recent significant production losses in the state of Arkansas, primarily due to increased processing plant condemnations and mortality related to respiratory disease. Although at least three strains of MS have been identified by *vlhA* sequencing of recent MS isolates from Arkansas, the strain that was the primary cause of the outbreak was designated "S-10". Based on field reports, it appeared that the S-10 strain is considerably more pathogenic and transmissible than other MS strains from the area. We evaluated the pathogenicity and transmissibility of an isolate of the S-10 genotype and compared it to another recent MS isolate from Arkansas of the S-17 genotype, as well as to a virulent MS reference strain (K1968). We showed that the S-10 strain was significantly more virulent and highly transmissible. The shift in MS infection from strains of low virulence that are not of major concern to the industry to strains that are highly virulent and transmissible has had a significant impact on the approach to MS control in the United States as evidenced by the introduction of a live MS vaccine. We have also conducted preliminary studies evaluating MS strains of low virulence for potential as live vaccines and evaluated challenge methods for MS trials.

PrincipaI Investigator: Naola M. Ferguson-Noel


Brad Gilleland


Chris Herron

Endosymbiotic Bacteria in the Parasitic Ciliate *Ichthyophthirius multifiliis*

The ciliate *Ichthyophthirius multifiliis* (Ich) is an obligate parasite of freshwater fish that infects epithelia of the skin and gills. Ich infects all fresh water fish and has a world-wide distribution. In closed systems, such as hatcheries, or aquaculture ponds, *I. multifiliis* infections can rapidly overwhelm naive fish and cause mortality of the entire population. Infection is initiated through invasion of the skin and gills by free-swimming theronts, which rapidly differentiate into ameboid-like trophonts that burrow into the epithelial tissues of the skin and gills. Trophonts feed on epithelial tissue leading to extensive damage of the skin and gill, and grow to 0.8 mm in diameter. At this point they are visible as white spots; hence the name, "white-spot disease". After feeding for seven to ten days, trophonts leave the fish, proceed through approximately 10 cell divisions, and differentiate into infective theronts to complete the life cycle.

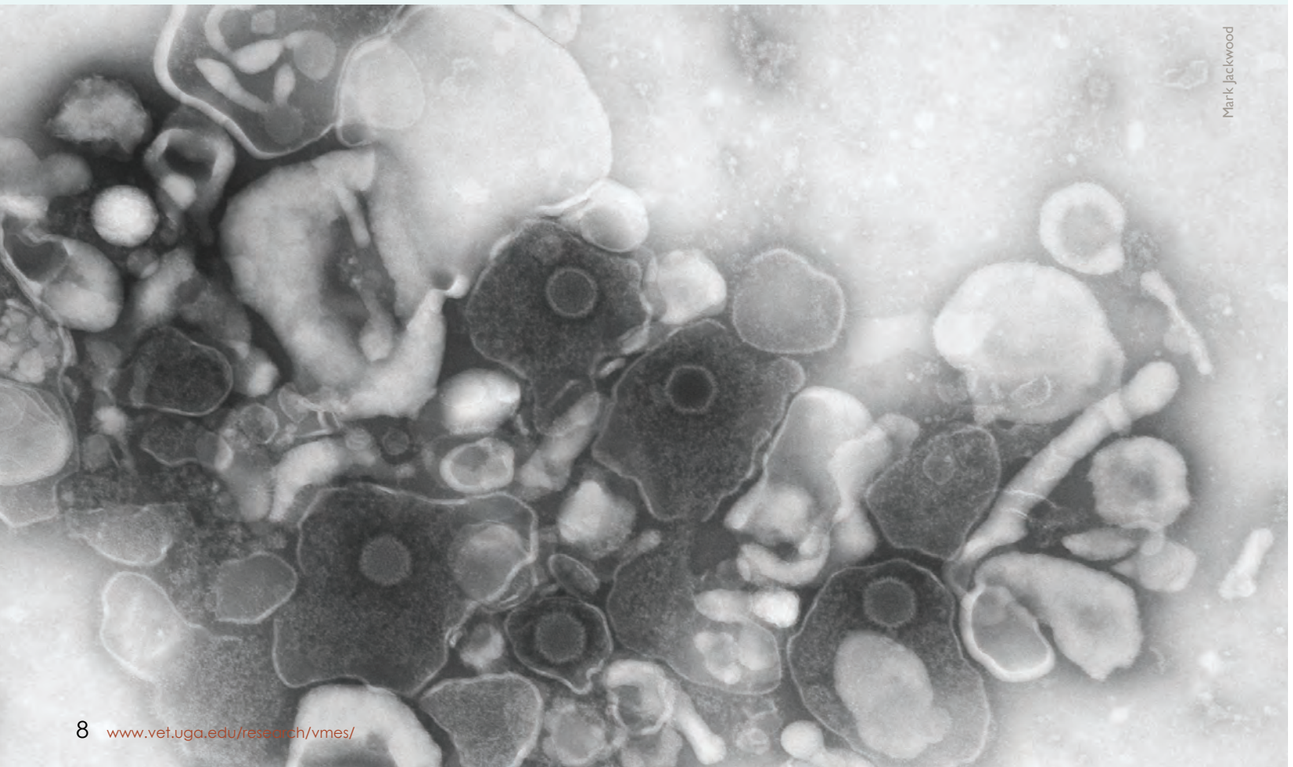
DNA sequencing of the *I. multifiliis* genome at the J. Craig Venter Institute revealed unexpectedly that bacterial DNA sequences were present in *I. multifiliis* DNA preparations. Intracellular, or endosymbiotic bacteria, are commonly found in ciliates, and this sequence data suggested that Ich also contained endosymbionts. Our subsequent sequence analysis of PCR products amplified from *I. multifiliis* DNA, using primers that bind conserved sequences in bacterial 16S rRNA genes, demonstrated that 16S rRNA gene sequences from an *Alphaproteobacteria* (*Rickettsiales*) and a *Sphingobacteria* were present. Fluorescence *in situ* hybridization showed that these bacteria are cytoplasmic endosymbionts of *I. multifiliis*. The *I. multifiliis Sphingobacteria* is a novel and previously uncharacterized species. The role that these endosymbiotic bacteria play in the life history of *I. multifiliis* is not clear, however. It is not known if the endosymbionts contribute to the growth of *I. multifiliis* or influence its pathogenicity. It also remains to be determined if they provide their host with any selective advantage, or if they influence the immune response of fish infected with *I. multifiliis*.

