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Poster Number: 01 *

Analyis of the intestinal microbiome of pigs divergent in feed efficiency

Abstract

Improving feed efficiency (FE) is a viable means to improve on farm profitability and reduce the environmental impact of pig production. Due to its inherent importance in gut health and nutrient breakdown the intestinal microbiome is potentially a key driver of feed efficiency. Therefore, the aim of this project was to characterize the influence of the microbiome in determining feed efficiency in pigs identified as being divergent for residual feed intake (RFI). Two trials were conducted to identify pigs that were divergent in FEwith 16 pigs identified in trial 1 (8 high RFI (HRFI), 8 low RFI (LRFI)) and 24 identified in trial 2 (12 HRFI & 12 LRFI). Intestinal microbial diversity, composition and functionality were assessed using 16S rRNA gene sequencing of three regions (Ileum, Caecum, Colon). In the ileum the less efficient HRFI pigs exhibited greater α diversity compared to the LRFI pigs based on the rare operational taxonomic units (OTUs), (Chao1) or richness and evenness (Shannon and Simpson). Compositional changes at phylum level were identified with HRFI pigs having increased *Firmicutes* in the caecum (38% vs 23%) while the LRFI pigs had increased *Bacteroidetes (70* vs. 55%). While representing less than 1% total species abundance on the colon the HRFI pigs had increased *Lentisphaerae* and *Actinobacteria* compared to the LRFI pigs. At the family level the LRFI pigs had increased *Lactobacillaceae* in the ileum compared to the HRFI group. The results in this study suggest changes in bacterial composition may be important in determining FE in pigs.

Stafford Vigors[1], John O' Doherty[2], Torres Sweeney[2]

[1]Teagasc, Dublin, Ireland. [2]University College Dublin, Dublin, Ireland

*Poster presented during flash poster presentation

Poster Number: 02*

Dynamics of cellular immune response to Non-typeable *Haemophilus influenzae* (NTHi) colonisation in pre-inflamed middle ear of *Junbo* mouse.

Abstract

NTHi is a major pathogen causing acute otitis media (AOM). Pathological chronicity of AOM increases during long-term infection in the middle-ear (ME) but, cellular immune response to bacterial colonisation in this pre-inflamed environment is poorly understood. Using the *Junbo* mouse, a characterised NTHi infection model, we analysed the response of immune cells to NTHi infection. The ME fluids (MEF) collected at day-1 to 7 from *Junbo* mice intranasally inoculated with NTHi were analysed by flowcytometry to identify the immune cells. NTHi infection significantly decreased the proportion of live cells in the MEF at day-1 and this further decreased gradually on each day up to day-7. Neutrophils were the dominant immune cells in the MEF and NTHi infection significantly increased their proportion whereas monocyte decreased. Dendritic cell population was dominated by CD11b type and was constant for first two days of infection but gradually dropped by day-7. A small proportion of eosinophils and macrophages was detected in the MEF; NTHi infection initially increased this but by day-7 their number was similar to non-NTHi inoculated MEF. T-cells were dominated by Th-cells, mostly T-regs, and numbers increased at days-4 and 5 post NTHi infection. A low proportion of NK cells was observed; NK-T cells were predominant in response to NTHi. Even in the pre-inflamed ME-environment, neutrophils are the first responder to NTHi followed by T-helper and NK-T-cells. Depletion in monocyte and dendritic cells might be a significant contributing factor to survival of NTHi in *Junbo* mouse MEF.

Pratik Vikhe, Tom Purnell, Steve Brown, Derek Hood

MRC Harwell Institute, Harwell, United Kingdom

*Poster presented during flash poster presentation

Poster Number: 03*

In vitro effects of seaweed extracts on intestinal commensals and pathogens of weaned piglets

Abstract

While the inclusion of certain seaweed extracts in weaner piglet diets leads to a beneficial gut microbial profile, the mode of action is not known. The aim of this study was to evaluate the prebiotic and antimicrobial potential of *Laminaria digitata* and *Ascophylum nodosum* extracts *in vitro*. Both extracts were two-fold diluted from 2 mg/ml to 0.25 mg/ml. The following strains were used at 10^6-10^7 colony-forming unit(CFU)/ml concentrations: *Lactobacillus plantarum*, *L. reuteri*, *Bifidobacterium thermophilum*, Enterotoxigenic *Escherichia coli* O149 and *Salmonella enterica* ser Typhimurium PT12. Each concentration of each extract and controls (0 mg/ml) were incubated for 18 h at 37 °C aerobically or anaerobically (*B. thermophilum*). Final bacterial concentrations were determined by spread plating. All experiments were carried out with technical replicates on three independent occasions. All data were logarithmically transformed and analysed using the PROC GLM (SAS 9.4). The *L. digitata* extract increased *B. thermophilum* 0.7 LogCFU/ml at 0.25 mg/ml ($P<0.05$) and ≥1 LogCFU/ml from 0.5-2 mg/ml ($P<0.05$), with no effect on lactobacilli. The *A. nodosum* extract increased *B. thermophilum* up to 0.9 LogCFU/ml at all concentrations tested ($P<0.05$). Additionally, a 0.2 LogCFU/ml increase of *L. reuteri* and *L. plantarum* was observed at 2 mg/ml ($P<0.05$) and 1mg/ml ($P<0.05$), respectively. Both extracts displayed no antimicrobial activity against ETEC or *S.* Typhimurium. In conclusion, both extracts exhibited bifidogenic activity *in vitro*, with an additional slight increase of *Lactobacillus spp*. for *A. nodosum*, indicating a prebiotic potential.

Brigkita Venardou, Mary J McDonnell, Marco Garcia-Vaquero, Gaurav Rajauria, John V O'Doherty, Torres Sweeney

University College Dublin, Dublin, Ireland

*Poster presented during flash poster presentation

Poster Number: 04*

Supplementation of a milk-protein hydrolysate combined with yeast beta-glucan has a beneficial effect on the gut microbiota compared to zinc oxide in the newly weaned piglet.

Abstract

The commercial use of zinc-oxide (ZnO) in animal-feed will be banned in the EU in next five years. The aim of this study was to investigate the effects of supplementing a newly-weaned piglet-diet with a milk-protein hydrolysate (MH), yeast beta-glucan (BG), a combination of MH+BG or ZnO on the caecal and colonic microbial composition. On the day of weaning, 40 piglets were randomly assigned to either: 1) control diet; or control diet supplemented with 2) ZnO; 3) MH; 4) BG; or 5) MH+BG (n=8 piglets/group). On day 10 post-weaning, caecal and colonic digesta were collected post-mortem. DNA was extracted from digesta; phylum, species and genus specific primers were used to amplify the 16s rRNA gene. Standard curves were generated by carrying out qPCR on serial dilutions of amplicons using the specific primers for absolute quantification of gene copy numbers. In the caecal digesta, both MH+BG and ZnO supplementation increased *Bacteroidetes* abundance ($P<0.05$) and decreased Attaching and Effacing *E.coli* abundance ($P<0.05$) whereas only ZnO was associated with a decrease in *Bifidobacteria* spp. abundance ($P<0.008$) compared to the un-supplemented controls. In the colonic digesta, only ZnO supplementation was associated with reduced *Bifidobacteria* spp. abundance relative to the un-supplemented controls ($P<0.05$). Therefore, the MH+BG and ZnO supplementations had similar effects on the microbial composition. However, ZnO had a negative effect on beneficial *Bifidobacteria* spp. Hence we conclude that the MH+BG combination has a more beneficial effect on the gut microbiota than ZnO.

