Message from AAVI President Chris Davies

With Jim Harp’s retirement from the USDA, Agricultural Research Service and resignation from the AAVI Board, my term as president has been extended for six months and Doug Bannerman’s term will begin six months early on June 1, 2009. I greatly appreciate Doug’s willingness to share the extra responsibilities with me and I am confident he will provide exceptional leadership for AAVI. I also wish Jim Harp great success in his new career as a musician and the best of times in retirement.

I welcome Gary Splitter to the position of AAVI Vice President, Philip Griebel to the AAVI Board, and Dave Benfield and Isis Mullarky to the Nomination Committee. I thank all of the AAVI Officers and Committee Members for their contributions to the AAVI. I am particularly indebted to the committee chairs: Jim Roth, last year’s Nomination Committee chair; Pat Shewen, this year’s Nominations Committee chair; Carol Chitko-McKown who is continuing as chair of the Student Awards Committee; Ron Schultz who is continuing as chair of the Fund Raising Committee; Joan Lunney, the chair of the Constitution/By-Laws Committee; and Susan Eicher, our Newsletter Editor. A special note of thanks goes to Gina Pighetti for the great job that she has done in her first year as our Secretary/Treasurer. Clearly AAVI cannot succeed without the dedicated service of many of our members. Therefore, I encourage you to help your professional association by volunteering to serve on an AAVI committee. A complete list of AAVI Officers and Committee Members is included later in the newsletter.

Chris Davies presiding at the annual AAVI meeting in Chicago.

Notable events at this year’s Conference for Research Workers in Animal Diseases (CRWAD) included: an excellent and well-attended pre-conference AAVI/ACVM Symposium on Stress and Innate Immunity; active participation in the graduate student Immunology Section competition with six oral and 18 poster presentations; a new and improved venue for our business luncheon, Buca di Beppo a family style Italian restaurant directly across the street from the Chicago Marriott; a
A wonderful DVI presentation by this year’s AAVI-DVI recipient Lorraine Sordillo, a former AAVI President; and a great time for all at the annual Jeanne Burton Greek Night dinner. I congratulate the winners of the Student Competition: J. Jee from The Ohio State University first place oral presentation, K.S. Chattha from the University of Guelph second place oral presentation, A.A Elliott from the University of Tennessee third place oral presentation, H.L.X. Vu from the University of Nebraska first place poster presentation, M.C. Heller from the University of California at Davis second place poster presentation, and M. Bharathan from Virginia Polytechnic Institute and State University third place poster presentation. Please take a look at their outstanding extended abstracts, which are included later in this newsletter. I also again congratulate Dr. Sordillo on being selected as the 2008 AAVI-DVI.

At the December AAVI Board meeting, the Board decided to name the Greek Night dinner in honor of our prematurely deceased colleague Jeanne Burton of Michigan State University and to use $500 per year from the Jeanne Burton memorial fund to subsidize graduate student participation in Greek Night. If you are interested in contributing to the Jeanne Burton Memorial fund, you can make a contribution when you pay your dues or by sending a check to Gina Pighetti, the AAVI Secretary/Treasurer. Another important news item is that we have finally succeeded in implementing an easy to use online dues payment system that can be accessed from the AAVI website http://www.theaavi.org. I encourage everyone to pay his or her dues annually; if AAVI is to succeed we must maintain and grow our membership base. Our current membership is approximately 128 active, individual members and two corporate members: Merial and Pfizer Animal Health. If you have colleagues or students who should be members but are not please encourage them to join. In addition, if you have contacts at corporations that might be interested in becoming corporate members, please send the contact information to Ron Shultz, the chair of the AAVI Fund Raising Committee.

If you attend IMMUNOLOGY 2009 in Seattle, Washington, please come to the AAVI/American Association of Immunology – Veterinary Immunology Committee (AAI-VIC) Symposium, which is scheduled for Sunday May 10 from 12:30 to 2:30 PM. The topic for this year’s symposium is Comparative Biology of Non-Classical MHC Class I Molecules and we have four excellent speakers. For details see the meeting announcement later in this newsletter. I would also encourage you to consider attending the Fifth International Veterinary Vaccines and Diagnostic Conference (IVVDC), July 19-24 in Madison, Wisconsin. The IVVDC will be dedicated to the pioneers of veterinary vaccine development including Skip Carmichael, John Gorham and Max Appel. AAVI will be applying for a USDA trainee travel grant for IVVDC. Consequently, if you have a graduate student or postdoctoral fellow who is planning to attend IVVDC encourage him or her to apply for an AAVI travel stipend. I look forward to seeing you and other Veterinary Immunology colleagues at IMMUNOLOGY 2009, CRWAD and other scientific venues. If you have questions or comments about AAVI please do not hesitate to contact me by E-mail or phone. May veterinary immunology and all of science prosper and thrive in the new era of political change, positive thinking, and scientific discovery!

The annual meeting, which was held at Bucca di Beppo, was well attended with a wonderful lunch and great atmosphere.
Lorraine M. Sordillo initiated her early training at the University of Massachusetts, Amherst, receiving a BS in Animal Science and a MS in Lactation Physiology. In 1987, she earned her PhD in Immunology from Louisiana State University, Baton Rouge. Dr. Sordillo then began her post-doctoral work at the University of Tennessee and in 1988 joined the Immunology group at the Veterinary Infectious Disease Organization, University of Saskatchewan. In 1992, Dr. Sordillo joined the faculty ranks of the Veterinary Science Department at Pennsylvania State University, and became director of the Center for Mastitis Research. In a short period of time Lorraine rose to associate and then full professor prior to relocating to Michigan State University. In 2004 Dr. Sordillo was the first to be offered the prestigious Meadow Brook Chair position in Farm Animal Health and Well Being in the Department of Large Animal Clinical Sciences at Michigan State where she continues her research today.