PrincipaI Investigator: R. Craig Findly

Development of Genetically Engineered Live Attenuated Infectious Laryngotracheitis Virus (ILTV) Strains: Knock out/Knock in System to Genetically Manipulate the Viral Genome

Infectious laryngotracheitis (ILT) is a very serious widespread respiratory disease of poultry that produces devastating losses to the chicken industry world-wide. The disease is caused by the *Gallid herpesvirus 1* infectious laryngotracheitis virus (ILTV). In the past four years, the broiler industry of the US has experienced long and costly outbreaks of the disease. Field and experimental evidence strongly indicate that the unrestricted use of live virus vaccines and poor mass vaccination practices have allowed virulent revertants of current live-attenuated ILTV vaccines to persist in the field causing severe, long lasting outbreaks of the disease. The long-term goal of this project is to develop safer live-attenuated ILTV marker vaccines that can be administered in ovo while providing robust protection. Two ILTV glycoprotein J (gJ) deletion mutants were derived from the United States standard challenge strain (USDA-ch): a gJ deletion mutant with a GFP-expression cassette inserted into the viral genome (ADgJ4.1) and a gJ deletion mutant void of any foreign DNA (BDgJ3.2). As experimental control, a gJ rescue mutant (gJR4.3) with restored gJ expression was generated and characterized *in vitro* and *in vivo.* The replication efficiency of ADgJ4.1 and BDgJ3.2 in the chorioallantoic membranes (CAMs) of 9-day-old chicken embryos was diminished when compared to USDA-ch and the gJR4.3 rescue mutant. However, titers of mutant ADgJ4.1 in CAMs increased after serial passages whereas replication of BDgJ3.2 resulted in low viral titers. Cell entry kinetics of gJ deletion mutants ADgJ4.1 and BDgJ3.2 into LMH cells were comparable to USDA-ch and gJR4.3, indicating that gJ does not play an important role during viral entry. Two-step replication kinetics were performed to determine if the absence of gJ expression influenced the *in vitro* replication of the viral mutants. While cell associated titers of USDA-ch and gJR4.3 were of the same magnitude as titers in the supernatants of infected cells, cell-associated viral titers of the gJ deletion mutants were approximately a thousand fold higher than viral titers in the supernatants. The same phenomenon was also shown for replication in embryonated chicken eggs. While titers of CAM extracts of USDA-ch or gJR4.3 infected chicken embryos were of the same magnitude as viral titers in the allantoic fluid (AF), titers in CAM extracts of ADgJ4.1 infected chicken embryos were a thousand fold higher than viral titers in the AF. From these data it can be assumed that viral egress is impaired by the lack of gJ expression. Thus, it can be speculated that gJ might be involved in viral morphogenesis and release from cells. In summary, ILTV deletion mutants were generated and characterized *in vitro.* The ability of the ILTV gJ deletion mutant ADgJ4.1 to replicate efficiently in CK cells and CAMs makes it worthwhile to further evaluate this genetically defined strain as a potential live-attenuated ILTV marker vaccine.

Principal Investigator: Maricarmen Garcia
Co-Investigators: Alice Mundt, Sylva M. Riblet, Egbert Mundt



Mark Jackwood

Michael Yabsley

Understanding the Introduction of Antimicrobial Resistance through the Pet Trade: using the Tokay Gecko (*Gekko gecko*) as a Model

We proposed the pet trade, particularly the conditions under which wildlife is imported, has the potential to influence the rate of antimicrobial resistance development. Through experimental manipulations, using the Tokay gecko as a model for the pet trade, we investigated 1) the antimicrobial resistance of *Enterobacteriacae* isolates, and 2) how the conditions by which reptiles are imported, for the pet trade, impact antimicrobial resistance.

The objectives of the project were to 1) describe the community of enteric bacteria isolated by culture from imported tokay geckos under varying housing conditions, 2) to describe the prevalence and serotypes of *Salmonella* cultured from imported tokay geckos under varying housing conditions, and 3) to describe the antimicrobial resistance patterns of commensal and potentially pathogenic bacteria of the family *Enterobacteriaceae* isolated from imported tokay geckos under varying housing conditions.

Wild Tokay geckos living in peridomestic environments were imported from their native range in Indonesia, housed individually after collection, and during import. We collected fecal samples while individually housed, and then placed animals in four treatments groups of varying densities (control, low, med, high) and collected feces again. Using standard microbiology techniques, we cultured lactose fermenting bacteria in the family *Enterobacteriacae* and determined the phenotypic antimicrobial resistance using Minimum Inhibitory Concentration (MIC) plates.

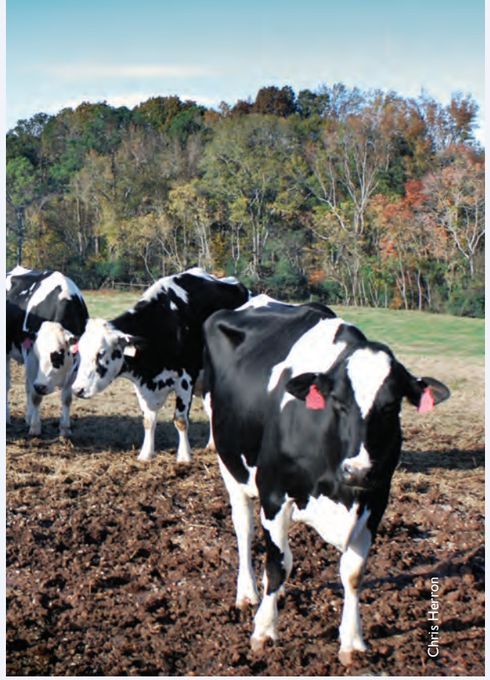
Our data demonstrated that: 1) the community composition of *Enterobacteriacae* shifted such that some genera, which predominated in individually housed or low density groups were replaced by other genera in the high density groups, 2) the prevalence of antimicrobial resistance of commensal bacteria isolated from varying density groups was usually less than that of individually housed geckos, 3) the prevalence of *Salmonella* sp. isolated increased from 40% in the groups of individuals to 58% in groups of the geckos with higher densities, 4) in addition to serogroups associated with reptiles, *Salmonella* sp typically associated with livestock (Serogroups D, E, F) represented 41% and 45% of the isolates cultured from the two groups of individuals and 10.5% of the isolates from geckos living in low and high densities.

A surprising number of *Citrobacter* sp. and *Enterobacter* sp. isolates showed intermediate resistance to chloramphenicol, to which these bacteria should be susceptible. Approximately 14% of the *Klebsiella sp*. isolates were resistant to tetracycline, which is contrary to previous studies. Three isolates of *Enterobacter* sp., one isolate of *Klebsiella* sp. and one isolate of *Citrobacter* sp were resistant to streptomycin. While this drug is infrequently used in humans in the USA, it is commonly used in Indonesia, and is a common veterinary drug worldwide. Two isolates of *Enterobacter* sp., and two isolates of *Citrobacter* sp. were resistant to multiple antibiotics.

