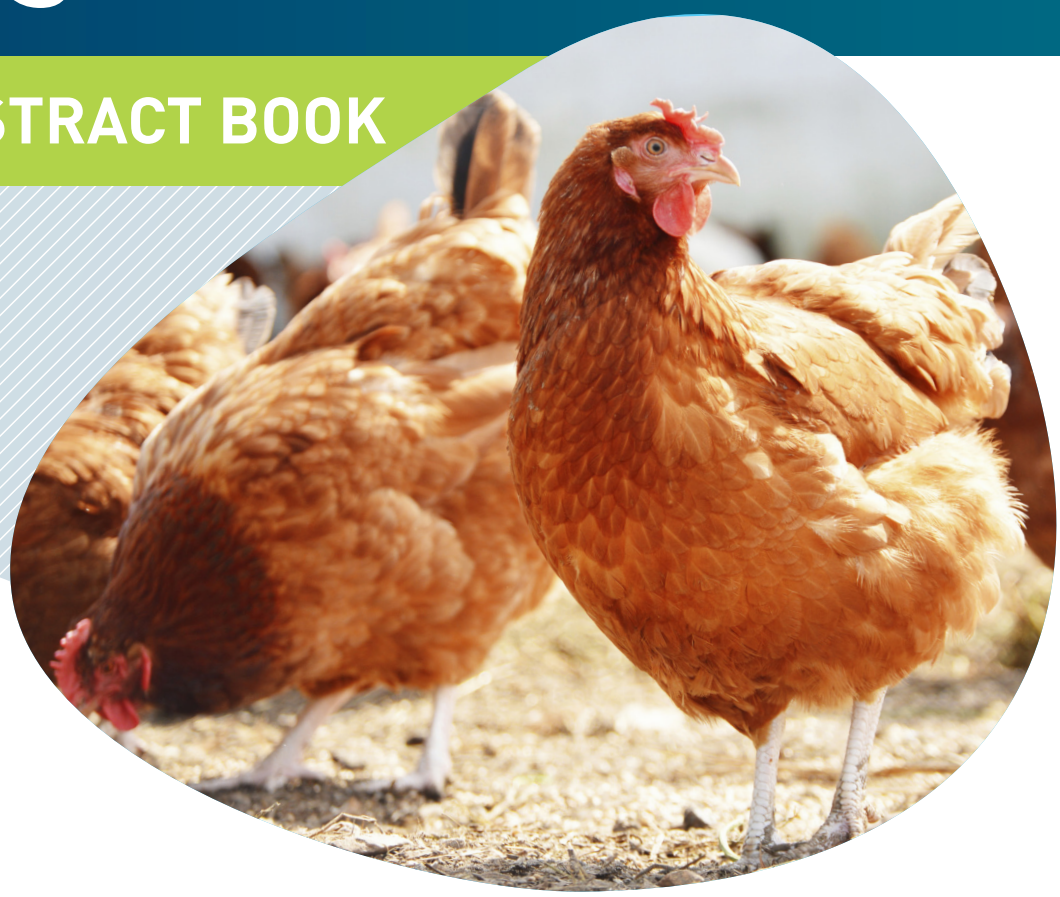
MICROBIOLOGY SOCIETY

FOCUSED MEETING 2016

Molecular Biology and Pathogenesis of Avian Viruses

POSTER ABSTRACT BOOK



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Focused Meeting 2016: Molecular Biology and Pathogenesis of Avian Viruses
Poster Abstracts

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P1

Combined hexon- and fiber-based analysis provides new insights into the molecular profile of specific FAdV genotypes consolidated for pathogenesis of inclusion body hepatitis

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Particular fowl aviadenoviruses among the five species (FAdV-A to -E) and their affiliated serotypes (FAdV-1 to -8a, -8b to -11) have been qualified as primary aetiologic agents. While hepatitis-hydropericardium syndrome and adenoviral gizzard erosions are confined to certain FAdV-C and FAdV-A strains respectively, association of inclusion body hepatitis (IBH) with FAdV-D and -E is documented within Europe mainly by individual reports.

To elucidate relevance of specific genotypes, 48 FAdVs recruited from actual IBH outbreaks were subjected to molecular typing, by performing the first combined analysis of hexon and fiber genes in such isolates.

Hexon-based analysis coherently identified genotypes harmonizing with FAdV-2/-11 or FAdV-8a and -8b, substantiating those as main agents of IBH.