Dr. Sordillo’s primary research has focused on developing solutions to control mastitis in dairy cattle by understanding basic mammary gland physiology and immunology. She has built an extensive research program that studies both the molecular mechanisms of disease and the whole animal response during infection. To help support her program, she has garnered more than $7.5 million in funds from industry and various state and national programs including the USDA-NRI and the National Institute of Health. To date, Dr. Sordillo has trained more than 20 graduate students. She has translated her drive and passion for solving problems to her students developing their ability to design and conduct high quality research and become future leaders in the field. Three former students are faculty members at 4 year-research institutions and 11 are in university or industry-based positions. She has served as a mentor for undergraduate honor’s students, visiting scholars, and post-doctoral associates. Her research has generated 5 U.S. patents aimed at mastitis therapy and more than 80 peer-reviewed journal articles in high impact journals. In recognition of her research leadership, Dr. Sordillo has received multiple awards to include the Young Scientist and West Agro Awards from the American Dairy Science Association. Her work is highly respected in various fields as is evidenced by the many invitations to present her work at national and international meetings, universities, and in book chapters.

Dr. Sordillo is a strong proponent of returning her expertise to the scientific community. She has dedicated time to serve on grant review panels, editorial boards and only recently completed her term as Editor for the Physiology and Management Section of the Journal of Dairy Science. She also provided leadership and guidance to the American Association of Veterinary Immunologists, not only by continuously participating in multiple committees but also by serving as the association’s president.

Dr. Lorraine Sordillo’s drive, passion, and excitement for science and solving problems have been a cornerstone for many. Through her research, training of future immunologists, and service Lorraine has significantly impacted the veterinary immunology community and continues to inspire fellow scientists across the world.

Dr. Sordillo is married to Jeff Gandy who works at her side conducting research in her laboratory. They have one daughter, Candice Gandy, currently enrolled in nursing school and three dogs, better known as the three amigos.

Secretary/Treasurer’s Note

By Gina Pighetti

Good news everyone – Online Payment is up and running! I greatly appreciate the efforts of Chris Davies and Syed Sayem to set up the website and information collection system. To pay dues for 2009, back dues for
2008, or make contributions to the Jeanne Burton fund, go to our online site (www.theaavi.org) and open the membership application link. Fill out the online application form and submit it. Then continue to Google checkout and fill in the needed info. Yes, you need to fill out both our online application form and Google checkout info. After you submit the application form, an email is sent to you and the secretary treasurer – me. You also will receive an email from Google Checkout after I charge your credit card. On your credit card statement, you should see AAVI. If you have any questions, please don’t hesitate to ask (pighetti@utk.edu).

Dues need to be in by March 1 for 2009 to be considered current. Also in March you will receive an email from me regarding nominees for the AAVI Distinguished Veterinary Immunologist. So, get your thinking caps on and begin considering those you feel worthy of the nomination. I will follow up with details for the process soon.

Watch for the minutes of the board meeting and the business meeting that will be available on the website soon. A copy may be obtained by contacting the secretary-treasurer.

AAVI-ACVM 2008 Symposium

By Doug Bannerman

The annual American Association of Veterinary Immunologists (AAVI) – American College of Veterinary Microbiologists (ACVM) Symposium was held on Sunday, December 7, 2008 at the Chicago Marriott. The symposium was held in conjunction with the annual meeting of the Conference of Research Workers in Animal Diseases (CRWAD). The theme of the AAVI-ACVM symposium was “Stress and Innate Immunity.”

The first presentation of the symposium, titled "Making Sense About Stress and Immunity," was delivered by Janeen L. Salak-Johnson of the University of Illinois at Urbana-Champaign. Dr. Salak-Johnson’s presentation provided an outstanding overview of the area of stress and innate immunity, as well as some insights into her research on the role of dysfunction in the neuroendocrine-immune interface in mediating stress-related disease in swine. Jesse Goff of the Iowa State University gave the second presentation of the symposium, which was titled “Transition Cows & Impact of Metabolic Disease on the Immune System.” Dr. Goff’s talk focused on the fascinating link between the metabolic demands associated with milk production at parturition and temporally-associated diseases, such as mastitis, milk fever, and metritis. The third talk of the symposium, titled “Does Stress Play a Significant Role in Fatal Bovine Respiratory Disease?”, was delivered by Philip Griebel of the University of Saskatchewan’s Vaccine and Infectious Disease Organization. This presentation provided an overview of Dr. Griebel’s seminal research on the impact that stress can have on the pathogenesis and clinical outcome of bovine respiratory disease. The final presentation was given by Kip L. Lukasiewicz of Sandhills Cattle Consultants, Inc. In his talk, titled “The Reduction of Stress via Caregivers’ Approach,” Dr. Lukasiewicz presented behavioral and environmental approaches for reducing the stress experienced by cattle. Based on the strong evidence provided by the previous speakers for a cause and effect relationship between stress and immune dysfunction, Dr. Lukasiewicz provided practical advice for potentially reducing disease associated with heightened stress.

The joint AAVI/ACVM symposium presenters addressed aspects of stress and innate immunity.
I would like to thank my co-chairs, Scott McVey and Chris Chase, for their collaborative efforts in developing a theme and identifying outstanding speakers for the symposium. The upcoming 2009 AAVI-ACVM Symposium will be co-chaired once again by Chris Chase, as well as the current AAVI Vice-President, Gary Splitter.

Message from the Veterinary Immunology Committee (VIC) of the International Union of Immunological Societies (IUIS) and plans for the 9th International Veterinary Immunology Symposium

By Dr. Wayne Hein

Many AAVI members may not be aware that there is an international Veterinary Immunology Committee (VIC). This committee is organized under the International Union of Immunological Societies (IUIS). The IUIS is an umbrella organization for regional and national societies of immunology throughout the world. The IUIS objectives are: 1) to organize international cooperation in immunology and to promote communication between the various branches of immunology and allied subjects; 2) to encourage within each scientifically independent territory co-operation between the Societies that represent the interests of immunology; and 3) to contribute to the advancement of immunology in all its aspects. More information is available at http://www.iuisonline.org.