Conclusions: We concluded that tokay geckos could be used as a model to demonstrate that the stressful conditions under which reptiles are imported for the pet trade can result in a shift in fecal bacteria community composition (which may directly affect their health), an increase in shedding of pathogenic bacteria in these animals (ie; *Salmonella*), and the introduction and development of antimicrobial resistance. These findings could prove to be extremely significant for people who purchase these animals as pets, often for children. The US currently imports more than 200 million live animals for the pet trade per year and almost 70 % of wildlife imported into the US for the pet trade originates from Southeast Asia.

Principal Investigator: Sonia Hernandez

Establishing the Roles of Local and Systemic Inflammation in Reproductive Failure in Dairy Cows



The dairy industry has the problem that many cows are not easy to get pregnant. To have a profitable dairy business, each cow must reliably become pregnant within about 3-4 months after her calf is born. This provides for optimal milk production and the best use of input resources. Unfortunately, even with modern tools to enhance ovulation and the use of artificial insemination, many dairy cows require many attempts at establishing pregnancy to achieve the goal. It appears that most of these cows ovulate under the protocol and that the semen is good. What appears to be the problem is that the developing blastula does not effectively implant in the uterine wall and allow the pregnancy to develop. Prior work indicated that the presence of cells associated with inflammation, neutrophils, in uterine mucus indicated a poorer chance of successful pregnancy.

In this study, we flushed the uterus of 32 cows with sterile saline and evaluated the flush fluid for the presence and number of neutrophils and other white blood cells, for the growth of coliform bacteria, for the growth of common pathogens, for the level of IgG and IgA antibodies, and for the level of prostaglandin E2. We found that cows had a wide distribution of these measurements in uterine flush fluid. They ranged from no white cells to many, low levels of antibody to very high, and low levels of prostaglandin to very high levels. We found a clear correlation among these values, in that those cows with high numbers of uterine white blood cells also had high levels of uterine IgG and IgA. These same animals had the highest levels of prostaglandin E2. Many of these same cows had detectable numbers of bacteria that are associated with uterine infections.

We have sampled a number of cows that did not become pregnant after several tries a second time. All of these cows had high levels of IgG antibody, high levels of prostaglandin E2, yielded bacterial isolates consistent with pathogens, and most had white blood cells in the uterine flush sample. Our working model for how these immune and inflammatory factors impact pregnancy is that infectious agents colonize the uterus after birth and establish an ongoing inflammatory and immune response. We plan to assess the specificity of the antibodies in uterine mucus in an attempt to determine which pathogens are associated with the immune and inflammatory response.

Principal Investigator: David J. Hurley
Co-Investigators: Michael W. Overton, Jakob Scherzer, Isaiah J. Smith and Stephen C. Nickerson

Avian salmonellosis among wild birds: Strain specificity and natural history

We have diagnosed avian salmonellosis among 11 species of passerine birds. Pine siskins and gold finches are the most numerous of our accessions, particularly during periods of population incursions in the southern United States. Northern Cardinals are the next most numerous species of those associated with feeders. Various species of blackbirds are also commonly affected but the epidemiologic pattern focuses on outbreaks among large flocks of usually single species in agricultural settings as opposed to the mixed flocks of songbirds associated with feeders.

We examined 92 passerine isolates by PFGE (Pulsed Field Gel Electrophoresis). PFGE clonal group A represented 86% of the avian isolates. Subtypes of this group showed a strong association with avian species. Type A2c was responsible for the majority of cases involving pine siskins and American goldfinches. Type A1c was closely associated with disease in Northern cardinals, even when outbreaks were nearly simultaneous with mortality among siskins and goldfinches. We continue to assess prevalence of *Salmonella typhimurium* from birds in backyard settings. This year we have seen no mortality but have sampled birds from a variety of taxa. We were able to collect 1,118 samples including 129 from birds previously sampled. These are currently being analyzed by culture and PCR to assess prevalence in birds in the absence of clinically apparent disease.

Two of the most common PFGE types were also identified among human isolates described in the CDC PulseNet database. Type A1c was matched 511 PFGE patterns of the total 61,594 *S. typhimurium* isolates in the CDC database. Type A2b, matched the PFGE patterns of 196 of the human isolates.

Principal Investigator: Kevin Keel

Effect of BRSV-Infected Bovine Bronchial Epithelial Cells on T Cell Responses

In a variety of species, disease syndromes are recognized that are caused by co-infection by viral and bacterial pathogens with morbidity that is more severe or persistent than that caused by the individual pathogens alone. The mechanisms by which viral-bacterial co-infection leads to enhanced disease are poorly characterized for most recognized pathogen combinations in all species. In cattle, a preeminent syndrome caused by viral-bacterial co-infection and leading to extensive morbidity and mortality is the Bovine Respiratory Disease Complex (BRDC). The BRDC is known to be caused by co-infection by viral and bacterial pathogens, and exacerbated by a variety of management practices. Viral infection is nearly always the primary infectious insult in the BRDC, with subsequent infection by opportunistic bacterial organisms which exacerbate the resulting lung pathology. Although much research has been carried out to determine the means by which single pathogens contribute to the morbidity and mortality of the BRDC, very little is known about the mechanisms by which viral and bacterial pathogens interact to cause this disease.

Epithelial cells which line airways are the first line of defense against respiratory infection. Bronchial epithelial cells (BEC), which have long been recognized to provide a mechanical barrier to respiratory infection, have in recent years been identified as key players in the initiation of the host immune response to respiratory infection. BEC express many of the same cell surface markers and cytokines as dendritic cells and other "professional antigen presenting cells" that activate naïve T helper (TH) lymphocytes and direct their differentiation into TH effector subtypes that influence the outcome of many infectious diseases, or which may drive a harmful immunopathologic response. Recently, we have developed a primary cell culture system that allows culture bovine bronchial epithelial cells (BBEC) from living cattle. Utilizing bronchial brushings of epithelial cells and an air/liquid interface (ALI) culture system, we can successfully grow primary bronchial epithelial cells in their fully differentiated state into secretory and ciliated cells which reflect the mucociliary structure and function of differentiated epithelium as seen in vivo. This system provides our laboratory the opportunity to investigate the influence of airway epithelial cells on the outcome of infectious bovine respiratory disease by revealing the pathways operative in the earliest phase of respiratory infection and provides a platform for evaluation of treatments to mitigate BRDC through modulation of inflammatory mediators expressed by the airway epithelium.

The most promising treatments identified with this *in vitro* system can then be selected for *in vivo* confirmation. The validation of this model of interaction between BBEC and cells of the immune system will support our long-term goal to discover how airway epithelial cells influence the immune response of cattle to polymicrobial respiratory infection, and to determine how these responses can be modulated to improve the resistance of cattle to respiratory disease.