High conservation in both target genes suggests a close genetic relationship of circulating FAdV-D IBH strains, whereas more strongly diverging FAdV-E IBH strains are distinguished by a serotype-characteristic pattern within the fiber.

Besides a more conclusive demarcation of acknowledged serotypes, fiber-based phylogeny revealed exceptional FAdV-E isolates with deviating hexon and fiber subtype specificities, an information missed by conventional typing. Whole genome sequence analyses indicate greater overall genomic identity of such strains with their respective fiber-specific type, despite high intertype conservation throughout FAdV-D and -E genomes with main exceptions for hexon, fiber and ORF19.

P2

Regulation of avian type I IFN genes

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Type I IFNs are comprised of multiple IFN-αs and a single IFN-β in mammals, and in chickens there are also two major type I IFNs, named ChIFN1 and ChIFN2. It is currently unclear as to whether these are the chicken equivalents of IFN-α/β because the coding sequences of ChIFN1 and ChIFN2 are equally diverged from mammalian IFN-α and IFN-β, whilst other avian IFNs show low homology to their chicken counterparts, unlike mammalian IFNα/β. We show that despite coding region divergence between avian IFNs, the avian IFN2 promoters show conservation of key elements with the mammalian IFN-β gene. Thus the activation of both mammalian IFN-β and avian IFN2-like genes requires the co-ordinated activation of both NF-κB and IRF-3/7, in contrast to the mammalian IFN-α and avian IFN1-like genes which are not dependent upon NF-κB activation. We will also show elements within each ChIFN promoter which are essential for maximum induction. These data suggest that the need to retain more than one distinct pathways of type I IFN gene activation has been retained through evolution.

P3

Identification and characterization of a chicken TRIM21-like protein

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Tripartite motif proteins (TRIM) are a multigenic family playing a role in numerous cellular processes including antiviral defense. Conservation of TRIM proteins between birds and mammals suggests that orthologous proteins might be responsible for similar functions. Among these TRIM proteins, mammalian TRIM21 is an antiviral cytosolic receptor that detects incoming antibody-bound pathogens and directs them for proteasomal degradation. To test whether chickens express an ortholog of the mammal TRIM21 pull down assays using chicken antibodies as bait were performed. Mass spectrometry analysis of IgY-immunocomplexes led to the identification of TRIM15 as a binding partner of IgY antibodies. Coimmunoprecipitation experiments confirmed the interaction between TRIM15 and IgY molecules. Quantification of the binding affinity showed that the PRYPSRY domain of TRIM15 interacted potently with IgY with a Kd value in the submicromolar range and presenting a stoichiometry of two PRYSPRY domains for one homodimeric IgY molecule. Crystal structure of the PRYSPRY domain solved at a resolution of 2.1 Angstrom revealed a classic PRYSPRY structure with a backbone made of compact beta folds with extended loops likely responsible for the specificity of the interaction. Investigations aiming to probe for antiviral properties of the chicken TRIM15 are ongoing.

P4

Chicken IFITM proteins restrict the replication of the non-enveloped avian birnavirus, IBDV, *in vitro*

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Interferon inducible transmembrane (IFITM) proteins are located in the plasma and endosomal membranes and are known to restrict the replication of viruses in host-cells. Studies in mammalian cells suggest they inhibit the entry of both enveloped and non-enveloped viruses, however surprisingly little is known about the antiviral function of avian IFITM proteins. In order to address this, we characterised the effect of chicken IFITM (chIFITM) proteins on the replication of the non-enveloped avian birnavirus, infectious bursal disease virus (IBDV), *in vitro*. The expression of chIFITM 1, 2, and 3 mRNA in avian DF-1 cells significantly increased following infection with IBDV strain D78 by 51, 21, and 5 -fold respectively ($p < 0.05$). In cells that were pre-treated with siRNA constructs to knock-down chIFITM 1, 2 and 3 expression, the replication of D78 increased by an average of 2.3, 3.8 and 1.9-fold respectively, compared to cells treated with a scrambled siRNA control ($p=0.01$, 0.01 and 0.07). Moreover, in cells transiently overexpressing chIFITM1, 2 and 3 proteins, the average replication of D78 was reduced by 0.2, 0.5 and 0.7-fold respectively compared to cells that were transfected with a negative control ($p=0.01$, 0.1 and 0.01). Taken together, these data suggest that chicken IFITM proteins restrict the replication of non-enveloped IBDV strain D78 in avian cells.

