must, therefore, be anintegral part of any pandemic preparedness plan. To address this need, weconstructed a consensus hemagglutinin (HA) sequence of H7N3, H7N7, and H7N9based on the data available in the NCBI in early 2012-2015. This artificialsequence was then optimized for protein expression before being transformedinto an attenuated auxotrophic mutant of Salmonella Typhimurium, JOL1863 strain. Immunizing chickens with JOL1863, delivered intramuscularly, nasally ororally, elicited efficient humoral and cell mediated immune responses,independently of the route of vaccination. Our results also showed that JOL1863deliver efficient maturation signals to chicken monocyte derived dendriticcells (MoDCs) which were characterized by upregulation of costimulatorymolecules and higher cytokine induction. Moreover, immunization with JOL1863 inchickens conferred a significant protection against heterologous H7N1 viruschallenge as indicated by lower morbidity and viral shedding.

Conclusions: We conclude that this vaccine, based on aconsensus HA, could induce broader spectrum of protection against divergent H7influenza viruses and thus warrants further study.

P-004

Innate immunity by live attenuated *Salmonella* Typhimurium and it protection against lethal infection with H1N1 influenza virus

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Introduction: Pre-stimulation of toll-like receptors (TLRs) byagonists has been shown to increase protection against influenza virusinfection.

Materials and Methods: In this study, we evaluated the protective responsegenerated against influenza A/Puerto Rico/8/1934 (PR8; H1N1) virus by oral and nasal administration of liveattenuated *Salmonella enterica* serovar Typhimurium, JOL911 strain, in mice.

Results: Oral and nasal inoculation of JOL911 significantly increased the mRNA copy number of TLR2, TLR4 and TLR5, anddownstream type I interferon (IFN) molecules, IFN-α and IFN-β,both in peripheral blood mononuclear cells (PBMCs) and in lung tissue. Similarly, themRNA copy number of interferon-inducible genes (ISGs), Mx and ISG15, were significantly increased in both the orally andthe nasally inoculated mice. Post PR8 virus lethal challenge, the nasal JOL911 and the PBS control group miceshowed significant loss of body weight with 70% and 100% mortality,respectively, compared to only 30% mortality in the oral JOL911 group mice. Post sub-lethal challenge, the significant reduction in PR8 virus copy numberin lung tissue was observed in oral [on day 4 and 6 post-challenge (dpc)] andnasal (on 4 dpc) than the PBS control group mice. The lethal and sub-lethalchallenge showed that the generated stimulated innate resistance (StIR) in JOL911 inoculated mice conferred resistanceto acute and initial influenza infection but might not be sufficient to preventthe PR8 virus invasion and replication in the lung.

Conclusions: Overall, the present studyindicates that oral administration of attenuated *S.* Typhimurium canpre-stimulate multiple TLR pathways in mice to provide immediate early StIRagainst a lethal H1N1 virus challenge.

P-005

Quinolone susceptibility and genetic characterization of *Salmonella enterica* subsp. *enterica* isolated from pet turtles

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Introduction: Turtle-borne *Salmonella enterica* owns significance as a leading cause in human salmonellosis. It was found to cause a variety of illnesses ranging from common food poisoning to severe typhoid fever. Quinolones are often used to treat invasive salmonellosis in humans and animals, but a significant number of cases have been detected with isolates of resistant or reduced susceptibility.

Materials and Methods: A total of 21 strains of *S. enterica* subsp. *enterica* were isolated from six commercially popular pet turtle species by means of fecal enrichments. The susceptibility of nalidixic acid, ciprofloxacin, ofloxacin, and levofloxacin was tested. PCR was used to screen plasmid mediated quinolone resistance genes and mutations in quinolone resistance determining region (QRDR).

Results: The majority of the isolates were susceptible to all tested quinolones except three isolates showing reduced susceptibility to nalidixic acid. In genetic characterization, none of the isolates were positive for *qnr* or *aac(6')-Ib* genes and no any target site mutations could be detected in *gyrA, gyrB,* and *parC* quinolone resistance determining regions (QRDR). In addition, neighbor-joining phylogenetic tree derived using *gyrA* gene sequences exhibited two distinct clads comprising; first, current study isolates, and second, quinolone-resistant isolates of human and animal origin.

Conclusions: All results suggest that *S. enterica* subsp. *enterica* isolated from pet turtles are not resistant to tested quinolones and their QRDRs were genetically more conserved than that of quinolone-resistant strains.

References

[1] Bosch S, Tauxe RV, Behravesh CB. Turtle-Associated