Principal Investigator: Thomas Krunkosky
Co-Investigator: Amelia Woolums

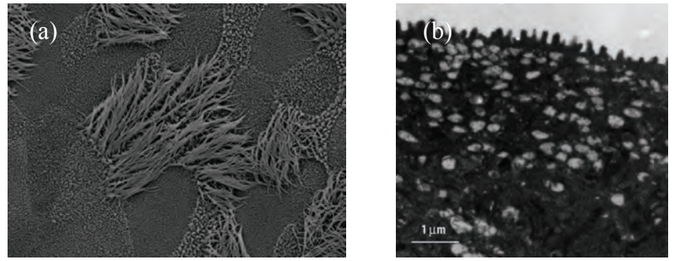


Figure 1: Scanning electron micrograph of normal bovine bronchial epithelial (BBEC) cells grown in the air-liquid interface system and differentiating media showing both (a) ciliated cells by scanning electron microscopy and (b) goblet cells by transmission electron micrograph illustrating goblet cells containing electron-dense "cores"

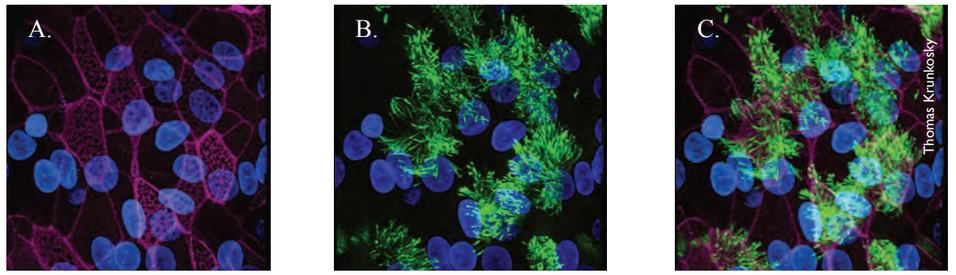


Figure 2: Confocal of normal bovine bronchial epithelial (BBEC) cells grown in air-liquid interface system, showing:A. cellular archecture (Purple, F-actin) with nuclear detail (blue, DAPI), B. ciliated cells (green, beta tubulin) with nuclear detail (blue, DAPI), C. merged panels.

Evaluation of immunization strategies in broiler breeders for reducing the economic impact of RSS on commercial broilers

Worldwide outbreaks of Runting Stunting Syndrome (RSS) have been reported in broiler companies with an intense production program. The outbreaks are seasonal and occur in the winter and spring. RSS is a transmissible disease of the digestive tract affecting young broilers between 1-2 weeks of age. Despite reports of RSS dating back to the 1970s, an etiology has yet to be identified. The disease causes severe weight suppression during the first few weeks of age, lack of flock uniformity, diarrhea, distention of the intestines, and an increased feed conversion. Cystic enteropathy is consistently observed by histopathological examination of small intestines from affected chickens.

To study RSS, a reliable challenge model was established as a prerequisite for vaccine development. The clinical disease was reproduced in broilers using filtered intestinal homogenates from clinically affected broilers, thus implying viral etiologic agents. Several viruses were isolated and used in experimental challenge studies. While a marked decrease in body weights in virus challenged groups were observed compared to non-challenged groups, the clinical disease (decreased body weights and cystic enteropathy) was not reproduced. Using Metagenomics, four different viruses which might be involved in the disease were identified by applying a subtractive approach with bioinformatic tools. Since most of the enteric viruses cannot be isolated in vitro, the protective immunogens were expressed in a recombinant baculovirus system. The recombinant proteins were purified and used for the establishment of ELISA systems. To verify that viruses which encoded for these proteins might be involved in RSS, specific-pathogen-free chickens were infected with the filtrate and the immune response was analyzed. The data showed that three of the four proteins were detected specifically in the indirect ELISA from the serum of infected chicken while the non-infected controls showed no reactivity. Furthermore, experiments using in situ hybridization showed clearly that three of the four viruses were involved in the etiology of the disease. The hybridization probes used in these studies did not show any signal and this correlated with a lack of immune response for the same virus in an indirect ELISA. In parallel studies, broiler breeders were vaccinated with one recombinant protein for each group. Subsequent challenge experiments with the offspring showed no significant protection. The next experiments will be performed in broiler breeders that will be vaccinated using a cocktail that consists of the four recombinant proteins. The offspring will be challenged using our challenge model.

Principal Investigator: Egbert Mundt
Co-Investigator: Holly S. Sellers

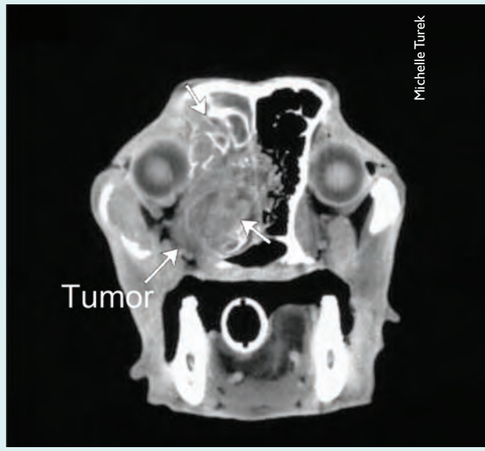
Role of Class A Scavenger Receptors in Response to Mycobacterial Trehalose Dimycolate and Mycobacterium Tuberculosis Infection

Mycobacterium tuberculosis (Mtb), the causative agent of tuberculosis (TB), infects macrophages, a type of white blood cell that is critical in the initial response to bacterial infections. After being taken up by macrophages, Mtb manipulates this cell to inhibit the normal pathway of bacterial degradation. The process by which this occurs is still unclear. One of the Mtb cell wall components, trehalose dimycolate (TDM), has been shown to interfere with the degradation pathway when presented to macrophages on the surface of bacteria-sized particles. Previous work from our laboratory has shown that TDM binds to a type of receptor on macrophages called MARCO, a class A scavenger receptor. Because MARCO is specifically expressed on the surface of specific macrophage populations of importance to TB, such as those in the lungs, we believe that this binding interaction is likely to be significant in the pathogenesis of TB.

To determine the role of MARCO and other class A scavenger receptors in the response to Mtb and other mycobacterial species, such as *M. bovis* Bacille Calmette-Guerin (BCG), the strain of *M. bovis* that is used in many countries for vaccination against TB, we have a colony of mice deficient in these receptors. Macrophages from these mice can be studied *in vitro* after infection with mycobacterial species or stimulation with TDM-coated particles and compared to responses by macrophages from wild-type mice in order to determine the specific role of these receptors. Published data from our laboratory have shown that macrophages from deficient mice have impaired cytokine responses to TDM and Mtb infection. These mice are also a useful animal model for intratracheal infection, and preliminary results have shown that early pathologic lesions in response to infection are more severe in the mice that lack these receptors than in the wild-type mice.