The IUIS VIC is chaired by Dr Wayne Hein (wayne.hein@agresearch.co.nz). In 2009 Dr. Hein is seeking AAVI member input on how VIC can best support Veterinary Immunology research. Please send him your ideas and suggestions. A major activity of the IUIS VIC is sponsorship of the International Veterinary Immunology Symposium (IVIS). The 9th IVIS will take place in Tokyo, Japan from August 16-20, 2010. Please note these dates in your meeting diaries.

The 9th IVIS Local Organizing Committee is led by the Chair: Onodera, Takeshi, University of Tokyo aonoder@mail.ecc.u-tokyo.ac.jp; Vice-chair: Onuma, Misao, Hokkaido University monuma@vetmed.hokudai.ac.jp; and Vice-chair: Kai, Chieko, University of Tokyo cki@ims.u-tokyo.ac.jp. They have organized a 9th IVIS Local Organising Committee and Scientific Committee who are working on the details of the symposium program, which will follow a pattern similar to previous symposia. At past symposia, the Veterinary Immunology Committee has sponsored Workshops on topics considered relevant at the time, e.g., Veterinary Immune Toolkit Progress, Comparative MHC, NK Cells, Comparative Immunoglobulin and Lymphocyte Markers.

The IUIS-VIC invites feedback from the international veterinary immunology community on topics that should be adopted for Workshops at the 9th IVIS. Suggestions should be sent to the VIC Chair, Dr Wayne Hein (wayne.hein@agresearch.co.nz) or to a VIC Member Dr Joan Lunney (Joan.Lunney@ars.usda.gov), Dr Cynthia Baldwin (Cbaldwin@vasci.umass.edu), Dr Jan Naessens (J.Naessens@cgiar.org), Dr Falko Steinbach (fstvirol@zedat.fu-berlin.de) or Dr Misao Onuma (monuma@vetmed.hokudai.ac.jp).

AAVI Graduate Student Competition at CRWAD 2008

By Carol Chitko-McKown

Results of the 2008 AAVI Student Presentation Competition

The annual AAVI Student Presentation Competition was held during the 89th annual CRWAD meeting, December 7-8, 2008, in Chicago, Illinois. The response to our call for participants was quite impressive – 6 students participated in the Oral Presentation category and 18 students presented Posters! The students represented 13 universities from Canada, the United States, and Mexico. The range of topics presented and animal models continue to grow each year. This year, food animal species were well-represented, as well as horses and wildlife. Three judges presided over the Oral presentations, and due to the large number of Poster presentations, six additional judges were assigned three to a poster. The first place winners in each category received $500, a plaque, and membership for a year in AAVI. The second place winners received $200, a plaque and membership in AAVI, and this year we added third place awards – membership in AAVI.

First place in the Oral category was awarded to Junbae Jee from the Food Animal Health Research Program and the Department of Veterinary Preventative Medicine, The Ohio State University, Wooster, Ohio.

Second place was awarded to Kuldeep S. Chattha from the Department of Pathobiology, Ontario Veterinary College, Guelph, Ontario, and third place went to Alexandra A. Elliot from the Department of Animal Science, University of Tennessee - Knoxville. The winners in the Poster category were Hiep L. Vu in first place from the Department of Veterinary and Biomedical Sciences, University of Nebraska – Lincoln. Second place was awarded to M. C.
Heller from the Department of Veterinary Medicine and Epidemiology, College of Veterinary Medicine, University of California – Davis. The third place poster presentation was made by Mini Bharathan from the Department of Dairy Science, Virginia Polytechnic Institute and State University, Blacksburg, Virginia.

AAVI extends its thanks to Glenn Zhang, Laura Miller, Tawni Crippen, Matt Sylte, Janice Zanella, Pat Shewin, Isis Mullarky and Manuela Renaldi for doing a fine job judging such great presentations. Thanks also to all of the student participants, the faculty mentors, and the CRWAD organizing committee – we couldn’t do this each year without all of you!

Extended Abstracts of the Oral Student Presentation Winners

Effect of Vitamin A on Bovine Coronavirus Infection, Vaccination and Immunity in Feedlot Calves.

Jee, J. 1,2*, M. Azevedo 1,2, A. Vlasova 1,2, A. Hoet 2, S. C. Loerch 3, C. Pickworth 3, J. Hanson 1 and L. J. Saif 1,2.

1Food Animal Health Research Program; 2Department of Animal Sciences, Ohio Agricultural Research and Development Center, The Ohio State University, Wooster, Ohio 44691; 3Department of Veterinary Preventive Medicine, The Ohio State University, Columbus, Ohio 43210

Vitamin A is recognized as an essential micronutrient for immune function. Vitamin A effects on immunity to infectious agents has been demonstrated in community- and hospital-based studies, resulting in reduced morbidity and/or mortality in children supplemented with adequate dietary vitamin A. The importance of vitamin A in immune responses to and resistance against infectious agents has been studied mainly for human disease or in mouse models. The zoonotic potential of several animal infectious diseases and their association with host vitamin A status is poorly understood. Feedlot calves are subject to multiple infectious diseases and especially the bovine respiratory disease complex, BRDC (shipping fever). This is attributed to the stress associated with shipping animals to feedlots and the co-mingling of animals from multiple farms at auction barns and within the feedlot. Bovine coronavirus (BCoV) was recently recognized as one of the causative agents of the BRDC. The BCoV was isolated from nasal samples and lung tissues of cattle with shipping fever pneumonia. In addition, respiratory tract infections with the BCoV, followed by secondary infections with Pasteurella spp. were observed in natural outbreaks of shipping fever. In feedlot cattle, dietary vitamin A restriction has been used to increase intramuscular fat, or marbling, resulting in higher quality beef production in terms of meat palatability. However, it is unclear whether vitamin A restriction which increases beef carcass value, exerts detrimental effects on the immune responses to pathogens or vaccine in cattle.