Anindya Mukhopadhya[1], John O'Doherty[2], Marion Ryan[1], Torres Sweeney[1]

[1]School of Veterinary Medicine, UCD, Dublin, Ireland. [2]School of Agriculture and Food Sciences, UCD, Dublin, Ireland

*Poster presented during flash poster presentation

Poster Number: 05*

Use of intestinal organoids to identify potential therapeutic approaches for human norovirus

Abstract

Human noroviruses (HuNoV) have been identified as the leading cause of viral gastroenteritis worldwide. Despite their huge socioeconomic impact, there is still no licensed antivirals or vaccines against HuNoV. Using the recently developed stem cell derived culture system, we have identified potential therapeutic approaches for HuNoV. First, the HuNoV organoid culture system was established in vitro using intestinal epithelial cells from either the proximal duodenum or terminal ileum using HuNoV GII positive clinical samples. We then evaluated the ability of a purine ribonucleoside analog (CMX521), identified by Chimerix, to inhibit HuNoV replication in the organoids. CMX521 was effective against HuNoV GII.3 and GII.4 in human mucosal stem cell derived organoids. Additionally, this compound has also shown a pan-genotypic activity against other caliciviruses tested. Furthermore, we demonstrated that HuNoV replication is restricted by the innate immune response and that modification of the innate immune response can impact on norovirus replication. These works identify therapeutic approaches for the treatment and prevention of human norovirus infection.

Myra Hosmillo[1], Yasmin Chaudhry[1], Komal Nayak[2], Randall Lanier[3], Matthias Zilbauer[4], Ian Goodfellow[5]

[1]Division of Virology, University of Cambridge, Cambridge, United Kingdom. [2]Department of Pediatrics, University of Cambridge, Cambridge, United Kingdom. [3]Chimerix Inc, Durham, North Carolina, USA. [4]Department of Pediatrics, University of Cambridge, University of Cambridge, United Kingdom. [5]Division of Virology, University of Cambridge, Cambridege, United Kingdom

*Poster presented during flash poster presentation

Poster Number: 06*

Investigating the biological substrates of the*Salmonella* GDSL lipase autotransporter, ApeE.

Abstract

ApeE is an autotransporter protein that is conserved amongst all currently sequenced *Salmonella*. Previous work showed that ApeE is a GDSL lipase autotransporter protein that is able to cleave napthyl esters and expression of ApeE is induced upon phosphate limiting conditions. Autotransporter proteins, also known as type 5 secretion, have 3 functional domains; an N-terminal sec dependent signal sequence, secreted effector (passenger) domain and a β-barrel translocation domain. Evidence suggests that many autotransporters are important outer membrane proteins during Gram-negative pathogenesis. A recent study linked the phospholipase activity of an ApeE homolog to a potential role in virulence. As ApeE is upregulated in phosphate limited conditions, we wanted to investigate whether ApeE has phospholipase activity because to date, a biological substrate for ApeE has not been identified. Here, we use an *in vitro* recombinant protein system to determine biologically relevant substrates of ApeE and the Michaelis–Menten kinetics for these substrates, under different pH and determine the optimal conditions for enzyme activity. We show that ApeE is required for the growth of *Salmonella enterica* serovar Typhimurium in minimal growth medium with phospholipid as the sole carbon source and that ApeE can bind to host relevant lipids. These data indicate that ApeE could be important for the interaction of *Salmonella* with lipids derived from the host.

Jessica Rooke[1], Jack Bryant[1], Richard Strugnell[2], Daniel Slade[3], Ian Henderson[1]

[1]University of Birmingham, Edgbaston, United Kingdom. [2]University of Melbourne, Melbourne, Australia. [3]Virginia Tech, Virginia, USA

*Poster presented during flash poster presentation

Poster Number: 07*

The Interaction of *Pseudomonas aeruginosa* with Mucin

Abstract

The abundance of mucus in the lungs of CF patients promotes infection with the opportunistic pathogen *Pseudomonas aeruginosa.* We hypothesise that the interaction of *P. aeruginosa* with mucin plays a role in initiation of colonisation and maintenance of chronic bacterial infection in CF. Using high throughput microarray platforms containing mucins from animal species and neoglycoconjugates we assessed binding of a *P. aeruginosa* CF isolate strain, S1961, and the well characterized strain, PAO1. Infection of A549 cells grown under conditions that do and do not promote mucin secretion was also assessed.

The CF-isolate, strain S1961, bound with greater intensity to animal mucins compared with binding of strain PAO1. Strong binding to mucin from chicken large intestine and proximal small intestine, which have an abundance of suphate moieties, occurred. Strain S1961 bound with significantly less intensity than strain PAO1 to many of the NGCs tested, suggesting that the multivalent presentation of glycans on mucin and/or the presence of novel glycans on mucins may mediate binding of strain S1961. Profiling of Tn5 insertion mutants revealed potential roles for both FliC and the lectin PilY1 in the process of mucin binding. Mucin secretion by A549 cells altered the spatial orientation of the bacteria on the cells and caused a reduction in the numbers of strain PAO1, but not S1961, that bound to the cells.

Differences in the adhesins expressed on strains and in the glycosylation profile of mucins may explain why some people become chronically infected with *P. aeruginosa* while others do not.

James O' Connor Moneley, Marguerite Clyne

University College Dublin, Dublin, Ireland

*Poster presented during flash poster presentation

Poster Number: 08*

Ethanolamine Metabolism in Urinary Tract Infection

Abstract

Urinary Tract Infections (UTIs) result from ascent of gut microbes into the bladder.. Bacterial microcompartment-mediated metabolism of small carbon compounds promotes overgrowth of Enterobacterial pathogens during acute enteric infection. We investigated a metabolic role for the Eut operon in *E. coli* urinary tract infection.

Methods

103 clinically infected urine samples containing visible bacteria and white cells were cultured. Sixty-one *E. coli* strains were isolated, 47 sequenced. Urine was assayed for ethanolamine and bacterial metabolites by HPLC, Eut operon expression by RT-PCR and cytokines by ELISA. 12 non-infected urines were analysed . *eutR* and *eutB* knockouts were made in selected strains by transduction from Keio library strains.

Results

The Eut operon was conserved and uninterrupted in 46 of 47 *E. coli* strains sequenced, ethanolamine enhanced growth in nitrogen-limited minimal medium in forty-five strains. Mean urine ethanolamine level was 0.5 mM in infected urine. Mean infected urine acetate content was 33mM. *eutB* and *eutR* mRNA expression detected in infected urine correlated with urine ethanolamine levels (Spearman's rank correlation coefficient r=0.7321 p=0.0027 n= 15, and r=0.4696, p=0.0274 , n= 22 respectively). All *E.coli* infected urines contained raised IL6, IL8, IL-1beta compared with non-infected urine. 0.5 mM ethanolamine added to artificial urine medium enhanced growth of wild-type *E. coli* but not delta*eutB* and delta*eutR* mutants.

Conclusion

Ethanolamine utilisation is a highly conserved property of *E. coli* causing urinary tract infection. Ethanolamine is present in urine at sufficient levels to offer a competitive growth advantage to *E. coli*. Acetate is consistently present in infected urine.

Katherine Dadswell[1], Sinead Creagh[2], Edward McCullough[2], John MacSharry[1], Michael Prentice[1,2]

[1]University College Cork, Cork, Ireland. [2]Cork University Hospital, Cork, Ireland

*Poster presented during flash poster presentation

Poster Number: 09*

High resolution mutagenesis analysis of the genetic requirements for anaerobic growth of *Escherichia coli* in the presence of nitrate

Abstract

Escherichia coli can use host-derived nitrate as a terminal electron acceptor for anaerobic respiration during growth in the inflamed gut. Therefore we wanted to gain a better understanding of the molecular requirements for the growth of *E. coli* in the presence of nitrate. To do this, we constructed a Tn5 transposon mutant library containing 193,495 unique insertion sites in *E. coli strain* MG1655 and transposon directed insertion sequencing (TraDIS) was used to analyze this library after anaerobic growth in M9 minimal medium with glucose in the presence or absence of nitrate. This revealed 749 and 192 genes with significantly altered log fold-change (logFC) values following growth with or without nitrate, respectively. Gene set analysis was applied to these gene lists and requirements for fitness under both growth conditions were compared. This revealed that, alongside a requirement for mixed acid fermentation in the absence of nitrate and nitrate-dependent respiration pathways in the presence of nitrate, a large proportion of genes were uncharacterized genes. Indeed uncharacterized genes formed 34.31% of the list of genes selected following growth in the presence of nitrate. This indicates that much is yet to be understood about gene networks employed during anaerobic growth in the presence of nitrate and these genes may represent potential intervention targets for control of *E. coli* growth during inflammation.

