The primary objective of any poultry production system is to optimize the economic efficiency of converting poultry feed into human food. Highly successful breeding and selection programs have provided the platform for annual improvements in biological efficiency as measured by feed conversion. From a biological perspective, efficiency is determined by the anatomical structure of the intestinal tract (form), and the physiological process of digestion and absorption (function) (4). The degree to which the host genes governing intestinal form and function are expressed appears to be altered by the output of the resident microbiota (flora) (2).

The physical nature of both the intestinal lining and its content display detectable changes in the early stages of disease. Villus height to crypt depth ratios have for example been used to indicate intestinal integrity (3; 5). This is possible because the length of an intestinal villus is kept constant by continuous enterocyte replacement. The delicate cells lining the intestinal tract are continuously exposed to potentially damaging luminal content and not surprisingly they require frequent replacement. It has been shown that the life span of a typical enterocyte is 3 to 4 days and consequently complete replacement of the intestinal epithelial lining occurs in this period of time by a process of cell division in the crypt area, sequential migration of the enterocyte to the tip of the villus and finally extrusion from the tip into the lumen (1). The body’s first homeostatic response to accelerated enterocyte attrition is enhanced cell division in the crypt area and to achieve this, the crypt increases in size. It stands to reason that a slight decrease in villus height to crypt depth ratio in the absence of any change in villus height is the first indicator that the conditions within the intestinal tract have

Key Words: Salmonella, vaccine, chitosan, oral vaccination

M56 Effects of feeding processed cassava waste meal on growth and apparent nutrient digestibility of broiler chickens. Stanley Omoh Omoikojo1, Daniel Maya, Clement Ebanenin Eisdahmen, Eromosele Theophilus Ehebha Ambrose Alli University, P.M.B 14, Ekpoma, Edo State, Nigeria

In an eight-week feeding trial, four broiler starter and finisher diets formulated to contain 0, 15, 30, and 45% processed cassava waste meal (PCWM) were randomly assigned to a total of 144 Anak 2000 broiler chickens in a completely randomized design to assess their growth and apparent nutrient digestibility. Each treatment group contained three replicates of eight chicks each, thus making a total of twenty four chicks per treatment group. Results on the growth performance of broilers showed that only the average live weight was not significantly (P>0.05) increased by the inclusion of PCWM at the starter phase, but at the finisher phase all the growth performance indices except feed: gain ratio followed a similar pattern with that of the live weight at the starter phase. The nutrient digestibility results showed that only the apparent digestible ash was significantly (P<0.05) increased by the inclusion level of PCWM at the starter phase. At the finisher phase, the values of the apparent digestible dry matter, crude protein, crude fibre, ash and nitrogen free extract were significantly (P<0.05) increased with corresponding increase in the inclusion levels of PCWM from 0% to 45%, whereas apparent digestible ether extract values were not affected by the test diet. The over all results showed that PCWM can successfully replace maize up to 45% level in the diets of broiler chickens without any adverse effect. Key: Broilers, cassava waste meal, growth, nutrient digestibility.

Key Words: Broilers, cassava waste meal, growth, nutrient digestibility
changed sufficiently to increase the rate of enterocyte attrition. This level of challenge seldom manifests as a change in nutrient assimilation or clinical disease because intestinal surface area is not affected, but does however indicate a shift from normal. While is impossible for even an experienced clinician to detect the change in the thickness of the intestinal wall induced by an increase in crypt depth there are other changes that give insight into what is happening. Since even minor cell damage induces an inflammatory response, cell debris and inflammatory mediators, including mucus, begin to accumulate in the lumen faster than normal. Apart from causing the villi to stick together and lose optimal alignment (visible to the naked eye) the mucus and cellular debris accumulates to the point where orange coloured mucus forms aggregates or strings within the lumen.