P5

Morphology of avian influenza A virus

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Pleomorphism is a characteristic of influenza A virus (IAV), which can produce spherical particles of ~100nm in diameter and filaments up to 30µm. Studies on filamentous mammalian strains led to the identification of the viral matrix protein M1 as the main determinant of this phenotype. Evidence suggests that the filamentous phenotype, seen in most human strains, may facilitate movement of virions across the mucous barrier, thereby affecting viral pathogenesis and transmission. Conversely, morphology of avian strains remains unknown. Therefore, the aim of this project is to determine the phenotype of avian IAV by examining the morphology of avian strains and to correlate this with M1 sequence variation. Mammalian and avian cells infected with fully avian and reassortant viruses (constructed in a PR8 background with segment 7 of avian strains) were imaged by confocal microscopy. Interestingly, avian strains appear to be as pleomorphic as mammalian strains, regardless of cell origin. Sequence alignments suggest that residues 41 and 95 of M1 (as for mammalian viruses) determine the filamentous phenotype of avian strains. These results show that the filamentous phenotype is not restricted to mammalian strains, making it important to perform further studies on how this correlates with pathogenesis and spread.

P6

Making 'flu-friendly" eggs for influenza vaccine production

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Culture in embryonated eggs still provides the mainstay for producing vaccines to many viruses, including influenza virus. Approaches to increase yields focus primarily on adapting the viruses' growth properties in eggs. A key factor is the receptor preference for α2,3 or α2,6-linked sialic acid on the cell surface. Growth in the allantoic cavity typically depends on using the α2,3 receptors on the allantoic membrane. For human and some swine-tropic strains of influenza, this requires adaptation of the HA (and/or NA), which may be difficult and result in antigenic changes that are detrimental to vaccine efficacy.

We are taking the novel approach of improving the egg as a virus production vessel. Our goal is to generate genetically modified chickens that produce eggs with properties conducive to efficient growth of human-tropic influenza virus strains. Specifically, we intend to increase expression of ST6Gal1 to produce more α2,6-linked sialic acid receptors and to use inhibitory genes to cripple the innate immune response of the embryo. Our progress towards this goal will be reported.

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P7

Development a sensitive real time fast-qPCR based on SYBR® Green for detection and quantification of ChPV

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Chicken parvovirus (ChPV) is a DNA virus associated with enteric problems in poultry, principally with runting - Stunting Syndrome (RSS). ChPV has been detected in chickens since one-day old, causing principally diarrhea, lost feed conversion and mortality. The diagnose of ChPV have been carried out using molecular techniques as conventional PCR, and real time PCR. The aim of the present work was development a new faster diagnosis technique for detection and absolute quantification of CHPV based SYBR® Green Fast. Primers oriented to amplify a fragment of NS gene of ChPV were designed and used in the present work. An isolated strain of ChPV were orally inoculated in one-day chicks, and maintained for 42 days, to each seven days five chickens were slaughtered and segments of duodenum, jejunum and ileum were taken. The infected chicks showed signs of enteric disease principally diarrhea. The limits of detection of fast-PCR method were from 10^1 to 10^{10} copies of plasmid dilutions. Using the diagnosis protocol described here ChPV was detected in all intestinal segments taken, showing a high concentration of viral particles in the days 7, 14 and 21, after that the viral concentration decreased significantly in the days 28 and 35 arriving to the lowest concentration at day 42. The negative control not showed any alterations or clinical signs and the detection of ChPV was negative. The qPCR based on SYBR® Green for ChPV detection showed faster and effective for detection of the virus studied compared with other conventional molecular methods.

P8

Gene expression profiling of chicken primary B cells infected with IBDV of different pathotypes

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Infectious Bursal Disease Virus (IBDV) is a birnavirus of economic significance to the poultry industry. Infection of the bursa of Fabricius (BF) results in B cell depletion causing morbidity and mortality in infected birds and immunosuppression in convalescent birds. Host genetic background has been shown to play a role in susceptibility and resistance to disease caused by IBDV, although the genes responsible are unknown. Previous studies have used whole bursal tissue from chickens of different susceptibility phenotypes to investigate gene expression in the BF during *in vivo* infection. However, mixed cell populations in the infected BF, due to inflammation and effector immune responses, might have affected experimental outcomes. In this study, primary B cells were cultured *ex vivo* in media supplemented with CD40 ligand enabling them to grow continuously for 4 days. Microarray was used to investigate differences in gene expression of these cells during infection with IBDV vaccine strain D78, relative to mock. Data analysis showed an up-regulation of 65 genes, 21 of which were genes associated with IFN induction or signalling, including IFIT5, MX1 and STAT1 (relative fold changes 78.22, 13.95 and 2.55, respectively). Results show that our B cells are immune competent and provide a useful *ex-vivo* cell model for studying IBDV and other viruses, which can be used to identify the gene(s) that determine the different pathotypes induced by vv and attenuated IBDV strains by RNA-seq.