Finbarr Buttimer[1], Emma Smith[1], Christine Boinnett[2], Amy Cain[2], Francesca Short[2], Trevor Lawley[2], David Clarke[1]

[1]University College Cork, Cork, Ireland. [2]Wellcome Trust Sanger Institute, Cambridge, United Kingdom

*Poster presented during flash poster presentation

Poster Number: 10*

Investigating the role of host-virus protein-protein interactions in determining the host specificity of the recently emerged, *Schmallenberg virus*

Abstract

Schmallenberg virus (SBV) is primarily a bovine and ovine pathogen, which first emerged in Germany in 2011 and spread rapidly, causing outbreaks of disease across most of Europe by 2014. SBV causes abortions, still births and physical deformities of foetuses and new-borns upon infection of pregnant sheep and cattle. SBV is transmitted by Culicoides midges that bite not only sheep and cattle, but also humans. Also, outbreaks of SBV have arisen in urban areas making the likelihood of human exposure to SBV high, but despite this it has not been detected in humans. One potential host determining factor of viruses is the presence or absence of host-virus protein-protein interactions (PPIs), which allow viruses to manipulate host cells or the host to stop the virus. Host species-specific PPIs of SBV nucleocapsid protein (N) were identified by mapping the interactome of SBV N in A549 (human) and SFTR (ovine) cell lines using a proteomics approach. Mass spectrometry (MS) was used to identify proteins by mapping peptide sequences against reference proteomes using MaxQuant MS analysis software and proteins interacting with SBV N were predicted by comparing protein quantities in SBV-infected samples and mock-infected controls using Perseus statistical analysis software. SBV N protein was found to interact with 27 host cell proteins, 16 of which were unique to the human cells, 6 were unique to the sheep cells and 5 were common to both cell lines. Small interfering RNA (siRNA) was used to knock down proteins predicted to interact with SBV N in A549 cells and the effect of knock downs on virus infectivity measured to identify interactors with a significant beneficial or detrimental effect on SBV replication. The siRNA screen revealed that knock down of several predicted interactors of SBV N significantly decreased (indicating they benefit replication) or increased (indicating they inhibit replication) SBV infectivity. Further investigation is required to validate predicted interactors and characterise the mechanisms of how some interactors are involved in SBV replication. Results to date have shown there are differences in the interactome of SBV N in human A549 and ovine SFTR cells and that some of these predicted interactors play important roles in SBV replication. These results support the hypothesis that PPIs may contribute to the host specificity of SBV because species-specific PPIs were identified and the siRNA screen showed some of these interactors are important determinants of SBV infectivity.

Tristan Russell[1], Kara O'Connor[1], Meg O'Connor[2], Gerald Barry[1]

[1]University College Dublin, Dublin, Ireland. [2]Dublin Institute of Technology, Dublin, Ireland

*Poster presented during flash poster presentation

Poster Number: 11*

Bacterial immobilization induces *LEE* gene expression

Abstract

Enterohaemorrhagic *Escherichia coli* pathogens represent major difficulties to health as they cause complications in animals.The locus of enterocyte effacement (*LEE)* pathogenicity island is the major player in triggering the attaching and effacing lesions that eventually induce death of host cells.Only under suitable conditions is expression of the *LEE* activated. The global regulator of *LEE* activator (GrlA) transcription is considered to be especially important in this process. The LEE1 promoter, though displaying some basal level of activity, requires GrlA for maximal expression. GrlA binds to the LEE1 promoter and activates transcription initiation *in vitro*, the fold of activation was only ~ 1.5 fold. The aim of this work was to discover factors trigger GrlA activity.It is shown bacterial attachment to host cells triggers GrlA activity to an extent not seen during planktonic growth, with up to ~20 fold activation. Data also show that the level of free unbound GrlA defines the activity of the *LEE* promoter by GrlA, and the previously characterised GrlR anti-GrlA protein merely serves to buffer the level of GrlA. Free GrlA plays a major role in activating transcription initiation at the LEE1 promoter. Results also reveal that the cytoskeleton rearrangements caused by EHEC depend on GrlA acting at the LEE1 promoter. The results point to a critical strategy in bacterial pathogenesis, whereby the microbe must know when it is attached before it turns on its “pathogenic features”. Attachment to host cells is crucial, and it seems that EHEC actually senses and knows when to start pathogenesis.

Ghadah Alsharif

King Abdulaziz University, Jeddah, Saudi Arabia

*Poster presented during flash poster presentation

Poster Number: 12*

A potential oxygen sensing prolyl hydroxylase regulates susceptibility of tetracycline antibiotics in *Pseudomonas aeruginosa*

Abstract

Antibiotic resistance in the opportunistic pathogen *Pseudomonas aeruginosa* (*P. aeruginosa*) represents a global healthcare threat. *P. aeruginosa* infections frequently occur in hypoxic microenvironments in host tissues such as the lung and skin. Recently a prolyl hydroxylase enzyme (Pseudomonas prolyl hydroxylase, PPHD) was identified in *P. aeruginosa* as a potential oxygen sensor.

We compared minimal inhibitory concentrations (MIC) of wild type (PAO1) and PPHD knock out mutant (PAO310) for a panel of antibiotics by VITEK2, E-test and micro broth dilution. For tetracycline We found its antibiotic resistance dramatically increased when PPHD is absent (wild type MIC 16 µg/ml, PPHD knock out mutant MIC 256 µg/ml). For other members of the tetracycline family, doxycycline and minocycline, the PPHD knock out mutant also has an increased resistance towards these antibiotics. Treating wild type *P. aeruginosa* with a panel of prolyl hydroxylase inhibitors did not change tetracycline MIC, suggesting that the increase in resistance in the PPHD knockout mutant is independent of hydroxylation but dependent on the presence of the PPHD protein itself. For colistin we found an increase in MICs with two pharmacological hydroxylase inhibitors suggesting here a hydroxylation dependent mechanism of PPHD.

We hypothesize, based on our data, that PPHD plays a role in determining *P. aeruginosa* pathogenicity through the regulation of antibiotic resistance. By understanding how PPHD determines bacterial virulence we will be able to target these processes in the identification of new anti-infective agents.

Bettina Schaible[1], Kirsten Schaffer[2], Cormac Taylor[1]

[1]UCD, Dublin, Ireland. [2]St. Vincent's University Hospital, Dublin, Ireland

*Poster presented during flash poster presentation

Poster Number: 13*

Pre-symptomatic changes in the microbiome of cows developing postpartum endometritis

Abstract

Bacterial colonization of the uterus affects a high percentage of animals during the postpartum period. Failure to clear bacterial infection within three weeks postpartum is defined as postpartum endometritis. This study focused on the composition and dynamics of the microbiome and its relationship with the health status of the reproductive tract of dairy cows at the peripartum period. For this, 16S rDNA amplicons (V1-V3 regions) obtained from bacteria associated to mucus from vagina and uterus were analysed using both DNA profiling and pyrosequencing. Significant differences were observed at different times in animals that developed postpartum endometritis, especially at 7 days postpartum. In contrast to healthy cows, a remarkable high similarity between vaginal and uterine microbiomes was observed at 7 days postpartum, suggesting a delay in the differentiation of the microbiome in animals developing endometritis. A massive reduction in bacterial diversity associated with the presence of highly dominant species was found as a pre-symptomatic signature in the reproductive tract of animals developing endometritis. Biomarkers associated to this dysbiotic event hold promise for early diagnostics of postpartum endometritis.

This project was supported by the Irish Department of Agriculture, Food and the Marine Reference number: 13–S–472

Raúl Miranda-CasoLuengo[1], Erin J. Williams[2], Junnan Lu[1], Aleksandra A. Miranda-CasoLuengo[1], Stephen D. Carrington[3], Alexander C.O. Evans[4], Wim G. Meijer[1]

[1]UCD School of Biomolecular and Biomedical Science and UCD Conway Institute, University College Dublin, Dublin, Ireland. [2]The Roslin Institute and Royal (Dick) School of Veterinary Studies, University of Edinburgh, Easter Bush Campus, Midlothian, Scotland, United Kingdom. [3]Veterinary Sciences Centre, UCD School of Veterinary Medicine, University College Dublin, Dublin, Ireland. [4]UCD School of Agriculture and Food Science, University College Dublin, Dublin, Ireland

*Poster presented during flash poster presentation

Poster Number: 14*

Filamentous influenza virion morphology confers increased resistance to inhibition by respiratory mucus.