As the severity of the intestinal challenge escalates so too does the rate of enterocyte attrition. Villus height starts to decline when the rate of enterocyte destruction exceeds the maxim capacity for replacement. At this point the intestinal wall becomes detectably thinner and the intestine loses muscle tone and tensile strength. The mucosal lining of the intestinal wall appears pale and dull giving it a parboiled appearance because of the plethora of dead or dying cells on the luminal surface. The inflammatory exudate makes the shortened villi clump together and their typical zigzag alignment is lost. At this stage there is sufficient reduction in surface area and enough villus damage to compromise intestinal function. There is a net efflux of water into the intestinal lumen, causing what is referred to as, watery enteritis. If the irritation persists of worsens, the enteritis becomes more chronic. There is an influx of inflammatory cells causing the gut associated lymphoid tissue to appear congested and the luminal content becomes dominated by mucus giving rise to a typical mucoid enteritis. At this stage enzymatic digestion and nutrient absorption is sufficiently compromised for bacterial fermentation of undigested nutrient to result in gas accumulation. Initially the intestinal content become foamy but as the ecology of the intestinal lumen deteriorates the destabilization of the microbiota manifests as the accumulation of free gas.

Changes in the composition of the intestinal microbiota have been associated with deterioration in intestinal function as measured by feed conversion efficiency. Dysbacteriosis, as it is commonly referred to in the poultry industry, became common place after the moratorium on in-feed antibiotics was introduced in the European Union. These undefined shifts in the intestinal microbiota are difficult to diagnose, even with advanced molecular techniques and yet they appear to be associated with visible intestinal changes. There are changes in the thickness, appearance, muscle tone and tensile strength of the intestinal wall. Signs of inflammation are evidenced by a parboiled appearance of the mucosal surface, the accumulation of inflammatory cell aggregates, congestion and the development of a watery to mucoid exudate in the intestinal lumen. Gas by-products of bacterial fermentation provide confirmation of ecological disturbance.

Interestingly, the phenotypic expression, or community output, of the intestinal microbiota contributes to bird performance by influencing host gene expression and feed efficiency (2). This makes it is easy to see why a detrimental change in the intestinal microbiota structure and composition, regardless of cause, can result in a deterioration in gut health and bird performance. The intestinal tract and more specifically the caeca serves as a stable bioreactor which sustains a complex web of nutrient substrate conversion facilitated by secreted enzymes and resident organisms. The stability of the intestinal microbiota is consequently governed by the amount and type of substrate. As with any hindgut fermenter the chicken caeca is designed to support organisms that aid in digestion of the non-digestible components of the diet but unfortunately, such conditions are very suitable for many of the common enteric inhabitants that are potential pathogens. An oversupply of nutrient to the hindgut rapidly changes the composition of the microbiota since the resident organisms are able to shift from steady-state to exponential growth phase. Potential pathogens such as *Clostridium perfringens* gain competitive advantage under such circumstances and rapidly dominate the microbial community thus compromising intestinal health.

Astute observation on the part of the clinician can provide enough information to detect and diagnose subclinical disease in apparently healthy birds if necropsy is performed on a small sample of individuals on a regular basis at very little cost.

**Key Words:** avian intestine, GI function

**M58 Evaluation of a novel chicken astrovirus isolate in the pathogenesis of Runting Stunting Syndrome in chickens** Kyung-il Kang**, **Holly Sellers, Erich Linneman, Egbert Mundt** PDRC, University of Georgia, Athens, GA

Runting and Stunting Syndrome (RSS) is an enteric disease of unknown etiology affecting young broilers. Weight suppression and lack of flock uniformity associated with diarrhea are observed with RSS. The disease was reliably reproduced using filtered intestinal homogenates from RSS affected broilers, implying a viral etiology. Recently we reported the presence of three chicken astroviruses (CkAstv) in the intestine of the affected broilers as determined by *in situ* hybridization (ISH). The isolation of a new CkAstv in cell culture enabled us to investigate the pathogenicity of this virus isolate in broiler chickens after infection and subsequent serial passage. To this end, one-day-old commercial broiler chickens were orally inoculated with the new CkAstv. Hatchmates were inoculated with cell culture media serving as a negative control. At 5 days post inoculation, intestinal homogenates were collected. For the next passage, birds were orally inoculated with the homogenate obtained from the previous passage until passage 4. At day 5 p.i., birds were weighed and formalin-fixed paraffin-embedded tissues of the duodenal loop were analyzed by ISH and microscopic examination. During the first passage where chickens were inoculated with the cell culture adapted CkAstv, no significant weight depression was observed in comparison to the control group. Interestingly, increased numbers of diluted cysts were present and the nucleic acid of CkAstv was detected in the crypt epithelial cells by ISH. During the following passages, using intestinal homogenates from the previous passage, 6 to 8 % weight retardation was observed in the groups originating from the CkAstv inoculated group. In all inoculated groups, a CkAstv specific RT-PCR and ISH was positive, and dissatisfied cysts were observed. Our data provide strong evidence that a novel CkAstv is involved in RSS pathogenesis as it replicated in the crypt epithelial cells. Furthermore, it was concluded that the virulence of the cell culture adapted CkAstv changed during the passage as indicated by an increased weight depression.