Principal Investigator: Kaori Sakamoto

Validation of Antibodies and qRT-PCR Primers Required to Assess Epidermal Growth Factor Receptor Expression in Canine Tissue



Epithelial cancers represent a major disease entity in dogs, and their treatment remains a significant challenge. Novel therapies are desperately needed. The epidermal growth factor receptor (EGFR) is a cell membrane bound tyrosine kinase receptor that plays an important role in cell signaling to promote cell growth and survival. Abnormalities in EGFR are found in many human cancers and have been implicated in the pathogenesis of these diseases. Strategies to target EGFR have been developed and have been shown to be beneficial in certain human tumors with aberrant EGFR expression. Despite these advances in the treatment of human cancer, little is known about the role of EGFR in normal canine tissue and in canine cancer.

In order to investigate the role of EGFR in canine neoplasia and its relevance as a therapeutic target, the objective of this study was to perform the necessary validation steps for canine EGFR protein detection using commercially-available non-canine antibodies, and gene expression analysis. In doing so, we examined the profile of EGFR expression in a variety of healthy canine tissues. Knowledge of the role of EGFR in normal tissues will establish a baseline against which EGFR expression in canine tumors can be compared in future studies, and will inform the understanding of adverse events associated with EGFR-targeted therapies.

Western blot analysis of banked normal canine tissue showed that three of the four anti-human EGFR antibodies that were evaluated are specific for canine EGFR. EGFR expression was documented in a variety of canine epithelial tissues known to be susceptible to the development of neoplasia including skin, mammary tissue, bladder, prostate, and lung. Faint EGFR expression was identified in liver, lymph node and spleen. Skeletal and cardiac muscle showed no identifiable expression by western blot. Use of the antibodies for immunohistochemical detection of canine EGFR has proven challenging due to non-specific and/or poor antigen binding in formalin-fixed tissues. RT-PCR was performed and produced an appropriately sized amplicon of 76 base pairs in lung and bladder suggesting successful identification of EGFR mRNA in these tissues. Validation of a real-time RT-PCR protocol for relative quantitative assessment of canine EGFR gene expression is ongoing.

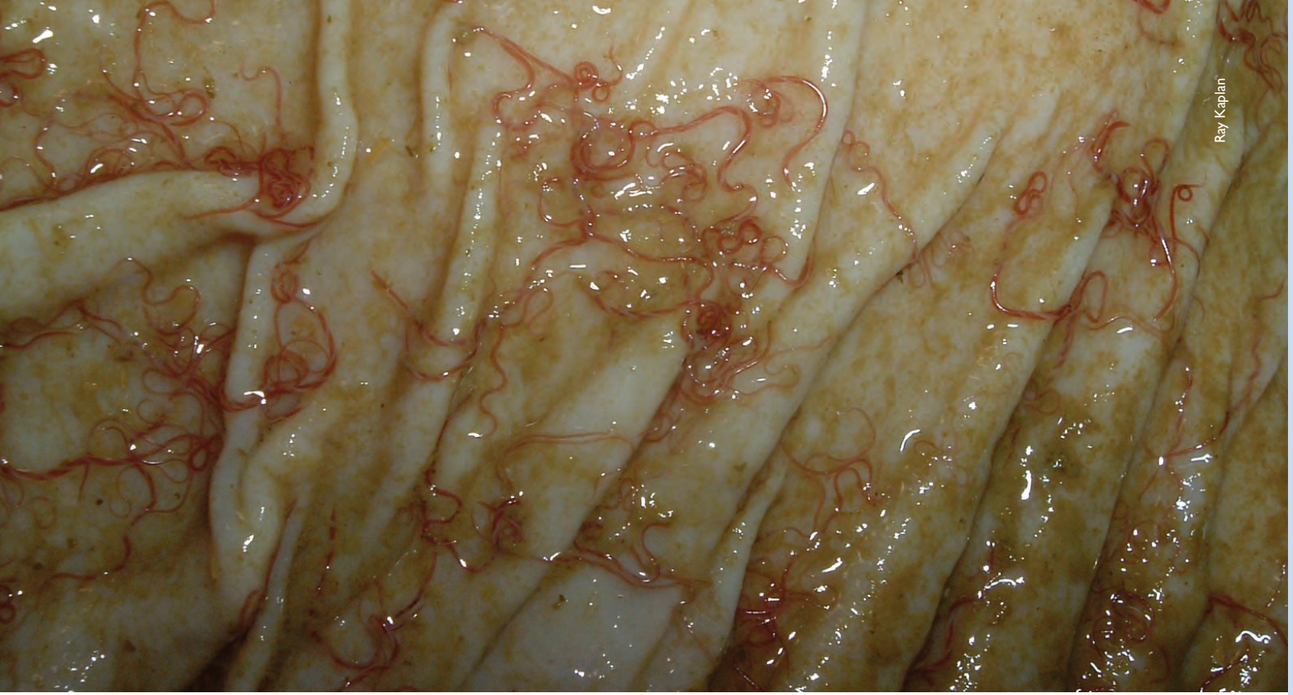
This study is laying the ground work for future investigations which will aim to describe EGFR expression in canine tumors and its relevance as a therapeutic target.

Principal Investigator: Michelle Turek
Co-Investigators: Corey Saba, Michel Vandenplas, Jessica Lawrence, Carla Jarrett

Use of retroviral vectors for *in vitro* expression of immunogenic proteins of chicken infectious anemia virus

The primary goal of this project was to express immunogenic proteins of chicken infectious anemia virus (CIAV) and other fastidious poultry pathogens in cell lines of avian origin. The first required and accomplished step was to isolate and clone non-defective endogenous avian leukosis viruses to be used as vectors for gene delivery. Four of such viruses were isolated and three were fully sequenced. Their complete genomic sequences were deposited in the public database GenBank. One of such viruses has been cloned into a plasmid vector to be used as an infectious clone for gene delivery. Subclones of this infectious non-defective ALV clone were produced and were used for gene delivery and expression using reverse genetics techniques. An additional infectious clone of a non-defective ALV was used to deliver foreign genes using the same approach. Transfection and co-transfection studies demonstrated that our approach is successful for gene delivery and expression. Green fluorescent protein (GFP, a reporter protein), was expressed *in vitro* in the DF-1 avian cell line. Viral protein-coding genes of chicken infectious anemia virus (CIAV), infectious laryngotracheitis virus (ILTV), and infectious bursal disease virus (IBDV) were cloned into our endogenous ALV plasmid vector system. Transfection experiments resulted in the successful expression of ILTV proteins in vitro. ILTV protein-expressing DF-1 cells were used to vaccinate SPF chickens, which produced antibodies against ILTV. These experiments demonstrated that our system can be used for production of immunogenic proteins and that such proteins can stimulate an immune response. Our most important long-term objective is to develop a system for production of poultry vaccines that do not require the use of chicken embryos, and a system that would be suitable for production of immunogenic proteins from fastidious poultry pathogens.