Abstract

Virions from low passage clinical isolates of Influenza A virus are pleomorphic. Pleomorphic populations of virus particles can range from spherical particles that are 150 nm in diameter, to filamentous virions that extend to ≥1 µm in length. The pleomorphic phenotype can be lost when the virus undergoes passage in either embryonated chicken eggs or MDCK cells, where the vast majority of virions become uniformly spherical. This suggests that filamentous particles confer an advantage *in vivo*, while a spherical phenotype is more advantageous *in vitro*. Recent studies have demonstrated a correlation between a filamentous morphology and increased transmissibility in different animal models (Lakdawala *et* al., 2011; Campbell *et al*., 2014). However, the biological significance of filamentous virions is not well understood. Isogenic viruses displaying either a pleomorphic or spherical-only virion phenotype were generated. We demonstrate that virus stocks that contain filamentous particles are more resistant to inhibition by respiratory mucus compared to stocks with spherical-only particles. We also demonstrate that the increased resistance to inhibition is dependent on a filamentous morphology and the action of the viral sialidase enzyme, neuraminidase. Despite retaining greater levels of infectivity in a plaque reduction assay, viruses with a filamentous phenotype do not have significantly increased replicative capacities in human nasal epithelial cells.

Kieran Dee, Neal Leddy, Kim Roberts

Trinity college, Dublin, Ireland

*Poster presented during flash poster presentation

Poster Number: 15*

Probiotic modulation of inflammatory microRNA-21 in a mouse model of colitis

Abstract

A complex interplay exists between the gut microbiota, pathogenic bacteria and the host organism within the intestinal environment. The homeostasis of the intestinal epithelium and resident immune cells are central to this relationship, and are influenced dynamically by host nutrition, infectious insult, and commensals.

The *Lactobacilli* are a genus of commensal bacteria with a well-established reputation for producing health benefits in the host. Each probiotic strain of *Lactobacillus* affects the host in unique ways, through immunomodulation, bacteriocin production, stimulation of mucin secretion and competitive inhibition of pathogens. Here, we show that *L. salivarius* has emerged as an effective probiotic, protecting against severe pathology in a murine model of inflammatory bowel disease (IBD) as well as promoting recovery from DSS-induced colitis. Furthermore to protection against colitis, treatment with *L. salivarius* was shown to reduce expression of a microRNA, miR-21.

MicroRNAs are small, non-coding RNAs which serve to post-transcriptionally regulate gene expression. MicroRNA-21 is abundantly present in mammals, and its expression has been associated with various cancers and inflammatory diseases. Indeed, studies have shown a role for miR-21 in directly influencing the pathogenesis of IBD. We postulate therefore that *L. salivarius* reduces expression of miR-21 to protect against loss of intestinal homeostasis. This novel interaction opens the possibility that the relationship between commensals and miR-21 may be examined further to enable therapeutic modulation of host microRNAs through probiotic treatment.

Michelle Williams, Dr. Sinead Corr

Trinity College Dublin, Dublin, Ireland

*Poster presented during flash poster presentation

Poster Number: 16

Spontaneous loss of IL28RA expression leads to an enhanced HuNoV replication *in vitro*

Abstract

Human norovirus (HuNoV) is the main cause of acute gastroenteritis worldwide and no therapeutics are currently available. Interferon lambda (IFN-λ) is a key cytokine controlling murine norovirus infection *in vivo* and *in vitro*. Here, we utilize a HuNoV replicon in human gastric tumor (HGT) cells to identify host factors involved in promoting or inhibiting HuNoV replication. We observed that an IFN-cured population of replicon-harboring HGT cells (HGT-cured) was enhanced in their ability to replicate transfected HuNoV RNA compared to parental HGT cells, suggesting that differential gene expression in HGT-cured cells created an environment favoring the replication of viral RNA. Microarray was used to identify genes differentially regulated in HGT-NV and HGT-cured compared to parental HGT cells. We then observed that the IFN-λ receptor alpha (IL28RA) expression was undetectable in HGT-cured cells and significantly reduced in HGT-NV cells. All three cell lines responded to exogenous IFN-β by inducing interferon stimulated genes (ISGs), however, HGT-NV and HGT-cured failed to respond to exogenous IFN-λ. Similar observation was made in U2OS-NV replicon harbouring cells. Reconstitution of IL28RA rescued HGT-NV and HGT-cured cells response to IFN-λ. We therefore conclude that type III IFN is important in inhibiting HuNoV replication *in vitro* and that the loss of IL28RA enhances replication of HuNoV.

Sabastine Arthur, Frederic Sorgeloos, Myra Hosmillo, Ian Goodfellow

University of Cambridge, Cambridge, United Kingdom

Poster Number: 17

Periopathogenic bacteria in patients undergoing implantation therapy in dentistry

Abstract

Aim of the study: The purpose of the study was to compare presence of periopathogenic bacteria in sites intended for implantation therapy in dentistry with success of this therapy.

Material and methods: Fifty-four patients underwent implantation therapy by titanium implants from BTLock company (Italy). Before dental implantation therapy started, saliva samples from implantation site were collected. From these samples, microbial DNA was isolated by Dneasy Blood&Tissue Kit (QIAGEN). Presence of periopathogenic bacteria *Tannerella forsythensis, Porphyromonas gingivalis, Prevotella intermedia, Prevotella nigrescens, Aggregatibacter actinomycetemcomitans* and *Capnocytophaga ochracea* was determined by qualitative PCR using periopathogenic bacteria specific primers.

Results: *Prevotella nigrescens* was found in 64.3 % of patients, *Prevotella intermedia* was found in 28.6 % of patients*, Tannerella forsythensis* was found in 25 % of patients, *Porphyromonas gingivalis* was found in 14.3 % of patients, *Capnocytophaga ochracea* was found in 12.5 % of patients and *Aggregatibacter actinomycetemcomitans* was found in 1.8 % of patients. Implantation therapy was successful in all patients.

Conclusion: Presence of periopathogenic bacteria in implantation sites was common in patients undergoing implantation therapy. Despite this fact, there was no problem with implant healing and implantation therapy was successful in all patients.

Acknowledgement: The study was supported by project PROGRES Q29/1LF (First Faculty of Medicine, Charles University, Czech Republic), by project 15-37368G (Grant Agency, Czech Republic) and by project 17-30753A (Czech Health Research Council, Ministry of Health, Czech Republic).

Stepan Podzimek, Tatjana Janatova, Edgar Oganessian, Jaroslav Mysak, Jana Duskova

Institute of Dental Medicine, First Faculty of Medicine and General University Hospital in Prague, Charles University, Prague, Czech Republic

Poster Number: 18

Identifying the inhibition of TIR Proteins involved in TLR signaling as an anti-inflammatory strategy

Abstract

Toll/IL1 receptor (TIR) adaptor proteins continue to be an integral part of Toll-like receptors (TLR) signaling involved in inflammation. Signaling is likely to be initiated by these TIR adaptors when they are recruited to a TIR–TIR interface formed by TLR dimerization. Among these; myeloid differentiation factor-88 (MyD88), MyD88 adapter-like protein (Mal), TIR domain-containing adaptor protein inducing interferon-β (TRIF), TRIF-related adaptor molecule (TRAM), play pivotal roles at many steps in the signaling events leading to inflammation. The presence of the conserved BB loop residues in the TIR domain of all these important adaptor proteins make them possible targets to be inhibited by synthetic compounds. We have designed compounds based on an already known MyD88 TIR dimerization inhibitor, T6167923 which binds well not only to the original target but also to the TIR domains of Mal,TRIF and TRAM. The designed inhibitors are based on modifications of bromophenyl-sulfonyl-thiophenyl-piperazine-carboxamide series of compounds. We have further suggested modifications in these high-affinity compounds for efficient absorption inside the body. Further, a pharmacophore model highlighting important structural interaction features has been developed. The screened compounds are better in binding to the TIR proteins then the parent compound and hence are good starting points for multi-TIR inhibition.

uzma Saqib

Indian Institute of Technology, Indore, India

Poster Number: 19

Investigating the role of universal stress proteins in the pathogenesis of *Burkholderia cepacia* complex and their contribution to chronic infection in Cystic Fibrosis.