**Key Words:** running stunting syndrome, chicken astrovirus, passage, *in situ* hybridization, etiology

**M59 Immunity against a virulent field isolate of ILTV induced by recombinant and modified live virus vaccines in commercial Layers** Victor Palomino**, **Guillermo Zavala, Sunny Cheng** University of Georgia, Athens, GA

The main objective of this research was to establish the onset of immunity and protection against Infectious Laryngotracheitis (ILT) induced by 6 different vaccination programs with recombinant and live-modified virus vaccines. One hundred and fifty Hy-Line W-36 commercial layers were randomly distributed in 7 and 8 groups of birds, vaccinated in various ways and challenged at 4 or 9 weeks of age, respectively. All the birds were vaccinated with the CVI988 Rispens) strain of Marek’s disease virus at day of age. In the case of the 4th week challenge study, the
following programs were included: non-vaccinated challenged (Group 1); Pox-LT recombinant at hatch (Group 2); HVT-LT recombinant at hatch (Group 3); TCO at 2 weeks of age (Group 4); Pox-LT recombinant at hatch + TCO at 2 weeks (Group 5); HVT-LT recombinant at hatch + TCO at 2 weeks (Group 6); and non-vaccinated non-challenged (Group 7). For the 9th week challenge study the experimental groups were similar, except that the TCO vaccination was done at 4 weeks of age and an additional group received a CEO vaccine at 4 weeks (Group 7). Tracheal swabs were collected and clinical signs were evaluated at 5 and 7 days post-challenge (DPC). Challenge virus concentration in the trachea was examined by qPCR. Clinical sign scores were compared statistically by Kruskal-Wallis and Dunn tests. Five DPC there was no statistical difference between groups 1 and 2, with groups 4 and 6 exhibiting the highest protection against ILTV in both the 4 and 9 week old studies. In addition, Group 7 also showed the highest protection along with groups 4 and 6 in the 9th week old challenge.

Key Words: Infectious laryngotracheitis, layers, immunity, HVT-LT, POX-LT

M60 Continuous passage of low pathogenic avian influenza viruses in ducks resulted in a higher frequency of amino acid mutations in the hemagglutinin of a chicken isolate in comparison to a wild bird isolate Callie Ridenour1,2, Les Jones1, Mark Tompkins1, Ralph Tripp2, Egbert Mundt1 1Poultry Diagnostic and Research Center, Department of Infectious Disease, University of Georgia, Athens, GA, 2Department of Infectious Disease, University of Georgia, Athens, GA

Studies focused on the viral evolution of low pathogenic avian influenza viruses (LPAIV) during passage from host to host to are essential for understanding the continual virus circulation in water fowl. To address this topic, different parameters were investigated in establishing a consistent duck-to-duck transmission model of LPAIV isolated from chickens (H5N2-Ck, H5N2-Ck) and a wild bird (H5N1-WB). LPAIV antibody free Pekin ducks were inoculated with an EID50 of 10^3.5/100 µl and hatch mates were added to each group 24 h p.i. for transmission. Transmission was observed only with the H5N1-WB isolate as indicated by virus isolation from contact birds. A virus titer increase was required for transmission of the H5N2-Ck isolate. Neither modification in route of inoculation nor increase in virus titer of the H5N3-Ck isolate resulted in transmission. This results indicate differences in transmission capabilities between different LPAIV in water fowl. The remaining two LPAIV were used for six continuous duck-to-duck passages. Each swab sample positive for virus isolation was used for determination of the nucleotide and deduced amino acid (aa) sequences of the hemagglutinin (HA) and neuraminidase (NA) genes to identify altered aa sequences. Twenty-one aa exchanges were identified in the HA glycoprotein of the H5N2-Ck isolate. Fourteen of these exchanges were located within the globular head domain containing functional domains for virus attachment. In contrast, the HA sequence of the H5N1-WB isolate showed only six aa exchanges while passaged in ducks. Surprisingly, the aa sequence of the NA of the H5N2-Ck isolate showed only two aa exchanges while the NA sequence of the H5N1-WB isolate remained constant. These results indicate that the HA of LPAIV isolated from chicken undergo a stronger selection process during passage in water fowl in comparison to isolates obtained from wild birds. This might help to obtain a better understanding of prerequisites necessary for optimal transmission and viral evolution of LPAIV.