To understand the impact of vitamin A status in cattle on their immune responses, we investigated antibody (Ab) responses in feedlot calves naturally infected and intramuscularly vaccinated with BCoV. These calves were fed either high or low dietary vitamin A, defined as greater than or less than 2200 international units (IU)/kg of dietary dry matter (DM), respectively as recommended by the National Research Council. Angus steers (n=40, average 199 days old) were randomly assigned to two groups. One group received low dietary vitamin A (LVA group; 1100 IU/kg of dietary DM, n=20) to simulate low levels in the vitamin A restricted feedlot calves and the other group received high dietary vitamin A (HVA group; 3300 IU/kg of dietary DM, n=20). Supplemental vitamin A was fed at post-arrival day (PAD) 0, then daily throughout the 140 day study period (PAD 140). Because approximately 90-112 days are required to decrease vitamin A in serum from the liver stores, all calves were vaccinated intramuscularly with an inactivated BCoV vaccine (Scourguard® 3KC, Pfizer) at PAD 112 and boosted at PAD 126 to determine the effect of vitamin A status on vaccine-induced BCoV Ab responses at PAD 140. Fecal, nasal and blood samples were obtained at PAD 0, 4, 35, 112 and 140. Quantitative Real-time RT-PCR was used to detect BCoV shedding in the fecal and nasal specimens. Serum and fecal samples were assayed by ELISA for IgG1, IgG2, IgM, IgA and fecal IgA isotype Abs to BCoV. In addition, the ratios of IgG1 to IgG2 Abs were used to assess dominance of humoral immune responses (IgG1, Th2) over cell-mediated immune responses (IgG2, Th1) in cattle.
At PAD 0, the calves in both LVA and HVA groups showed statistically similar background levels of BCoV shedding and serum BCoV Ab. Therefore, no sub-categorical analysis was required. Twenty calves (50% of total calves) shed BCoV either in feces or nasally at least once shortly after arrival (at PAD 0 and 4). The prevalence of BCoV shedding increased from 21.05% at PAD 0 to 41.03% at PAD 4, but with no BCoV shedding detected thereafter (at PAD 35, 112 and 140). Before the decreased vitamin A levels in serum occurred (at PAD 0, 4 and 35), we investigated the isotype Ab responses induced at PAD 35 by natural BCoV infection in PAD 0 and 4. The BCoV fecal shedding was negatively associated with pre-existing serum IgA BCoV Ab titers at PAD 4 (based on the derived formula, probability of BCoV fecal shedding \( = 100\times[\frac{e^{(1.84–0.63\times\log_{10}\text{IgA})}}{1+e^{(1.84–0.63\times\log_{10}\text{IgA})}}] \)). In addition, BCoV nasal shedding was negatively associated with pre-existing serum IgA and IgM BCoV Ab titers at PAD 4 (based on the derived formula, probability of BCoV nasal shedding \( = 100\times[\frac{e^{(2.37–1.21\times\log_{10}\text{IgA})}}{1+e^{(2.37–1.21\times\log_{10}\text{IgA})}}] \) and probability of IgG1-BCoV Ab seroconversion \( = 100\times[\frac{e^{(0.25–0.61\times\log_{10}\text{IgM})}}{1+e^{(0.25–0.61\times\log_{10}\text{IgM})}}] \)), respectively. Serum IgA- and IgG1-BCoV Ab seroconversion at PAD 35 was positively associated with average BCoV RNA copy numbers detected in fecal samples at PAD 0 and 4 (based on the derived formulas, probability of IgA-BCoV Ab seroconversion \( = 100\times[\frac{e^{(5.52+1.25\times\text{average of BCoV copies})}}{1+e^{(5.52+1.25\times\text{average of BCoV copies})}}] \) and probability of IgG1-BCoV Ab seroconversion \( = 100\times[\frac{e^{(3.25+1.28\times\text{average of BCoV copies})}}{1+e^{(3.25+1.28\times\text{average of BCoV copies})}}] \)).

After vitamin A decreased in serum (by PAD 112), we investigated BCoV Abs induced at PAD 112 and 140 by an inactivated BCoV vaccine given at PAD 112 and 126. Serum IgG1 BCoV Ab titers and the ratios of IgG1 to IgG2 Abs in the HVA group were significantly higher at PAD 140 than at PAD 112 \( (p < 0.005) \), whereas those in the LVA group did not differ significantly. At PAD 140, serum IgG1 BCoV Ab titers and the ratios of IgG1 to IgG2 Abs were significantly higher in the HVA than in the LVA group \( (p < 0.05) \). Calves naturally infected with BCoV in the LVA group \( (n=11) \) were compared to assess the impact of BCoV infection and BCoV vaccination on Ab responses before the decreased vitamin A (at PAD 0 and 35) and after the decreased vitamin A (at PAD 112 and 140) in serum. The calves naturally infected with BCoV had predominantly serum IgG1 BCoV Ab at PAD 35 \( (p < 0.05) \). However, the calves previously recovered from natural BCoV infection had a compromised serum IgG1 BCoV Ab response to an inactivated BCoV vaccine at PAD 140.

To our knowledge, this is the first study to analyze the impacts of micronutrients, i.e., vitamin A on Ab responses to BCoV in feedlot calves. The vitamin A status in feedlot calves had a significant effect on Ab responses to an inactivated BCoV vaccine. Serum IgG1 BCoV Abs were induced predominantly under the high vitamin A dietary regimen, whereas they were compromised under the low vitamin A dietary regimen, suggesting that the low vitamin A diet suppresses the Th2 associated Ab (IgG1) development. Additional studies are needed to assess the effects of low vitamin A on systemic and mucosal immune responses to other vaccines and on vaccine-induced protection in feedlot calves.