Abstract

Burkholderia cepacia complex (Bcc) is a group of 21 closely related species of Gram-negative bacteria that cause chronic infections in people with cystic fibrosis (CF). Bcc infections are rarely eradicated and the high-level antibiotic resistance means that combatting these chronic infections is particularly challenging. The mechanisms by which Bcc survive and persist during chronic infection are not fully understood.

We previously found that a series of Irish sequential Bcc isolates from two chronically colonised CF patients increased their ability to attach to CF lung epithelial cells (CFBE41o$^-$) over time of infection. An in-depth proteomic analysis of these isolates also found 20 proteins encoded within the 50-gene low-oxygen-activated (*lxa)* locus were consistently upregulated over time of infection suggesting an important role for the *lxa* locus in chronic infection.

Six Universal Stress Proteins (USPs), were consistently upregulated in the sequential isolates and have not previously been studied in Bcc. A single gene deletion mutant (*Dpusp)* of one of these USPs showed 90% reduction in attachment to CFBE41o$^-$ cells ($p<0.005$), increased sensitivity to peroxide-induced oxidative stress ($p<0.0001$) and low pH ($p<0.05$) relative to wild-type Bcc, all relevant to the CF lung and macrophage environment. In the U937 human macrophage-like cell line, there was a reduction in both uptake and survival of *Dpusp* in comparison to wildtype, suggesting the USP plays a role in the intra-macrophage survival of Bcc. Overall, these proteins, previously associated with adaptation to low-oxygen conditions may play a considerable role in Bcc pathogenesis and its adaption during chronic infection.

Andrew O'Connor, Siobhán McClean

University College Dublin, Dublin, Ireland

Poster Number: 20

Impact of bacterial sialic acid metabolism on mucosal adaptation in the gut

Abstract

The colonic mucus layer is rich in sialic acid (Neu5Ac) residues terminating mucin glycans which can be used by gut symbionts as binding sites or nutrients. The human gut symbiont *Ruminococcus gnavus* has a strain specific ability to utilize these glycans. This ability is dependent on the expression of an intramolecular *trans*-sialidase (*Rg*NanH) which produces 2,7-anhydro-Neu5Ac from sialylated substrates, and is the first example of such an enzyme among gut symbionts.

We showed that, although *R.gnavus* is not able to grow on Neu5Ac as a sole carbon source, it is able to target Neu5Ac-rich regions in mucins and metabolize 2,7-anhydro-Neu5Ac to support its growth. We proposed that this mechanism provides a competitive nutritional advantage by releasing sialic acid in a form, 2,7-anhydro-Neu5Ac, that *R.gnavus* can preferentially use but that may not be accessible to other members of the gut microbiota.

More recently we showed that enteric pathogens *S.*Typhimurium and *C.difficile,* that scavenge free Neu5Ac in the mucosal niche released by other gut symbionts, cannot utilize 2,7-anhydro-Neu5Ac *in vitro*.

Our current work is focused on determining the pathway of 2,7-anhydro-Neu5Ac metabolism in gut bacteria*.* We have shown that the entire operon encoding *Rg*NanH is upregulated during *R.gnavus* growth on 2,7-anhydro-Neu5Ac or sialylated mucins, and that the encoded sialic acid aldolase uses Neu5Ac as a substrate. Investigations are on-going to characterize gene-products involved in the uptake and conversion of 2,7-anhydro-Neu5Ac to Neu5Ac. We are also investigating the mechanisms of adaptation *in vivo* using *R.gnavus*-colonized mice.

Andrew Bell[1], Louise Tailford[1], Emmanuelle Crost[1], Marie Monestier[1], Gary Rowley[2], Rob Field[3], Nathalie Juge[1]

[1]Quadram Institute Bioscience, Norwich, United Kingdom. [2]University of East Anglia, Norwich, United Kingdom. [3]John Innes Cente, Norwich, United Kingdom

Poster Number: 21

Mutual antagonism: A complex coexistence of *Aspergillus fumigatus* and *Pseudomonas aeruginosa* in the cystic fibrosis airway

Abstract

Cystic fibrosis (CF) airway infections are now known to be polymicrobial in nature and microbe-microbe interactions may play an important role in disease pathology. *Pseudomonas aeruginosa* and *Aspergillus fumigatus* are the most prevalent bacterial and fungal pathogens isolated from the CF airway, respectively. Co-colonisation with both pathogens was associated with a 13.8% reduction in FEV_1 (p=0.011), higher numbers of exacerbations (p=0.042), hospitalisations (p=0.023) and antimicrobials (p=0.014) compared to non-colonised patients and these clinical outcomes were comparable to those of patients persistently colonised with *P. aeruginosa*. Our objective was to examine the interactions between *A. fumigatus* and *P. aeruginosa*, specifically the effects of co-colonisation on biofilm formation and host pro-inflammatory responses.

Co-infection of bronchial epithelial cells (CFBE41o-) with both pathogens did not enhance IL-6 and IL-8 production beyond the levels observed following single infections. Quantification by qPCR revealed that both pathogens had mutually antagonistic effects on each other. *A. fumigatus* supernatants showed strong anti-Pseudomonal activity and gliotoxin is the main active agent. Gliotoxin showed varying levels of anti-biofilm activity towards other bacteria commonly found in the CF airways.

There are complex cross-kingdom dynamics at play in the relationship between *A. fumigatus* and *P. aeruginosa*. This competition between these species may contribute to the poor clinical outcome of CF patient's co-colonised with *A. fumigatus* and *P. aeruginosa*. Gliotoxin produced by *A. fumigatus* colonising the CF airways has anti-biofilm and anti-pseudomonal effects which may have a significant impact on the CF airway microbiome composition with potential clinical implications.

Emma Reece[1], Sean Doyle[2], Peter Greally[3], Julie Renwick[1], Siobhán McClean[4]

[1]Trinity College Dublin, Dublin, Ireland. [2]Maynooth University, Kildare, Ireland. [3]National Childrens Hospital, Tallaght, Dublin, Ireland. [4]University College Dublin, Dublin, Ireland

Poster Number: 22

Effects of extraction method on the prebiotic potential of *Ascophylum nodosum* extracts

Abstract

Seaweed-derived bioactive compounds exhibit various beneficial activities in humans and animals. A factor influencing their concentrations, and subsequent bioactivity, is the extraction method. Our aim was to evaluate the *in vitro* prebiotic potential of three differently-extracted *Ascophylum nodosum* samples. The samples were produced using either solid-liquid extraction with water (AN-W), or ethanol (AN-EtOH) as solvent or high pressure-assisted extraction with water as solvent (AN-HPW). All extracts were two-fold diluted from 2 mg/ml to 0.25 mg/ml. *Lactobacillus plantarum* (LP), *L. reuteri* (LR) and *Bifidobacterium thermophilum* (BT) were used at 10^6-10^7 colony-forming unit(CFU)/ml. Each concentration of each extract and controls (0 mg/ml) were incubated for 18 h at 37 °C aerobically or anaerobically (BT). Final bacterial concentrations were determined by spread plating. All experiments were carried out in triplicate with technical replicates. All data were logarithmically transformed and analysed using PROC GLM (SAS 9.4). AN-HPW increased BT (≤0.9 LogCFU/ml, $P<0.05$) at all concentrations and LR and LP (0.2 LogCFU/ml, $P<0.05$) at 2 mg/ml and 1mg/ml, respectively. AN-W increased BT (≤0.6 LogCFU/ml, $P<0.05$) at 1-2mg/ml, but decreased both lactobacilli; LP ≤0.7 LogCFU/ml and LR ≤5.4 LogCFU/ml at all concentrations ($P<0.05$). AN-EtOH increased LP (≤0.7 LogCFU/ml, $P<0.05$), but reduced LR (≤5.7 LogCFU/ml, $P<0.05$) at all concentrations and BT (≤4 LogCFU/ml, $P<0.05$) at 1-2 mg/ml. In conclusion, the extraction method influenced the prebiotic potential of the *A. nodosum* extracts *in vitro* with AN-HPW being the most promising.