Principal Investigator: Guillermo Zavala
Co-Investigators: Taylor Barbosa, Ying Cheng



Candidate ivermectin resistance genes in *Haemonchus contortus*

Haemonchus contortus is a gastrointestinal nematode parasite of small ruminants (sheep and goats) which is prevalent in the South-East United States. Haemonchosis is treated by drenching animals with anthelmintic drugs, including ivermectin and moxidectin, but parasites resistant to these drugs are becoming increasingly common. Efforts to identify genetic changes associated with ivermectin resistance are complicated by the genetic variability of these parasites. This makes it difficult to identify the specific differences between parasite populations that cause the drug resistance phenotype. We therefore studied an inbred strain of *Haemonchus contortus,* deliberately selected to be ivermectin resistant in only three generations. We examined a panel of candidate resistance genes to see if there were any changes associated with the acquisition of resistance. One fascinating observation was that the ivermectin-resistant worms carried genetic changes associated with resistance to a completely separate class of anthelmintics, the benzimidazoles.

The chief candidate resistance genes we focused on were the P-glycoproteins. These proteins are efflux pumps which actively remove foreign compounds from cells, and increases in P-glycoprotein activity have been implicated in drug resistance, not only in parasites but also in human and animal tumors. We searched the incomplete genome sequence database of *Haemonchus contortus* for sequences that looked like P-glycoproteins, and confirmed that they represented real genes by RT-PCR amplification. Nine potential P-glycoprotein genes were identified; these were most similar to *pgp-1*, *pgp-2*, *pgp-3*, *pgp-4*, *pgp-9*, *pgp-10*, *pgp-11*, *pgp-12*, and *pgp-14* of the model worm, *Caenorhabditis elegans*.

We then compared the levels of these P-glycoprotein mRNAs in the ivermectin-resistant and -susceptible parasites using quantitative real-time PCR. Quantitative PCR reactions were optimized for all the P-glycoprotein mRNAs, and the transcript abundance was measured relative to the expression level of actin, a gene unrelated to drug resistance which is expressed at similar levels in both resistant and susceptible parasites. The findings of these experiments did not find consistent changes in the expression of any P-glycoprotein genes in the resistant parasites, but it remains possible that these genes may be upregulated in the short-term in direct response to ivermectin. Although this project has not identified the cause of ivermectin resistance in this particular isolate of *H. contortus*, it has optimized a robust assay for P-glycoprotein expression levels in *Haemonchus contortus*, which we can use to further investigate the involvement of these genes in drug resistance.

Principal Investigator: Adrian Wolstenholme
Co-Investigator: Sally Williamson

Factors Interfering with the Control of Infectious Laryngotracheitis (ILT): Vaccine Interference, Persistence of ILTV in the Field, and Relative Virulence of Recent ILTV Isolates.

The principal objectives of this research included (not exclusively); a) determination of the influence of simultaneous vaccination against ILTV and IBV (Infectious Bronchitis Virus) and/or NDV (Newcastle Disease Virus) on protection vs. ILTV; b) comparison of the virulence of a reference ILTV (USDA challenge strain) and a field strain of ILTV currently circulating in North Georgia (PDRC 63140); and c) a survey of the North Georgia poultry industry to determine possible undetected ILTV circulating in the field.

Several experiments were designed and conducted to examine protection against ILTV when broilers were vaccinated with ILT vaccine alone or in combination with IBV and/or NDV vaccines. TCO and CEO virus titers in the trachea of vaccinated broilers were reduced 3-5 days post-vaccination when ILT vaccine was given in combination with IBV and/or NDV vaccines. However, protection against ILTV was compromised only in the case of TCO vaccine combined with NDV and/or IBV vaccines. Protection against ILTV was not compromised when CEO was given in combination with IBV and/or NDV vaccines. Although chickens were not challenged experimentally with IBV or NDV, the concentration of such vaccine viruses in the trachea was reduced when given in combination with ILT vaccines, particularly with CEO.

A comparison of the virulence of a reference ILTV (USDA challenge strain) and a recent field strain of ILTV was done by using a biological titration in unprotected broiler chickens. The Georgia field strain 63140 induced clinical signs at an earlier age in comparison with the USDA ILTV strain; clinical signs were more severe in broilers infected with the field strain 63140 in comparison with the USDA challenge strain; when administered at a low dose, the field strain 63140 persisted in the trachea longer than the USDA challenge train, and longer than in broilers receiving a high dose of the same virus. These results suggest that under certain circumstances, the USDA challenge strain may not be optimal for vaccine performance evaluations. In addition, our results confirmed indirectly the importance of vaccinating chickens with a high titer vaccine to prevent prolonged persistence of vaccine virus in the trachea.

Examination of the persistence of ILTV in the field was originally intended using molecular methods in vaccinated and non-vaccinated flocks within and outside ILT vaccination zones. Increased cost of reagents and the cost of *in vivo* trials forced us to utilize a serological approach. All (100%) of the broiler flocks sampled for AI monitoring and processed during one week in the Spring of 2009 were tested to detect antibodies against ILTV. Such broiler flocks represented all Georgia broiler flocks being processed in a single week during the Spring of 2009, at a time when the prevalence of clinical ILT in broilers was significant in North East Georgia but not in the rest of the state. A second similar testing involving 100% of the broiler flocks being processed in Georgia during one week of December 2009 was conducted. Our results reflected the field situation in the spring of 2009; that is, ILTV antibodies were detected exclusively in broilers raised in the ILT-affected zone, with the exception of one area in North West Georgia where a few flocks tested positive.