**Expression of CD21, CD32 and membrane IgM on calf lymphocytes varies with age.**

K.S. Chattha, M.A. Firth, D.C. Hodgins, P.E. Shewen, Department of Pathobiology, Ontario Veterinary College, University of Guelph, Guelph, ON, Canada

Induction of active antibody responses to vaccination is difficult in neonates. This has been attributed partly to interference by maternal antibodies (IgG) and partly to the limited functional ability of the neonatal immune system. Antibody production by B lymphocytes is regulated by various activating and inhibitory receptors such as CD21, CD32 and membrane IgM, the B cell antigen receptor. CD21 binds complement component C3d and lowers the threshold of activation for B lymphocytes; whereas CD32 binds the Fc region of IgG and when cross linked with mIgM, elevates the threshold of activation. Thus, an interaction among CD21, CD32 and mIgM determines the response of B cells, and alteration in expression of these receptors with age might influence the ability of neonatal B lymphocytes to respond to vaccination. This study evaluated the expression of mIgM, CD21 and CD32 in 15 Holstein calves from birth until 25 weeks of age. Adult cows were used for comparison. Peripheral blood mononuclear cells were triple stained with monoclonal antibodies specific for mLgM, CD21 and CD32 and were analysed using flow cytometry. There was a significant rise in both the absolute number and % of cells expressing mLgM and CD21 with age. The absolute number of cells expressing CD32 also increased significantly with age. Approximately >90% IgM positive lymphocytes expressed both CD21 and CD32 showing potential for response to activating and inhibitory signals. Mononuclear cells expressing CD14 also stained with CD32 antibody, and were higher in neonatal calves compared to adults. Calves were...
comparable to adults in expression of CD21 but differed from adults in decreased expression of CD32 and increased expression of mIgM receptors per lymphocyte. These age related variations may account for the observed poor immune response of neonatal calves.

**Altered actin expression by neutrophils from cows genetically more susceptible to mastitis.**

The largest loss in profit for dairy farmers occurs with mastitis, an inflammation of the mammary gland. Observed differences in genetic susceptibility to mastitis between cows have led to ongoing research to identify why these differences occur. Our prior research identified a marker in the CXCR1 gene, a receptor present on neutrophil surfaces for the chemoattractant interleukin-8 (IL-8). This polymorphism has been associated with mastitis and decreased neutrophil migration in vitro. Because neutrophil migration is critical for eliminating most infections, our ongoing research seeks to identify the specific mechanisms causing impaired migration. Actin polymerization, one of the first steps in neutrophil migration, increases F-actin formation at the front of the cell. This changes the cells shape and allows it to move. This study evaluated actin polymerization and cell morphology in cows with different CXCR1+777 genotypes. Neutrophils from cows with GG (n=11) and CC (n=11) genotypes were isolated and stimulated with zymosan activated sera (ZAS). Cells were fixed and stained for F-actin at 0, 15, 30, 60, 90, 120, and 180 seconds and subsequently evaluated for F-actin content, distribution, and cell morphology. F-actin content was quantitated by reading the fluorescent intensity of the F-actin stain on a flow cytometer. Neutrophils of the CC cows had significantly lower F-actin polymerization than the GG cows (P=0.05) at 15, 30, and 60 seconds after stimulation. Because F-actin polymerization drives neutrophil movement, lower amounts could partly explain reduced migration. For the second part of the study, neutrophils 0, 30, and 90 seconds after stimulation were given a score from 1 to 4 based on F-actin distribution and cell morphology. Cells receiving a score of 1 being perfectly round, with even F-actin distribution throughout the cell, and those receiving a score of 4 with all the F-actin located at the leading edge and outer edges very ruffled. Neutrophil morphology and F-actin distribution scores were found to be slightly higher at time 0 in CC cows, suggesting that neutrophils could be pre activated in this genotype. Our findings suggest altered F-actin polymerization could impair neutrophil migration in cows with the CC genetic background and may contribute to increased mastitis susceptibility. Because F-actin is a driving force behind directional migration, current studies aim to compare the actual paths and timing neutrophils from different genotypes are taking towards chemoattractants. Finding the reasons behind what makes some cows more genetically vulnerable to infection will provide an understanding which will help develop targeted strategies to prevent and treat mastitis infections.

**Extended Abstracts of the Poster Presentation Winners**

**Sub-typing PRRSV isolates by means of measurement of cross neutralization reactions.**
H.L.X. Vu 1*, M. Brito 1, W.I. Kim 2, K.J. Yoon 2, W. Laegreid 3, and F.A. Osorio 1. 1 Dept. of Veterinary and Biomedical Sciences, University of Nebraska-Lincoln, Lincoln, NE. 2Dept. of Veterinary Diagnostic and Production Animal Medicine, Iowa State University, Ames, IA. 3 Dept. of Veterinary Pathobiology, University of Illinois at Urbana-Champaign, Urbana, IL

Porcine reproductive and respiratory syndrome (PRRS) causes substantial economic losses to swine producers worldwide. Vaccines have been developed in an effort to control the disease. However, genetic and antigenic diversity among PRRS virus (PRRSV) strains is one of the major obstacles for the development of a successful vaccine that could provide a wide spectrum of protection. Current vaccines can provide effective protection against PRRSV strains that are closely similar to the vaccine strain. On the other hand, the protection conferred by PRRS vaccines against heterologous strains...
is highly variable and substandard. Sub-typing PRRSV isolates becomes of cardinal importance for making decisions on which viral strains to use when formulating a vaccine. It has been demonstrated that neutralizing antibodies are important contributors to PRRSV protective immunity. Cross-neutralizing activity amongst different PRRSV strains can therefore be a parameter that allows grouping the constellation of PRRSV strains. Grouping PRRSV strains based on cross-neutralization titers would better reflect their antigenicity and immunogenicity, rather than using the mere genetic comparison of selected genes of PRRSV. Moreover, the value of cross-neutralization for establishing subtypes of highly variable RNA viruses has been well proven in case of Foot and Mouth Disease virus.