Brigkita Venardou, Mary J McDonnell, Marco Garcia-Vaquero, Gaurav Rajauria, John V O'Doherty, Torres Sweeney

University College Dublin, Dublin, Ireland

Poster Number: 23

Potential role of Proteobacteria that commonly colonise the respiratory tract in licensing pathogenicity in Th17 cells

Abstract

Self-antigen reactive Th17 cells are important pathogenic effectors in numerous autoimmune diseases, but are innocuous immediately following differentiation. IL23 signalling is a critical requirement for Th17 cell pathogenicity. However, antigen-specific Th17 cells only upregulate the IL23 receptor following differentiation in peripheral lymphoid tissues. When and where self-reactive Th17 cells are exposed to this pathogenic signal is unknown. Based on preliminary data, we hypothesize that CD4 T cells that have invoked the Th17 genetic programme are initially attracted to the airways where putative respiratory tract bacteria induce IL23 expression by innate immune cells that confers pathogenic potential on Th17 cells.

We infected different populations of innate immune cells *in vitro* with live respiratory tract bacteria from five different phyla and examined IL23 and related cytokine secretion at selected time-points. *In vivo*, we colonised the upper respiratory tract of mice with selected bacteria to determine expression of IL23 and related cytokines in the airways. Our data indicates that Proteobacteria species, including *Klebsiella pneumoniae, Moraxella catarrhalis* and *Neisseria meningiditis* are strong stimulators of IL23 and IL12 *in vitro* and *in vivo*, while *Fusobacterium nucleatum* is also able to induce IL23 expression by DC. Conversely, bacteria from the phyla Firmicutes and Actinobacteria did not promote an IL23 response. We are currently assessing the ability of respiratory tract bacteria-induced IL23 to convert innocuous Th17 cells to pathogenic effector ex-Th17 cells, with associated changes in migratory and inflammatory markers. Our findings support the concept that bacteria that colonise the respiratory tract could regulate Th17 cell pathogenicity.

Jenny Mannion, Rachel McLoughlin, Stephen Lalor

Trinity College, Dublin, Ireland

Poster Number: 24

An imaging based investigation into the role of relaxation of negative DNA supercoiling in invasion of human epithelial cells and biofilm formation in *Campylobacter jejuni.*

Abstract

Campylobacter jejuni is the leading cause of bacterial gastroenteritis and is a significant health burden in Ireland and the rest of the developed world. Although the organism is known to be a prolific pathogen, in comparison to other organisms such as *Salmonella* and *E. coli*, the pathogenesis of *C. jejuni* infection is poorly understood. Recent data published in our lab suggests a role for relaxation of negative DNA supercoiling as a global regulator of *C. jejuni* pathogenesis, which may be induced by environmental stimuli. Relaxation of negative supercoiling results in an increase in *C. jejuni* strain NCTC11168 protein secretion resulting in increased attachment and invasion of intestinal carcinoma epithelial cell lines(1). Using a combination of immunofluorescence and vital staining, along with emerging microscopy techniques, we have developed imaging based approaches which we have used to investigate the connection between this invasive phenotype and the formation of adherent biofilms in a range of environmental conditions. Currently, we have shown an imaging based approach allows for a resolution of *Campylobacter* biofilms normally not possible using conventional biochemical approaches. This approach conveys information as to the viability and ECM components of the *C. jejuni* biofilm. We have also found that relaxation of negative supercoiling results in a stark increase in adherent biofilm formation and planktonic viability in both microaerophilic and aerobic environments. This biofilm phenotype presents with a stark increase in the eDNA matrix and a bacterial population that remains viable for long time points in the presence of oxidative stress.

Matthew Whelan, Jeremy Simpson, Tadhg Ó Cróinín

University College Dublin, Dublin, Ireland

Poster Number: 25

An investigation into the link between DNA supercoiling and protein secretion in*Campylobacter jejuni*

Abstract

Campylobacter jejuni is the foremost cause of food-borne, bacterial gastroenteritis globally yet the cellular mechanisms behind invasion remain to be elucidated. DNA topology is recognised as a global regulator in bacteria with many pathogens regulating virulence through changes in supercoiling. Previous work has demonstrated that relaxation of supercoiling in *C. jejuni* leads to increased protein secretion across multiple strains, coinciding with increased invasion of human cells *in vitro*.
This unique secretome, expressed under conditions of relaxed DNA supercoiling, was investigated via mass spectrometry analysis. As strain to strain variation is significant in *C. jejuni*, a panel of six isolates from a variety of sources, all displaying phenotypic variance, was chosen for investigation.
Proteins identified to be differentially secreted under conditions of a relaxed DNA supercoiling topology include those involved in protection against reactive oxygen species, virulence and flagellar function. *In vitro* validation of a cohort of these secreted proteins was carried out by confirming that relaxation of DNA supercoiling leads to increased resistance to H2O2 stress. 81-176 is a well-characterised laboratory strain of *C. jejuni* which is known to demonstrate high levels of invasion *in vitro* and relatively low levels of motility. 81-176 was revealed to have a naturally relaxed DNA supercoiling topology, high levels of baseline protein secretion and also an inherently high resistance to H2O2 in comparison to the reference strain 11168. This provided further evidence of the link between DNA topology and response to host-relevant environmental factors in the form of altered motility and protein secretion.

Laura Ardill, Dr. Tadhg Ó Cróinín, Dr. Gerard Cagney

School of Biomolecular and Biomedical Science, University College Dublin, Dublin, Ireland

Poster Number: 26

Short chain fatty acids control SPI-I phase variation post-transcriptionally

Abstract

Salmonella spp. express *Salmonella* pathogenicity island I (SPI-I) genes to mediate the initial phase of interaction with host cells. A number of reports show only a subpopulation express these genes and that the proportion of SPI-I expressing cells is important for pathogenicity. Using SPI-I transcriptional reporters in a combination of population and single-cell assays, we show a combination short-chain fatty acids, molecules produced by the ileal microbiota, decrease the proportion of SPI-I expressing cells. This effect is mediated by HilE, a known regulator of SPI-I. Curiously, the addition of SCFAs do not affect *hilE* transcription, suggesting the response is mediated post-transcriptionally. An *in silico* analysis of HilE protein structure indicates a putative intrinsically disordered domain; these domains are dynamic, assuming a structure upon binding to its cognate molecule. Experiments are underway to assess the importance of this domain for SCFA mediated repression of SPI-I expressing subpopulation development. Collectively, these results show physiologically relevant environmental signals affect *Salmonella* development, and by implication, infectivity.

Alyson Hockenberry[1,2], Gabriella Takacs[1,2], Martin Ackermann[1,2]

[1]ETH Zurich, Zurich, Switzerland. [2]EAWAG, Duebendorf, Switzerland

Poster Number: 27

Host mucin as a substrate and signal molecule for *Vibrio cholerae*.

Abstract

Vibrio cholerae penetrates the human mucosal barrier of the small intestine in order to establish microcolony formation in the epithelial crypts. There it produces virulence factors cholera toxin and the toxin co-regulated pilus. In addition to penetrating mucus to reach the site of infection, we hypothesize that *V. cholerae* uses mucus in other ways to promote colonization; constituent components of mucin may serve as energy substrates and signaling molecules. We screened a transposon library of *V. cholerae* El Tor C6706 for mutants unable to grow on minimal media supplemented with porcine gastric mucin. Mutants lacking E1 (*aceE*) and E2 (*aceF*) of the pyruvate dehydrogenase complex were identified and further characterized. Defined mutants exhibited altered colonization dynamics in the infant mouse. The *aceE* and *aceF* mutant strains were attenuated for colonization and elicit alterations in duodenum epithelial structure not observed with wild type, despite a lower bacterial cell burden. This work highlights the interrelationship between *V. cholerae* metabolism and colonization during infection. Beyond being a potential growth substrate, mucin may impact virulence factor production as an early environmental signal during colonization. In the presence of mucin, we identified increased transcription of *toxT,* encoding a key regulator of toxin and pilus expression. We did not, however, observe similar mucin-induced transcription of toxin and pilus genes. We hypothesize that mucin serves as a priming signal for *V. cholerae* to activate *toxT*, and that an additional layer of control prevents production of adhesion pili and cholera toxin in the mucin layer.