Principal Investigator: Guillermo Zavala
Co-Investigator: Maricarmen Garcia

Extramural Contracts & Grants

Allen, Sheila. FY2010 Animal Health Formula Grant. USDA-NIFA. $69,291
Allen, Sheila. National Animal Health Laboratory Network: GA. USDA-CSREES. $298,000
Barton, Michelle. Hydrocortisone therapy in septic foals. American Quarter Horse Foundation. $3,064
Barton, Michelle. Hydrocortisone replacement therapy in septic foals. NIH. $59,810
Brainard, Benjamin. Assessment of a bench-top coagulation analyzer for evaluation of dogs with abnormal coagulation times. Industry Sponsor. $4,890
Brainard, Benjamin. Sonoclot evaluation of whole blood coagulation and single and multiple dose subcutaneous heparin therapy in healthy adult dogs. Veterinary Emergency & Critical Care Foundation. $9,393
Brainard, Benjamin. The Effect of Aspirin and Clopidogrel on Equine Platelet Function and Serotonin Release. Morris Animal Foundation. $48,484
Braun, Christina. Assessment of maxillary and infraorbital nerve blockade for rhinoscopy. Industry Sponsor. $2,551
Brown, Corrie. Consortia for Promotion of Understanding of Environmental and Public Health Issues. FIPSE DOE (Sub-award under Michigan State University). $10,800
Brown, Corrie. Early events of vesicular stomatitus virus infection in cattle: Comparison of fly-bite with scarification. USDA-ARS-NAA. $13,089
Brown, Corrie. East African Regional Workshop on Transboundary Diseases and Impacts on Trade. USDA. $144,348
Brown, Scott. Residency in Small Animal Surgery. Western College of Veterinary Medicine. $96,942
Budsberg, Steven. Near infra-red spectroscopy to reduce prophylactic fasciotomies for and missed cases of acute compartment syndrome in soldiers injured in OEF/OIF. DOD (Sub-award under True Research Foundation). $433,249
Chen, Shiyou. Smad2 and smooth muscle differentiation from neural crest cells. NIH. $612,263
Corn, Joseph. Exotic Arthropod Surveillance in the Southeastern United States and Puerto Rico. USDA-APHIS-Veterinary Services. $300,000
Corn, Joseph. National Feral Swine Mapping System. USDA-APHIS-Veterinary Services. $25,221
Corn, Joseph. Risk of environmental transmission of Johne's disease for endangered Florida Key deer. Piedmont CESU. $129,915
Dickerson, Harry. The University of Georgia 2010 Veterinary Scholars Program: A Research Training Experience for Veterinary Medical Students. Merial Limited. $20,000
Divers, Steve. Drug-induced liver disease in common sliders (trachemys) and correlation with hematology, biochemistry, and endoscopic liver biopsy. American Association of Zoo Vets & Mazuri. $6,000
Divers, Steve. Internal sonic/acoustic transmitter implantation in sturgeon (Acipenseridae). USDOI Fish and Wildlife Service. $18,659
Epstein, Kira. Pharmacodynamics and monitoring of multi-dose low molecular weight heparin therapy in healthy horses. American College of Veterinary Surgeons. $9,908
Filipov, Nikolay M. Role of inflammation in Manganese Neurotoxicity: Molecular Mechanisms. NIH. $358,812
Fischer, John. Investigation of and assistance with wildlife disease problems in the SE region of the United States. U.S. Dept. of Interior. $247,527
Fischer, John. Relationships that may simultaneously involve wildlife, domestic livestock, and poultry. USDA-APHIS-Wildlife Services. $350,000
Fischer, John. Southeastern Cooperative Wildlife Disease Study. Other States - Tennessee. $17,760
Fischer, John. Southeastern Cooperative Wildlife Disease Study. Tennessee Wildlife Resources Agency. $5,431
Fischer, John. Southeastern Cooperative Wildlife Disease Study. Various Other States. $308,298
Fischer, John. USDA/APHIS Wildlife Services Disease Training. USDA-APHIS-Wildlife Services. $150,000
Fu, Zhen. Developing avirulent rabies virus vaccines. NIH. $289,395
Garcia, Maricarmen. Full length genomes of infectious laryngotracheitis virus strains and live attenuated vaccines: relevance in the design of recombinant ILTV vaccines. US Poultry and Egg Association. $108,215
Giguere, Steeve. Immune responses to a killed adjuvanted vaccine. Do foals respond like adult horses? Morris Animal Foundation. $35,272
Giguere, Steeve. Pulmonary disposition of a proprietary compound in foals. Industry Sponsor. $19,692
Giguere, Steeve. Pulmonary disposition of a proprietary compound in foals and effect of the method of injection on the pharmacokinetics of the drug. Industry Sponsor. $7,650
Harn, Donald. Effect of Helminth Infection of HIV-1 Vaccines. NIH. $626,636
Harn, Donald. Immune Responses to Schistome Egg Antigens. NIH. $719,981
He, Biao. AKT as a target to anti-RSV therapy. NIH. $126,133
He, Biao. Paramyxovirus as Oncolytic Agent. NIH. $92,676
He, Biao. Pathogenesis of Mumps Virus. NIH. $103,842
Hernandez, Sonia. Kitty Cams: A window into the world of free-ranging cats. Morris Animal Foundation Veterinary Scholars Program. $4,000
Hines, Murray E. Classical Swine Fever Surveillance. USDA. $6,600
Hines, Murray E. Diagnostic services relative to the control, diagnosis, treatment, prevention and eradication of livestock 2010. GA Dept of Agriculture. $1,875,040
Hines, Murray E. NAHLN Member Laboratory 2010 TVDIL. USDA-APHIS-VS. $55,000
Hogan, Robert J. Production of recombinant influenza HA proteins. Industry Sponsor. $41,684
Hondalus, Mary K. Nanoparticle delivery of experimental tuberculosis vaccines. Harvard University. $101,688
Hondalus, Mary K. Virulence of the Opportunistic Pathogen Rhodococcis Equi. NIH. $400,513
Howerth, Elizabeth. Pfizer Anatomic Pathology Fellowship for a Minority. ACVP/STP.$144,193
Howerth, Elizabeth. Gill Epithelial Nuclear Hypertrophy in the Soft-shell clam (Myaarenaria). Morris Animal Foundation. $29,352
Hurley, David. Impact of maternal cells, antibody/cytokine or their combination in preventing disease after BVDV type 1b challenge in 4-day old dairy calves. Industry Sponsor. $100,112
Hurley, David. Microbial ligand regulation of Toll-like receptor expression in isolated equine monocytes. Morris Animal Foundation. $34,497
Hurley, David. Streptococcus uberis Vaccine Candidate Assessment Project. Industry Sponsor. $177,701
Jackwood, Mark. Multiplex detection of avian influenza HA and NA types using microsphere assay. USDA (Sub-award under University of Maryland). $96,129
Jackwood, Mark. Studying IBV Hatchery Vaccination and its Effect on Field Boost in Broilers. US Poultry & Egg Association. $63,848
Johnston, Spencer. Evaluation of test drug in a urate crystal model in dogs. Industry Sponsor. $143,805
Kaplan, Ray. Animal Models of Infectious Diseases. NIH. $25,000
Kaplan, Ray. Antiparasitic Efficacy of a Novel Functional Food Compound Using an Ascaris suum Model in Pigs. Industry Sponsor. $438,787