In the present study, cross-neutralization reactions were used to sub-type field PRRSV isolates. Antisera were developed against six PRRSV reference strains. Antisera against Lelystad, 97-7895, VR2332 and MN184 were originally selected as reference because these strains represent a wide range of genetic distance among PRRSV strains when their ORF5 genes are compared. On the other hand, antisera 5424-00 and 1403-02 were developed against two field-isolates which were not significantly cross-neutralized by any of the four initial reference antisera, thus considered as “non-typable” isolates. The cross-neutralization profiles of 68 PRRSV isolates originated in the Mid-West of the US were measured using these six reference antisera. PRRSV isolates were then clustered according to cross-neutralization results in a manner that all PRRSV isolates in the same cluster share similar pattern of cross-neutralization. Using hierarchical clustering analysis, we are able to define 8 clusters of PRRSV isolates representing the complete range of cross-neutralization profiles. We hypothesize that incorporation of additional reference antisera prepared against selected “non-typable” isolates will confirm these clusters, while also being possible that these additional reference antisera could strongly exhibit cross reactivity with isolates in cluster 1 & 2 (which so far exhibit little or no reactivity with any of the reference antisera). The overall goal is to define number of valencies or specificities that should be represented in a PRRS vaccine to achieve broad protection. Along this line, future studies include: (1) Obtaining full genome sequence information of at least one representative isolate for each cluster; (2) comparing the range of genomic and cross-neutralizing phenotype of the PRRSV; and (3) conducting bilateral cross – protection studies in vivo using the isolates that represent each of the clusters.

A potential role for indoleamine 2, 3 dioxygenase (INDO) in Rhodococcus equi infection.

Rhodococcus equi is a facultative intracellular bacterial pathogen of horses; infected foals develop pyogranulomatous pneumonia, though adult horses are largely unaffected. Immunocompromised people develop a similar pneumonia if they become infected. R. equi is able to evade the defenses of the innate immune system and proliferate within host macrophages and dendritic cells (DCs). Previous studies in our lab identified the gene INDO as differentially expressed in R. equi infected equine monocyte-derived DCs and alveolar macrophages. INDO is the initial enzyme in tryptophan catabolism pathway. INDO also has immunoregulatory properties; expression in tumor and transplant tissue induces immune tolerance and INDO expression by DCs induces the generation of regulatory T cells. Two routes by which this enzyme may affect infection by intracellular pathogens are immune-regulation and tryptophan depletion. Depleting the host cell of tryptophan suppresses growth of tryptophan-dependent organisms such as Chlamydia. Our lab has shown that R. equi is not tryptophan dependent. The purpose of this study was to investigate an immune-regulatory role for INDO in R. equi infection in vivo using INDO<sup>−/−</sup> and C57BL/6J mice.

Indo<sup>−/−</sup> (B6.129<sup>Indom1Alm/J</sup>) (n=22) and strain matched control (C57BL/6J) (n=20) mice were infected with 5x10<sup>7</sup> R. equi ATCC 33701<sup>+</sup> by intraperitoneal injection. Mice were necropsied at 3 and 6 days post infection (PI) and organ weights of liver and spleen were recorded. Samples of liver and spleen were homogenized and plated in serial dilutions on brain-heart infusion agar to obtain bacterial counts. Histologic sections of all major organs were examined by a pathologist, without knowledge of grouping, and assigned an inflammation score. Differences in spleen and liver inflammation scores were analyzed using the non-parametric Mann-Whitney test. Liver and spleen tissue obtained at necropsy was stored in RNAlater at −80°C prior to RNA extraction and cDNA synthesis. Differences in efficiency of total RNA isolation and first strand synthesis were normalized to GAPDH for TaqMan<sup>®</sup> RT-PCR (Interferon-gamma (IFNγ), tumor necrosis factor-alpha (TNFα), IL-4, IL-6, IL-10, IL-12, IL-23) and 18SrRNA for SYBR Green RT-PCR (forkhead box
P3 (FoxP3), transforming growth factor-beta (TGFβ)). Cytokine and transcription factor gene expression data was analyzed using the students T test.

Liver weights at 6 days PI were heavier in INDO−/− mice (0.2195g ± 0.0910) than wild type (0.148g ± 0.0568) (P=0.02). No differences were seen in spleen weights or bacterial counts in spleen or liver between INDO−/− mice and controls.

Histologic sections of major organs including liver, spleen, mesenteric and thoracic lymph nodes, pancreas, and abdominal fat were examined and an inflammation score was assigned to each tissue based on number of inflammatory foci per 20X field; presence of coagulative necrosis was also taken into account. Liver tissue of INDO−/− had equivalent inflammation scores as liver from controls at 3 days post-infection, but higher scores at 6 days post infection (P=0.05), due to a decrease in inflammation in the controls. INDO expressing DCs promote the generation of regulatory T cells (Tregs) which play an anti-inflammatory role during an immune response. Therefore, a lack of INDO expressing DCs would be expected to diminish the induction of Tregs, and lead to an impaired anti-inflammatory response. Induction of Tregs is associated with TGFβ, and Tregs are characterized by expression of the transcriptional regulator forkhead box p3 (FOXP3). As expected, liver tissue of INDO−/− mice had decreased expression of TGFβ at 3 days PI (P=0.01), and decreased levels of FOXP3 expression at 3 days (P=0.02) and 6 days (P=0.03) compared to control tissue.

INDO−/− mice had higher inflammation scores in splenic tissue at 3 days post infection (P=0.02), however by 6 days post infection the inflammation scores were equivalent to those of the controls. Expression of TGFβ and FOXP3 was increased in splenic tissue of INDO−/− as compared to control mice at 6 days post-infection (P=0.01, P=0.04 respectively). This indicates the presence of a greater number of Tregs in INDO−/− splenic tissue.

In an effort to characterize the type of inflammatory response seen in liver and spleen tissues expression of cytokine mRNA was assessed using real-time PCR. No difference was seen in levels of cytokine transcripts between INDO−/− and C57BL/6 for the following genes: IL-4, IL-6, IL-10, IL-12, TNFα, and INFγ. Expression of IL-23 was significantly lower in INDO−/− spleen tissue at 6 days PI. (P=0.03).

