Salmonella enterica induce an early pro- inflammatory response in chickens, but the response is short-lived, asymptomatic of clinical disease, and results in a persistent colonization of the cecum. The underlying mechanisms that control persistent colonization of chickens by *Salmonella* are unknown. We hypothesize that a tolerogenic response is induced by alterations of host signaling pathways that mediate the influx and functional activation of T regulatory (Treg) cells. Using chicken-specific kinomic immune arrays, cell isolations, and T cell suppression bioassays of infected cecal tissue, we evaluated the development of immunological tolerance in chickens infected with *Salmonella enterica* serovar Enteritidis in a persistent infection model. The induction of a tolerogenic response in the cecum infected with *S.* Enteritidis began around 4 d post-infection. The response was induced by a series of phosphorylation-mediated changes in the ceca characterized by alterations in T cell signaling (dephosphorylation of phospholipase c-γ1 [PLCG1]) and mTOR signaling pathways (increased phosphorylation of AMP-activated protein kinase [AMPK]) and blockage of IFN-g protection through the disruption of the JAK-STAT signaling pathway (dephosphorylation of JAK2, JAK3, and STAT4). Further, the response is characterized by a reduction in pro-inflammatory cytokine mRNA expression ($P < 0.05$) and an increase in anti-inflammatory cytokine mRNA expression ($P < 0.05$). Last, we found an expansion of the Treg population and subsequent immunosuppressive functions at the site of the *Salmonella* infection. These studies define a mechanism by which *Salmonella* infection influences the host responsiveness resulting in the establishment of a persistent colonization of the avian cecum. The identified tissue protein kinases also represent potential targets for future antimicrobial compounds for decreasing *Salmonella* loads from the intestines of food animals.

Key Words: *Salmonella*, kinome analysis, regulatory T cells, signaling pathways

330P Differential profile of local inflammatory response after challenge with Brazilian field isolates of avian infectious bronchitis virus. C. H. Okino, L. Brentano, M. A. Z. Mores, P. A. Esteves, and I. M. Trevisol*, *Embrapa Swine and Poultry, Concordia, Santa Catarina, Brazil.*

Avian infectious bronchitis virus (IBV) causes a worldwide economically important disease in poultry. IBV replicates primarily at the tracheal mucosae, though virus pathology at local sites of IBV replication remains poorly elucidated. The present experiment aimed to evaluate the gene expression of inflammatory mediators, and compare viral load and scores of lesions, in chickens challenged with 2 Brazilian IBV field isolates (F3736 and F3715) previously identified as variants by S1 analysis. Thirteen-day-old SPF chickens were housed in 3 isolators (G1, G2 and G3) with positive pressure. At 39 d of age, 3 chickens in G1 were mock infected with diluent media, while 5 chickens from G2 and G3 were infected with 10^5EID_{50}/bird of F3736 and F3715 strains, respectively. At 5 d post-infection, birds were necropsied and tracheal samples collected from each group; a portion was processed for histopathology and the remaining part submitted to RNA extraction. RNA was processed by RT-qPCR using SYBR Green I for relative quantification analysis of cytokines IL6, IL1β and T-bet (Th1 lineage transcription factor), and for absolute quantification of IBV S1 gene. Comparisons of the mean relative changes in gene expression were performed using the Mann Whitney test, probability level for significance was set as $P < 0.05$. Our results showed that in both groups (F3736/G2 and F3715/G3) there was a significant increase of histopathology scores and viral load, compared with negative control group (G1), though no significant differences were observed between the challenged groups. IL6 and IL1β mRNA, pro-inflammatory cytokines precursors, were significantly upregulated only in the F3715 challenged group. TBET mRNA was upregulated in both challenged groups, with highest significant increase for F3715 group. Although similar profiles of tracheal viral load and scores of lesions were observed for both challenged groups, we found an exacerbated inflammatory response for F3715 group, indicating relevant differences in the pathology of the distinct IBV genotypes studied here.

Key Words: avian infectious bronchitis virus, IBV, RT-qPCR, inflammatory response, Brazilian isolates.

331P Efficacy of commercial H120 strain vaccine against avian infectious bronchitis virus isolates from Brazil. I. M. Trevisol*, C. H. Okino, G. L. M. Mattos, F. R. F. Jaenisch, and P. A. Esteves, *Embrapa Swine and Poultry, Concordia, Santa Catarina, Brazil.*

Avian infectious bronchitis is a widespread economically important poultry disease difficult to control. One possibility is that available vaccines are not able to protect fully against some serotypes or genotypes variants, once the causative infectious bronchitis virus (IBV) readily undergoes mutations during mixed infections, resulting in the emergence of new variants. In Brazil, only live-attenuated vaccines of the Massachusetts serotype are licensed, and full protection against IBV variants remains scarcely elucidated. This study aimed to determine efficacy of commercial H120 strain vaccine against viruses classified in genomic groups distinct from the vaccine strain. Among several IBV isolates recovered from breeders or broilers with respiratory signs and/or decrease in egg production, 4 viruses clustered as a group phylogenetically distinct form of Mass serotype were selected (F3137, F3738, F2771 and F3561). SPF birds were divided into 9 groups with 13 birds each and maintained in isolators with positive pressure: G1) non-vaccinated and non-challenged; G2 to G5 were only challenged with selected isolates; G6 to G9 were vaccinated and challenged 4 weeks after. Five days post-challenge, all birds were euthanized and tracheas removed for evaluation of ciliary movement. The scores used were: 0) all cilia vigorously beating; 1) all cilia slowly beating; 2) some cilia very slowly beating; 3) no cilia beating. All birds with median ciliary scores ≤1 were classified as protected and scores ≥2 unprotected. The live vaccine is suitable for use if at least 90% of the challenged and vaccinated birds show no evidence of IBV in their trachea, while 90% or more of the control birds should have evidence of the presence of the virus. Efficacy of challenge was confirmed for all challenged controls groups (G2 to G5), as 100% of birds presented ciliary activity score 3. On the other hand, all vaccinated groups were classified as protected, with median ciliary scores ≤1. Finally, according to the criteria here applied, H120 vaccine was able to induce cross-protection against 4 avian infectious bronchitis virus isolated from Brazil.

Key Words: avian infectious bronchitis, Brazilian isolates, Massachusetts vaccine

332P Supplemental organic zinc in broiler breeder diet enhances immunity in progeny. C. W. Li*[1,2], Y. M. Guo[1,2], J. Gao[1,2], S. S. Guo[1,2], and E. C. Du[1,2], [1]*China Agricultural University, Beijing, China,* [2]*State Key Laboratory of Animal Nutrition, Beijing, China.*

To investigate the effects of high maternal zinc nutrition status on immunity in offspring chicks, 1,200 Ross 308 broiler breeders (45-wk-old) were allocated randomly into 5 groups fed basal diets (containing 26.80~33.52 mg/kg zinc) supplemented with 0, 50, and 300 mg/kg zinc in either organic (methionine hydroxy analog chelated zinc, MHA-Zinc)