Andrew Van Alst, Victor DiRita

Michigan State University, East Lansing, USA

Poster Number: 28

The Effect of Mucin on the Global Proteomic Profile and Virulence of Pseudomonas aeruginosa

Abstract

Cystic Fibrosis (CF) is characterised by the accumulation of excessive amounts of mucus in the airways and chronic bacterial infection. *P. aeruginosa* is the most common bacteria found infecting the CF lung. Two strains of *P. aeruginosa*, CF strain, S1961 and the well characterised wound isolate, strain PAO1, were grown in Artificial Sputum Media (ASM) designed to mimic the sputum in the CF lung, in the presence and absence of mucin to assess the effect of mucin on the virulence and global proteomic profile of the bacteria.

The presence of mucin in ASM promoted a hyper virulent planktonic state in *P. aeruginosa* strain PAO1, including hyper motility, increased production of pyocyanin and enhanced antibiotic resistance. Proteomic analysis revealed that mucin induced increases in the abundance of virulence associated proteins involved in antibiotic resistance and motility for both strains. Many of the changes in protein abundance in the CF strain were related to nutrient acquisition.

Decreases in the abundance of iron sulphur cluster (ISC) biogenesis proteins were observed when both strains were cultured in ASM both in the presence and absence of mucin. Disruption of ISC biogenesis in other bacterial genera, such as *Escherichia coli* and *Azotobacter vinelandii*, has resulted in phenotypes similar to that of strains isolated from CF patients, such as excessive mucoidy, reduced motility and increased biofilm formation. This suggests that disruption of the ISC protein network may play a role in adaptation of *P. aeruginosa* to the CF lung.

James O'Connor Moneley, Marguerite Clyne

University College Dublin, Dublin, Ireland

Poster Number: 29

PROTECTIVE ACTION OF ALUMINIUM SILICATES ON ENTEROCYTES OF RATIRRITATED WITH DEXTRAN - ULTRASTRUCTURAL PRELIMINARY STUDY

Abstract

Background: Eating soil is a natural behavior in animals. The aim of the study is to investigate the effect of natural aluminosilicates on changes in the intestinal epithelium of rats with dextran (Dx) induced colitis.

Methods: The study obtained the ECE's approval for Animal Research. The rats were fed with standard feed (SF).

Sixty rats were subjected to seven days' damage of the intestinal epithelium using Dx. Then they were divided into three groups: W (N=12) –without any treatment; M (N=12) – treated intragastrically with mesalazine; and AS (N=12) – treated with 10% aluminosilicats added to SF.

Control groups: C_0 (N=12) – rats not subjected to any actions; C_{AS} (N=12) – rats on SF with 10% aluminosilicates.

On the first day after Dx exposure, and then twice every 2 weeks, some animals from each group were killed and biological material was collected for further testing.

Results: There were no differences in the ultrastructure of enterocytes in groups C_0 and C_{AS}. In groups W, M and AS all the animals examined on the day after Dx exposure showed contrastive structural differences regarding tight junctions (TJ). The tight junctions were atrophic, with vacuoles, and with secondary ultrastructural changes of the endoplasmatic reticulum. Two weeks later, a stable picture in group AS without a regression of the lesions was observed. After 4 weeks, the regression in group AS was larger than in group M.

Conclusions: The addition of aluminosilicates to the standard diet accelerated the regression of damaged intestinal epithelium within TJ's.

Michal Petelenz[1], Krzysztof Jasik[2], Barbara Blonska-Fajfrowska[1], Slawomir Wilczynski[1]

[1]Dept of Basic Biomedical Science, School of Pharmacy with Div. of Laboratory Medicine in Sosnowiec, Medical University of Silesia,,, Katowice, Poland. [2]Dept of Skin Structural Studies, School of Pharmacy with Div. of Laboratory Medicine in Sosnowiec, Medical University of Silesia, Katowice, Poland

Poster Number: 30

The orphan response regulator VirS of the intracellular pathogen *Rhodococcus equi* interacts with multiple sensor kinases

Abstract

Rhodococcus equi is a facultative pathogen which infects the lower respiratory tract of foals and immune-supressed humans. *R. equi* infection usually occurs through aerosol inhalation and leads to pyogranulomatous pneumonia. Two component regulatory systems are critical for niche adaption and therefore for pathogen survival in the host. Environmental signals are typically mediated by a sensor kinase, which phosphorylates a partner response regulator. In turn, the response regulator regulates transcription of target genes. Genes encoding sensor kinase and response regulator are most times in the same operon. VirS is an orphan response regulator of *R. equi*. It is essential for regulation of genes encoding the virulence associated proteins (*vaps*) and for bacterial replication inside macrophages. The genome of *R. equi* encodes 24 sensor kinases, of which all but one have their cognate response regulator. Given the specificity of the interaction between sensor kinase and response regulator, yeast two-hybrid screening was employed to identify the partner senor kinase of VirS. We found that VirS interacts with at least two sensor kinases. We then employed gene deletion analysis to determine whether the identified sensor kinases are required for the regulation of vapA and the survival of *R. equi* in macrophages. Neither single nor multiple deletion mutants showed any effect on signal transduction via VirS. Therefore, it is highly likely that there is redundancy in the signal transduction process and at least one more VirS sensor kinase partner need to be identified.

Cheng Cheng, Aleksandra A. Miranda-CasoLuengo, Xiaoxiao Chen, Ciaran Finn, Raúl Miranda-CasoLuengo, Wim G. Meijer

School of Biomolecular and Biomedical Science and Conway Institute, University College Dublin, Dublin, Ireland

Poster Number: 31

Detection of Beta-lactamase Loci in Municipal Wastewater Samples

Abstract

The occurrence and spread of antibiotic-resistant genes (ARGs) are pressing public health problems worldwide. A key factor contributing to the spread of ARGs is lateral gene transfer. Wastewater treatment plants (WWTPs) are measured hot spots of microbial diversity and resistance because they receive polluted wastewater from diverse sources and contain a variety of different environments with dense bacterial loads. Due to the man's overuse of antibiotics the genetic capacities of microbes have profited. This helps every source of resistance genes and every means of horizontal gene transmission to develop the multiple mechanism of resistance to each antibiotic used clinically, agriculturally, or by any other medium**.** In municipal wastewater plants, where gastrointestinal wastes from city residents co-mingle, the probability for lateral gene transfer events is greatly increased. In this study, we use PCR technique to detect four beta-lactamase loci to assess the prevalence of ARGs. Wastewater samples from municipal plant at different stages of treatment as well as water samples from the river upstream and downstream from the release site were collected, followed by total DNA extraction and purification. These were then used as templates in PCR-based detection of beta-lactamase (*bla*) resistance loci. Our data suggested that wastewater samples contain high level of impurity that suppresses PCR reactions. Further, our results showed the presence of 3 loci in influx wastewater but not in the efflux, nor in the river water samples. Up to now we can say there is no detectable levels of ARGs in WWTP effluent samples, upstream and the downstream rivers. These data are vital in understanding the role of WWTPs in contributing to the spread of antibiotic resistance in the environment. The project will continue to collect multiple samples at different stages of water treatment across seasonal changes to offer broader insight into the presence and prevalence of ARGs in municipal wastewater samples.