In summary, INDO−/− mice infected with R. equi had livers which were heavier and had a higher inflammatory score than livers of control mice. As expected, INDO−/− mice also had a lower level of TGFβ and FOXP3 expression in their liver tissue compared to control mice, indicating a lower level of T regulatory cells. An impaired T regulatory response would be expected in an INDO−/− animal and would explain a more robust pro-inflammatory response seen in the liver tissue. The results for splenic tissue did not parallel what was found in the liver tissue. While splenic tissue of INDO−/− mice had higher inflammatory scores at 3 days post-infection than controls, by 6 days post infection splenic inflammation was equivalent between the two groups. FOXP3 and TGFβ expression was equivalent between the two groups at 3 days post infection and was increased in INDO−/− mice at 6 days post infection. This indicates that, contrary to what is expected, the spleen tissue of INDO−/− mice do not have an impaired ability to generate T regulatory cells. Redundant pathways for induction of regulatory T cells within the spleen may account for the increase in FOXP3 expression seen in the INDO−/− mice. The presence of regulatory T cells within the spleen would account for the decrease in inflammation by day 6 to an equivalent level as control mice. Further work is currently being done to identify T regulatory cells in the spleen and liver tissue using immunohistochemistry.

Characterization of T lymphocyte response to Staphylococcus aureus sensitized monocyte derived dendritic cells from cows with prior Staphylococcus aureus mastitis.

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Staphylococcus aureus is a versatile pathogen causing a variety of diseases in both humans and animals. Reports indicate that infected individuals are not protected from subsequent infection because of defective immunological memory. S. aureus is known to produce a variety of toxins and immunomodulatory proteins providing this pathogen with the ability to evade host innate and adaptive immune mechanisms. Together, these facts may explain the lack of specific or persistent protection of available bacterins, capsular polysaccharides, and superantigens based vaccines. As an alternative approach to current vaccine development strategies, dendritic cells (DC) may be used as biological adjuvants in S. aureus vaccines to enhance T cell memory. The objective of this study was to evaluate and characterize T cell memory development in response to S. aureus loaded bovine monocyte derived DC. Peripheral blood monocytes (CD14+) were isolated from cows previously diagnosed with S. aureus mastitis (infected; n= 5). Control cows (n= 5) were chosen based
on lack of previous record of *S. aureus* mastitis. For DC differentiation, CD14+ cells were cultured for 7 days in RPMI based medium supplemented with recombinant bovine granulocyte-monocyte colony stimulating factor and recombinant bovine interleukin-4. Differentiated DC had a distinct phenotype from CD14+ cells and an increased expression of CD205, MHC II, and CD11c markers as well as decreased expression of CD14 as assessed by flow cytometry. To evaluate activation markers as well as decreased expression of CD14 as increased expression of CD205, MHC II, and CD11c markers as well as decreased expression of CD14 as assessed by flow cytometry. To evaluate activation profiles, DC were stimulated with *S. aureus* for 4hrs or with LPS and FACS analysis was done after 48hrs of stimulation. DC stimulated with *S. aureus* had a greater intensity of expression of CD11b, CD11c, MHC class I & II, CD205 and lower expression of CD14 compared to unstimulated and LPS stimulated DC. In order to assess the ability of DC to induce lymphocyte proliferation, DC were loaded with *S. aureus* isolates from infected cows. Day old cultures of autologous lymphocytes were added to respective wells 24 hours after loading of DC. All experiments were done in quadruplicate with appropriate controls. Lymphocyte proliferation was measured using Cell Titer solution (Promega) according to manufacturer’s instruction after 4 days. Data was analyzed by analysis of variance using SAS software. There was a significant increase in lymphocyte proliferative response in infected cows compared to control animals, indicative of a memory response to *S. aureus*. Proliferating lymphocytes were further characterized by labeling with CFSE and analyzing T cell markers including CD4, CD8 and WC1γδ. DC loaded with *S. aureus* induced increased proliferation of CD4, CD8, and γδ T cells suggesting a cell mediated memory response to *S. aureus* in previously infected cows. Future work will characterize proliferating T cells using memory markers; identify the type of T cell polarization, and to identify a potential vaccine antigen for *S. aureus*. In conclusion, understanding the DC - T cell interactions and the T cell subsets involved in response to *S. aureus* might aid in the design of a successful vaccine.

**Membership in AAVI**

*By Ron Schultz, Chair*

As Chair of the American Association of Veterinary Immunologists (AAVI) membership committee, I urge you to join (or rejoin) our Society. We are an international group devoted to the pursuit of knowledge in Veterinary Immunology. This is an excellent means to communicate and network with other scientists worldwide. We co-sponsor a joint symposium with the American Association of Immunologists – Veterinary Immunology Committee (AAI-VIC) at the AAI annual meeting. Members of AAVI can also become members of the International Veterinary Immunology Committee (IUIS-VIC) and of AAI-VIC. AAVI annual dues are $50 for full members and $20 for students. Electronic access to the Elsevier journal Veterinary Immunology and Immunopathology is available to either full or student members of AAVI for an additional $40.

**Veterinary Immunology and Immunopathology**

*the official journal of AAVI*

*By Cynthia Baldwin*

NEW FORMAT – TECHNICAL REPORT. There is a new format for short papers available in VII known as the Technical Report that may be of particular interest. A Technical Report is a description of: (A) a new gene or its expressed sequence (mRNA) that is comprehensive at least with regard to the coding sequence or the complete mature expressed protein, with annotation (leader/signal sequence, start, stops, and other features), comparison to other species (e.g. by cladogram, percent similarity of gene and deduced amino acid), evidence of deposition into a publicly available gene bank (e.g. GenBank with accession number); or (B) a new monoclonal antibody that convincingly shows specificity for a new target molecule or allows significant improvement of existing procedures or diagnostics (ELISA, flow cytometry, Western blotting, tissue sections); or (C) availability of a functional recombinant cytokine or chemokine with clear evidence that it has biological activity commensurate with the native molecule. A Technical Report should not occupy more than 4 printed pages including text with no headings and of not more than 1500 words, no more than 3 figures and tables, an Abstract of not more than 200 words, and References.