Key Words: LPAIV, ducks, transmission, HA, NA


The turkey disease Bordetellosis results from an infection caused by Bordetella avium (BA), which colonizes the epithelium of the trachea of turkeys causing severe respiratory disease or coryza. This study’s objective was to evaluate the oral administration of an inactivated BA vaccine in combination with either chitosan or a proprietary modification of chitosan as an adjuvant in turkey pouls. In these experiments, day-of-hatch turkey pouls were vaccinated parenterally or orally with chitosan+BA adjuvanted bacterin, modified chitosan (MC)+BA adjuvanted bacterin, or saline control. On d 14, pouls were boosted with either subcutaneous (SQ) BA+chitosan, BA+chitosan in the drinking water (DW), or BA+MC DW. Immune response was evaluated using an ELISA to detect anti-BA IgG. In experiment 1, d 14 IgG antibody levels for groups BA chitosan SQ prime/DW boost, BA chitosan DW prime/DW boost, and BA+MC SQ prime/DW boost were significantly higher than controls. IgG levels on d 21 follow a similar trend to d 14. However, no significant differences (P<0.05) were found. In experiment 2, a similar trend was noted at d 21 with BA+MC SQ prime/DW boost having significantly higher IgG levels as compared to controls. Currently, to prevent the disease, pouls are treated with live, temperature sensitive vaccines administered by spray at day-of-hatch and again at two weeks of age. While this technique is sometimes effective, this type of product inmately has storage and administration difficulties for producers, frequently leading to compromised effectiveness and potential questions of serotype variation. Of particular interest, the present research was able to achieve meaningful responses following boost with oral administration of the inactivated antigen, leading to a host of opportunities for improved compliance and potential mass-application of inactivated vaccines through the DW.

Key Words: Bordetellosis, Bordetella avium, Chitosan, ELISA, Oral Vaccine

Nutrition II

M62 Effect of different sources and levels of supplemental manganese on growth performance and tissue mineral content of growing broiler chickens Alyssa Conly1, Alyssa Conly1, Elizabeth Koutses2 1Animal Science Department, California Polytechnic State University, San Luis Obispo, CA, 2Micronutrients, Inc., Indianapolis, IN

The purpose of this study was to determine the relative bioavailability of a newly developed inorganic manganese (Mn) supplement, tribasic Mn chloride (TBMC), for growing broiler chickens. Broilers were inoculated with an EID50 of 10^6.5/100 µl and hatch mates were added at 30, 60 and 130 ppm supplemental Mn. Body weight and feed intake were measured at 3 time points each and the tibia, bile and liver were collected at d 21 for mineral analysis. The 30, 60 and 130 ppm TBMC diets contained 79, 109 and 183 ppm Mn, respectively, while the 30, 60 and 130 MnSO4 diets contained 70, 98 and 184 ppm Mn, respectively. There was no difference in body weight or feed intake among any treatment groups at any time points. Bile and liver Mn increased with increasing levels of dietary Mn regardless of source (P<0.05). The concentration of Mn in the liver reached a plateau with the 60 ppm diets while the concentration of Mn in the tibia was highest with the 130 ppm diets. Bile Mn increased with increasing dietary Mn levels, but these differences were not significant. Bile Zn was highest in birds fed the highest level of MnSO4 (P<0.05). There were no treatment-related mortalities or leg problems. The calculated bioavailabilities of TBMC