P9

Molecular pathogenesis of H7N7 LP-HPAIV mutation during UK outbreaks: 2008 and 2015.

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Avian Influenza (AI) is a notifiable avian disease (NAD) and a severe threat to the poultry industry. The UK has had periodic outbreaks of low pathogenicity (LP) and highly pathogenic (HP) AI. During 1959-2016 NAD included 12 HPAIV and nine LPAIV (H5&H7) outbreaks. Notably, these included two LP to HPAIV H7N7 mutational events, 2008 and 2015, in layer chickens. Three other UK chicken H7 LPAIVs did not mutate (2006-N3/2007-N2/2015-N7).

The two LP to HP mutation events generated A/chicken/England/11406/2008, [Banbury08], and A/chicken/England/26352/2015, [Preston15]. Evidence for initial H7N7 LPAIV entry followed by mutation to HPAIV was derived from disease patterns (egg-drop and mortality), pathogenesis, seroconversion along with the timing and proportions of H7N7 HPAIV shedding at the epidemiological units within the chicken premises. Furthermore, HA sequencing of specimens from Banbury08 revealed three HP polybasic CS variants: PEIPKRKKRGLF / PEIPKKKKRGLF / PEIPKKKKKKRGLF, and one LP-CS: PEIPKKRGLF. Preston15 had two HP-CS: PEIPRHRKGRGLF / PEIPRHRKRRGLF, and one LP-CS: PEIPKGRGLF.

We have employed a Banbury08 reverse genetics system in an *in ovo* chicken model to investigate the genetic and phenotypic LP/HPAIV change. Further studies are underway to understand the drivers (viral/host) of evolution for viruses that mutate from LP-HPAIVs to improve detection and control of NAD.

P10

Molecular characterisation of avian paramyxovirus 1 and pigeon paramyxovirus type 1 in the United Kingdom

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Newcastle Disease caused by virulent strains of avian paramyxovirus type 1 (APMV-1) is one of the most important avian diseases worldwide. Pigeon paramyxovirus type 1 (PPMV-1), a pigeon adapted variant of APMV-1, is endemic in most countries, including the UK. APMV-1 and PPMV-1 are distinguishable only by laboratory testing not clinical presentation. Detection of APMV-1 (eight since 2009) and PPMV-1 (4-5 p.a.) has traditionally been by virus isolation (VI) in embryonated fowls' eggs and/or primary tissue culture and conventional serotyping.

Since 2010, RRT-PCR has routinely been used as a screening tool for APMVs, and has since been refined to improve sensitivity. This methodology detected two avirulent strains of APMV-1 in the UK during 2015 (a Queensland-like APMV-1 lineage 1 [GKQGRL] followed by a lineage 2 strain [GRQGRL], in both instances aided by a 'pathostat' PCR assay which can differentiate virulent from avirulent strains of APMV-1. For PPMV-1 cases parallel testing by both VI and RRT-PCR of clinical material from 17 submissions (2014/2015) showed that these tests are directly comparable (1:1), 9/17, 53% positive. A PCR-positive result was also obtained for one VI negative sample, 10/17 (59%) - demonstrating improved, faster 2 versus7 days diagnostic sensitivity for the PCR.

P11

Game birds as a potential reservoir for the spread of Newcastle disease virus

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Newcastle disease, caused by Newcastle disease virus (NDV), an avian paramyxovirus type 1 (APMV-1), is one of the most devastating diseases of poultry. The last two incursions of NDV in the UK were in pheasants and partridges, 2005/2006. The susceptibility to NDV infection and its ability to transmit between gamebird species was assessed using two NDV isolates from these incursions. Infection of both pheasants and partridges with NDV resulted in minimal/complete absence of clinical signs. Detection of virus by real-time RT-PCR and immunohistochemistry in swabs and tissues demonstrate that NDV in game birds was highly infectious, causing systemic spread of virus, and high levels of virus shedding for up to three weeks. Transmission studies demonstrated that of the two strains only the pigeon variant virus isolate was capable of onward transmission, and only in the pheasants. However, field observations from the two separate UK outbreaks in this sector along with subclinical infections of NDV in game birds observed in the present study, indicates that these birds pose a significant risk pathway for introduction of ND to the *galliforme* production species, game bird poultry industries and mixed sector environments through subclinical propagation and spread of this notifiable avian disease.