HAVE AN IDEA FOR A REVIEW? VII is always interested in your ideas for mini-reviews or full-length reviews. Remember, all that work your graduate student does to review the literature for his/her dissertation introduction may be material that could see the light of day as a review article. *Mini-reviews and opinions* should cover subjects in specialized areas of veterinary immunology which are either topical, or of emerging...
importance, or in need of an update. They should not exceed 3000 words and be easy to read. Articles containing provocative opinions and hypotheses not yet firmly supported by experimental evidence should be suitable, provided that any biases expressed are acknowledged by the authors. Review Articles in a traditional, more comprehensive style are also published in the journal. Authors wishing to submit these, or Mini-reviews/opinions are asked to first submit a title plus abstract for advice on suitability to the Review Articles Editor Dirk Werling Dwerling@RVC.AC.UK.

HAVE AN IDEA FOR A SPECIAL ISSUES? We are always looking for ideas for Special Issues with you as the Guest Editor. If you have an idea pose it to one of the Editors. Ones expected this year include “Equine laminitis”, guest editors Belknap and Moore; the 8th IVIS in Brazil, guest editors Santos, Lunney and Ferreira; IgE in domestic animals, guest editor Laurel Gershwin; 9th FIV Symposium (Feline Retrovirus), guest editor Slattery and Vahlenkamp.

AD HOC REVIEWER PANEL. If you would like to serve as an ad hoc reviewer please contact one of the editors by email to be added. We are especially interested in updating our list of junior scientists willing to review articles. This assists VII in its bid to stay current and helps junior scientists to meet the requirement of a service component. Members of the Editorial Board are gleaned from the ad hoc panel of reviewers.

U.S. Veterinary Immune Reagent Network (US VIRN) Progress and Plans

PI: Cynthia Baldwin
University of Massachusetts Amherst

Year 3 of the US VIRN has been quite productive. Kingfisher Biotech Inc. has used a yeast expression system for production of cytokines and chemokines from multiple species as noted in more detail below. Progress with reagent development is reported, and updated monthly, at the US VIRN website (www.vetimm.org). The list of available reagents is noted at the Kingfisher Biotech website: http://kingfisherbiotech.com.

Additional work at the Wagner lab at Cornell University has been aimed at hybridoma fusions for producing monoclonal antibodies (mAbs) to cell surface molecules using their Ig and IL expression vectors as well as some cytokines and chemokines in addition to expressing the cell surface proteins for all species in a mammalian expression system. Similarly, efforts at the Baldwin lab at University of Massachusetts have been targeted at cloning and sequencing genes for all species involved in the Network and producing mAb to the expressed cytokines and chemokines as well as cell surface molecules to all species including catfish and trout. Results in mammals and poultry are noted below.

Cattle progress
MAb to T cell receptor gamma has been developed using protein expressed as a single C domain fused to equine IL-4. Many yeast expressed cytokines and chemokines have been evaluated in biological assays including IFNγ, CXCL9, CXCL10, and IFNα.

Horse progress
Reagent development for the horse focuses on cytokines and anti-cytokine reagents and on antibodies to cell surface marker. Recombinant equine IL-2, IL-4, IL-6, IL-15, GM-CSF and CCL2 were developed in yeast. MAb to equine CD23, FceRI, CD28, IL -4, IL -2 and CCL2 have been generated and are currently characterized. Additional cytokine reagents and antibodies to T-cell receptors and various cell surface marker will be developed within the next year.

Poultry progress
Recombinant chicken cytokines IL-10, IL-16 and IL-18 have been expressed and are commercially available through Kingfisher Biotech Inc., St. Paul, MN (http://kingfisherbiotech.com). MAb to chicken IL-10 and IL-16 are in progress following successful fusions.

Swine Progress
Year 3 of the US VIRN has resulted in numerous pig reagents. The Lunney lab has affirmed bioactivity for CXCL10 and CXCL11; tests for IL-13 and CCL2 and CCL5 are underway. Expressed chemokines are available through Kingfisher Biotech Inc. Hybridoma fusions have been screened for swine T cell receptors (TCRa and TCRb) but the original expression resulted in no positives. Bettina Wagner’s lab at Cornell is re-expressing the TCRs and we hope to screen additional fusions in year 4. Several hybridoma fusions for mAb against swine chemokines and cytokines and their receptors are planned or already being screened. Results indicate that mAb for IL-4R and CCL2 have been produced; final confirmation is in progress.

Updates of the recent work by the U.S. Veterinary Immune Reagent Network are available on the website: http://www.umass.edu/vetimm/. Please visit to see the
progress. The gene clone inventory was attached with the Spring 2008 newsletter.

Upcoming Meeting Announcements


The AAVI and the AAI - Veterinary Immunology Committee (AAI-VIC) will host a joint symposium, *Comparative Biology of Non-Classical MHC Class I Molecules* at IMMUNOLOGY 2009 on Sunday, May 10 from 12:30 to 2:30 PM. Chairs: Christopher J. Davies, Utah State University (President AAVI) and Mark A. Jutila, Montana State University (Chair, AAI-VIC).

**Speakers:**
- **Joan S. Hunt**, University of Kansas Medical Center. *Similar and dissimilar features of human and baboon MHC class Ib antigens in placentas.*
- **Thaddeus G. Golos**, University of Wisconsin. *Towards in vivo models to define the functions of primate MHC class I molecules.*
- **Christopher J. Davies**, Utah State University. *Placental expression patterns of cattle non-classical MHC class I genes and proteins.*
- **Laurent Abi-Rached**, Stanford University. *Species-specific evolution of NK cell receptors for MHC class I.*


**Important Dates:**
- Early Registration Opens January 9, 2009- $650.00
- Early Registration Graduate Students - $400.00
- Late Registration Fee Begins – May 16, 2009 - $800.00
- Late Registration Graduate Students Fee Begins – May 16, 2009 - $600
- Abstract Submission Site Opens - February 1, 2009
- Abstract Submission Site Closes - June 1, 2009

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**Conference for Research Workers in Animal Diseases (CRWAD),** December 6-8, 2009, Chicago Marriott Downtown Magnificent Mile. [http://www.cvmbs.colostate.edu/mip/crwad/index.htm](http://www.cvmbs.colostate.edu/mip/crwad/index.htm)
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