Kaplan, Ray. Dose Determination Study for Testing the Antiparasitic Efficacy of a Novel Functional Food Compound Using a Pig Model. Industry Sponsor. $195,765
Kaplan, Ray. Filariasis Research Reagent Resource Center. NIH. $486,118
Kaplan, Ray. Furnish Brugia malayi adult worms and/or B. Malayi infective larvae. NIH. $369,652
Kaplan, Ray. Sustainable Control of Gastroinstestinal Nematodes in Organic and Grass-fed Small Ruminant Production Systems. USDA-ARS. $7,650
Keel, Kevin. The Propagation and Decontamination of White-nose Syndrome (WNS) in the Environment. Northern Kentucky University. $43,001
King, Christopher. VCA: A monoclonal antibody toolkit for functional genomics of plant cell walls. NSF-National Science Foundation. $47,580
Lafontaine, Eric. Glanders Vaccine Development. University of Calgary. $37,473
Lee, Margie. Nutritional Quality of Meat and Bone Meal. USPEA (Sub award under Auburn University.) $16,231
Lee, Margie. Revealing the Mechanisms of Competitive Exclusion of Enteropathogens from the Intestinal Microbial Community. USDA-CSREES. $399,154
Mead, Daniel. Vector-Borne Disease Surveillance Chatham County. Chatham County. $37,950
Mead, Daniel. Vector-Borne Disease Surveillance-Fulton County. Clarke Mosquito Control. $3,300
Mead, Daniel. Vector - Borne Disease Surveillance-Dekalb County. DeKalb County. $9,900
Mead, Daniel. Vector-Borne Disease Surveillance Wild Bird and Mosquito Diagnostic Support. GA Dept of Human Services. $30,000
Moore, James. Differential Responses of Equine Monocytes and Neutrophilis to Microbial Ligands. Morris Animal Foundation. $73,549
Moore, James. Elucidating structure-function relationships of lipid A: A synthetic approach. NIH. $59,000
Moore, James. Learning Biological Processes Through Animations and Inquiry: A new Approach. NIH. $118,390
Moore, Julie. Immunopathogenesis of Severe Malaria During Pregnancy. NIH. $303,466
Moore, Julie. Trophoblast Immune Responses to Placental Malaria. NIH. $222,750
Mundt, Egbert. Development of a species-independent ELISA for detection of influenza A antibodies directed against H6/H7/H9. USDA (Sub-award under University of Maryland). $65,834
Mundt, Egbert. Proposal for research and development of intervention strategies to reduce or prevent economic losses from Runting-Stunting-Syndrome in chicken. GA Poultry Federation. $39,000
Mundt, Egbert. Testing of Components to mitigate Runting Stunting Syndrome in chicken in a challenge model. Industry Sponsor. $39,985
Northrup, Nicole. Maddie's Shelter Medicine Externship. Maddie's Fund. $6,000
Pence, Melvin. Georgia Johne's disease Demonstration Herd Project. USDA. $60,977
Peroni, John. Use of bioresorbable hydrogels and genetic engineering to accomplish rapid stabilization and healing in segmental long bone effects. DOD (Sub-award under Baylor College of Medicine). $319,676
Peroni, John. Osteogenic Differentiation of Marrow and Adipose Tissue Derived Equine Mesenchymal Stem Cells. Morris Animal Foundation Veterinary Scholars Program. $4,000
Rajeev, Sreekumari. Isolation and Characterization of Leptospira. Industry Sponsor. $19,000
Ritchie, Branson. Research Associate in Exotic/Zoo Infectious Disease and Pathology Postgraduate Program. Riverbank Zoo. $13,000
Robertson, Thomas. Vagal and glossopharyngeal afferent cell bodies as biological sensors. NIH (Sub-Award under University of Virginia). $134,439
Saba, Corey. Use of indocyanin green (ICG) and an intraoperative spectroscopy imaging system (ISIS) to determine tumor resection boundaries in dogs with soft tissue sarcomas. Emory University. $11,500
Saliki, Jeremiah T. Avian influenza, Exotic Newcastle, Scrapie Testing and Surveillance. USDA APHIS MRPBS. $46,200
Saliki, Jeremiah T. BSE Surveillance Testing. USDA. $170,799
Saliki, Jeremiah T. Classical Swine Fever Surveillance 2010. USDA. $12,100
Saliki, Jeremiah T. Diagnostic services relative to the control, diagnosis, treatment, prevention and eradication of livestock 2010. GA Dept of Agriculture. $1,357,788
Saliki, Jeremiah T. Editorial Support for the Journal of Veterinary Diagnostic Investigation. AAVLD. $120,000
Sanchez, Susan. 2010 Georgia Veterinary Scholars Summer Research Program. NIH. $41,116
Schatzberg, Scott. Development of a pan-retroviral PCR assay and its application in canine degenerative myelopathy. Morris Animal Foundation Veterinary Scholars Program. $4,000
Schatzberg, Scott. Use of genome-wide association to identify disease susceptibility in Pug dogs with necrotizing meningoencephalitis. Morris Animal Foundation Veterinary Scholars Program. $4,000
Schmiedt, Chad. Effect of Topical Thrombin on coagulation time and amount in a rodent liver laceration model. Industry Sponsor. $25,862
Schmiedt, Chad. The effect of warm ischemia-reperfusion injury on renal function and vasoactive peptide expression in feline kidneys. American College of Veterinary Surgeons Foundation. $9,993
Tennent-Brown, Brett S. Assessment of the Systemic Inflammatory Response in Colitis in Horses. Morris Animal Foundation. $107,218
Tompkins, Mark. Broadly reactive human antibodies against influenza M2 for therapy and prophylaxis. NIAID (Sub-award under LANL). $72,086
Tompkins, Mark. Developing a paramyxovirus-based vaccine for influenza A virus H5N1. NIH (Sub-award under Pennsylvania State University). $217,761
Tompkins, Mark. Flu conference support. NIH. $10,000
Tripp, Ralph. Aerosolization of Influenza Vaccines. Industry Sponsor. $75,000
Tripp, Ralph. Antibody inhibition of respiratory syncytial virus G protein activity. NIH. $349,578
Tripp, Ralph. Immune Mechanisms of Virus Control (U01/U19): Manipulating natural host immunoregulation via IDO during viral infection. Medical College of Georgia. $405,057
Tripp, Ralph. NIAID Centers for Excellence for Influenza Research and Surveillance. NIH. $740,358
Tripp, Ralph. RSV Nanocapsule Vaccine Engineered with a G Protein Peptide Payload. NIH. $593,052
Tripp, Ralph. Therapeutic Antibodies Against Influenza. Industry Sponsor. $207,441
Wagner, John J. Cocaine-induced Metaplasticity in the Hippocampus. NIH. $185,625
Watford, Wendy. TPL2-Dependent IFN-G Production: contribution to host defense and autoimmunity. NIH. $162,000
Williamson, Lisa. Dose Titration of Oral and Injectable Moxidectin and oral Morantel Tartrate in Camelids. Morris Animal Foundation. $22,074
Yabsley, Michael. Determining Exposure Levels of Wood Ducks to Avian Influenza Virus and Newcastle Disease Virus in the Eastern United States. Wildlife Management Institute. $35,000
Yabsley, Michael. Ecology of vector-borne pathogens, including a novel Borrelia, in African penguins from South Africa. Morris Animal Foundation Veterinary Scholars Program. $4,000
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