P12

Ability of Recombinant Infectious Bronchitis Virus expressing S1 subunits from M41 and QX to confer protection against homologous challenge

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One major challenge with IBV is the continuing emergence of new serotypes. Vaccination often induces insufficient cross-protection and combinations of antigenically different vaccines are used to improve protection. Differences in the S1 region of the spike (S) protein have been linked to poor cross-protection. Previously, using a reverse genetics system, the S gene of apathogenic Beaudette was replaced with the S gene from a pathogenic strain, to produce a recombinant IBV (rIBV). When used as a vaccine, this rIBV remained apathogenic but protected SPF birds against homologous and heterologous challenge. Here, we determined if rIBVs expressing S1 subunits derived from virulent QX or M41 were protective against homologous challenge. In contrast the previous studies with rIBV expressing full length S, there were some clinical signs and ciliostasis post-challenge in the S1 vaccinated groups. At 14 dpc, vaccinated groups had higher IBV-specific antibody titres compared to challenge-only groups. Serum from the vaccinated groups (14 dpc) was subjected to epitope mapping to identify peptide mimics of epitopes present on the S1 of QX and M41. In summary, vaccination with rIBV expressing S1 subunits induced an immune response and showed partial protection against homologous challenge.

P13

Detection of avian papillomavirus from papillomatous green-winged macaw species

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Papillomavirus is a group of non-enveloped, double stranded DNA virus which has small, closed circular genome with the size of approximately 7-8 kb. Infection by high-risk subtypes of human papillomavirus (HPV) may cause cervical cancer, one of the most common cancers in women worldwide. The two capsid proteins of HPV, L1 and L2, have been studied extensively, leading to the establishment of HPV vaccines. However, characterization of papillomaviruses from avian species has still lagged behind. It is not known whether the capsid proteins of avian papillomavirus (AvPV) are as immunogenic, which may offer as a basis of a good vaccine candidate. We have detected presence of AvPV from six faecal samples of two papillomatous, local green-winged macaws (*Ara chloropterus*) by using PCR primers, FcPV (5'-CTGTTTTGCGGGAGTTGGTG-3'; 5'-GCCTCCTAACACCCTCCCTA-3') and PePV (5'-ATGTGGGGGCAGAGGTAGAT-3'; 5'-CCAGGGTGCCCTTTCATCAT-3'), with product sizes of 800 bp and 220 bp, respectively. Subsequently, primers targeting L1 and L2 genes will be used to amplify those genes before they will be cloned into pTrcHis2-TOPO expression vector. Upon successful transformation into competent *E. coli* TOP 10 cells and Western blotting analysis, future work will involve analysis of these AvPV capsid proteins for their self-assembling abilities, including localization and solubility analyses.

P14

An Oral Vaccine Candidate for Infectious Bronchitis Virus through Chloroplast Engineering of *Chlamydomonas reinhardtii*

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Infectious bronchitis virus (IBV) is prominent in countries with an intensive poultry industry. However, vaccination is only partially successful owing to the high cost and emergence of antigenic variants. The genetically tractable microalga *Chlamydomonas reinhardtii* could be an ideal candidate for the production of oral IBV peptide vaccines. This is because: i) it has high growth rates, cost effective culturing and protein production; ii) it is generally regarded as safe to eat; iii) genetic engineering of the chloroplast genome (=plastome) allows high-level expression of recombinant proteins. The main goal of this research is to investigate the production of IBV peptide vaccines in the algal chloroplast, and the efficacy of the engineered algae as a low-cost oral vaccine for poultry. A synthetic biology approach was taken to design a multi-epitope IBV vaccine gene fused with the protein adjuvant, Chlolera Toxin B (CTB) based on optimised codons for the algal plastome. A plastome mutant of *C. reinhardtii* defective in photosynthesis was transformed with the plasmid pCTB-IBV and transformant colonies were selected based on restoration of phototrophy. *C. reinhardtii* was successfully transformed with the fusion plasmid, pCTB-IBV and integration of the gene confirmed by PCR. Western blot analysis confirmed the accumulation of CTB-IBV. A preliminary immunogenicity test was carried out by a collaborating research group at the University of Liverpool. They fed lyophilised CTB-IBV transformed *C. reinhardtii* to day 0 chicks. These chicks were culled on the 28th day and the sera obtained from this test were analysed by western blot and ELISA.

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