Sunil Pandey

Eastern Illinois University, Charleston, IL, USA. Audrey Rex, Charleston IL, USA. Kai Hung, Charleston IL, USA

Poster Number: 32

Hydrogel Based Delivery of Halogenated Furanones for the Inhibition of Quorum Sensing and Prevention of Biofilm Formation

Abstract

Pseudomonas aeruginosa is a ubiquitous Gram-negative pathogen capable of causing a range of infections. When *P. aeruginosa* infects a wound biofilms are likely to form making treatment considerably more challenging. These persistent biofilms prolong the inflammatory stage of wound healing, impair tissue repair and significantly delay wound healing. This often causes a wound to progress and become chronic, presenting a new range of problems for patients including increased risk of secondary infection, deterioration of the wound bed and surrounding tissue and an increase in treatment intensity.

This project has four main objectives. Firstly, to formulate and load a shear sensitive poly(vinyl alcohol) (PVA) hydrogel with two separate furanone compounds; 4-hydroxy-2,5-dimethyl-3(2H) furanone (HDMF) and 2-methyltetrahydrofuran-3-one (MTHF). Secondly, to characterise the stability of furanones when loaded into the hydrogel matrix. Thirdly, to investigate release kinetics of furanones from hydrogels. Finally, to assess the efficacy of the furanones in reducing biofilm formation PAO1.

HDMF can be readily loaded into the PVA hydrogel and is also easily released from it. However, HDMF has been found to have a low stability in solution and degrades rapidly (85% degradation in 24h). MTHF was also easily loaded into the gel and was considerably more stable however, the gel loading/release process appears to cause an as yet uncharacterised change in the molecule. Investigations into this change are currently underway. Experiments to characterise the effect of the furanone compounds on bacterial growth and biofilm formation are currently being conducted.

Chris Proctor[1], Nigel Ternan[1], Paul McCarron[2]

[1]NICHE - Ulster University, Coleraine, United Kingdom. [2]School of Pharmacy - Ulster University, Coleraine, United Kingdom

Poster Number: 33

Functionality of virulence-associated proteins (vap) of *Rhodococcus equi*.

Abstract

Rhodococcus equi is an intracellular pathogen which harbours a virulence plasmid that is essential for inhibiting the maturation of the endosome in which it resides, thus preventing it from being killed by the host cell. *R. equi* isolates from different animal hosts harbour different types of the virulence plasmid. The *vapA* type virulence plasmid (pVAPA) is typically identified in equine isolates, the *vapB* type virulence plasmid (pVAPB) occurs in porcine isolates, while the *vapN* type virulence plasmid (pVAPN) was recently identified in bovine isolates. VapA is the key virulence factor encoded within pathogenicity island (PAI) in pVAPA. In this study, macrophage infection approach was employed to investigate the functionality of Vaps in three types of virulence plasmid. VapA was the only Vap in pVAPA essential for virulence in equine isolates. Only VapK (K_1/K_2) in pVAPB and VapN in pVAPN have the ability to compensate the virulence of VapA in equine isolates with the deletion of *vapA*. We found that only one gene in each type of virulence plasmid was essential for virulence.

Keywords: Rhodococcus equi; virulence; macrophage infection

Yuting Yin, Raúl Miranda-CasoLuengo, Wim Meijer

University College Dublin, Dublin, Ireland

Poster Number: 34

High sero-prevalence of Hepatitis E virus antibodies among voluntary blood donors in resource Poor Settings.

Abstract

Background: Hepatitis E virus (HEV) is the most common cause of enterically-transmitted viral hepatitis worldwide; it is also known to be transmitted through blood transfusion [1]. The disease incidence has been calculated to be around 45/1000 person per year [2] and infects around 2.2 million people per year in India [3]. Routine blood testing in blood donors in India does not screen HEV infection posing a risk of transmission. Our study aims to find the prevalence of HEV in blood donors to estimate the risk of its transmission.

Methods: This study included a total of 410 non-duplicate serum samples from all voluntary blood donors in month of april 2018, sample were tested for anti-HEV IgG and IgM antibodies by ELISA kit. Serum AST levels were also performed from serum to assess any active liver disease in these patients. Consent was taken from all blood donors and confidentiality of the results was maintained.

Results: Out of 410 blood donors 15(3.7%) were positive for IgM, 14 of these had elevated AST levels. While 69(16.8%) were positive for IgG among whom 17 donors had elevated AST levels.
Conclusion :As suspected seroprevalance of HEV antiboidies is quite high in voluntary blood donors and high percentage of these also had altered liver function. Our results are an eye opener that risk of its transmission could be quite high, even much more than hepatitis B and C. Hence developing countries like India urgently need to start screening blood products for HEV.

Sumit Rawat, Amardeep Rai

Bundelkhand Medical College, Sagar, India, India

Poster Number: 35

Growth temperature changes pneumococcal behaviour

Abstract

Background: The interaction between *S. pneumoniae* and the mucosal epithelial cells of nasopharynx is a pre-requisite for initial attachment. In most cases, pneumococcus disseminates to the lower respiratory tract and other niches. During this progression, the pneumococcus copes with environmental stress and temperature variation which is one of the factors that can modify bacterial behaviour. The aim of this work was to identify the importance of temperature on pneumococcal phenotype associated with virulence and survival.**Methods**: To understand pneumococcal strategies in adapting to different temperatures, transcriptional gene expression was determined in mid- and late-exponential growth phase at 34°C and 40°C relative to 37°C. The role of seven differentially expressed genes was further examined by analysis of isogenic mutants by growth studies, biochemical assays, biofilm formation, and pH adaptation.

Results: Microarray data revealed that a large array of pneumococcal genes of diverse classes were differentially expressed at 34°C (491 genes) and 40°C (189 genes) relative to 37°C. The pneumococcus displayed differences in growth, biofilm formation, production of glycosidases and haemolytic activity, which are important for colonisation and invasive disease at different temperatures. The mutant analysis also attributed a role for selected genes in thermoregulation.

Conclusion: Taken together, these results indicated that growth temperature is an important stimulus to programme the transcriptional profile and phenotypes of *S. pneumoniae* at different niches. Functional characterization of differential gene expression at different temperatures suggest further experiments to understand thermal adaptation at various host environments.

Ozcan Gazioglu, Peter Andrew, Hasan Yesilkaya

Infection, Immunity and Inflammation University of Leicester, leicester, United Kingdom

Poster Number: 36

TprA/PhrA quorum sensing system: Its role in pneumococcus virulence

Abstract

Opportunistic human pathogen *Streptococcus pneumoniae* can cause life-threatening disease. The common occurrence of pneumococcal infections suggests its ability to detect and adapt to the changing environmental stimuli. Several transcriptional regulatory pathways have a crucial role on pneumococcal adaptation to environmental changes. TprA/PhrA quorum sensing (QS) system is one of a new family of regulatory systems shown to be involved in virulence, galactose utilisation, and neuraminidase production. Moreover, its potential utility as an anti-infective target has been demonstrated by our research group. However, it is not known in detail how TprA/PhrA contributes to the pneumococcal virulence. Therefore, the goal of this study is to reveal the mechanism of TprA/PhrA mediated virulence in pneumococcus.

Mutations were introduced into the selected genes shown to be regulated by TprA/PhrA QS system by splicing overlap extension. Pneumococcal strains were tested by growth studies using chemically defined medium supplemented with different sugars. LacZ transcriptional reporter assays were used to determine transcriptional control of selected genes by measuring promoter induction under specific conditions.

The results showed that the strains lacking *spd1517* or *spd1947* utilize galactose and mucin less efficiently than the wild-type, and the mutant strains had lower neuraminidase activity than the wild-type. In addition, the reporter assays showed that TprA and *spd1947* are activators of tagatose pathway, but these regulators have no significant effect on expression of *galK*, a key gene of Leloir pathway.

It is likely that TprA mediates pneumococcal virulence through its impact on complex regulatory cascade controlling galactose catabolism.

Banaz Kareem, Peter Andrew, Hasan Yesilkaya

University of Leicester, Leicester, United Kingdom

Poster Number: 37

Iron, Human and *Pseudomonas aeruginosa*

Abstract

The need of iron is not only restricted by human and other organisms, even bacteria need iron to survive inside their hosts. However, iron is not freely available in human body it's sequestered by being bond up by high affinity iron binding proteins transferrin Tf in blood and lactoferrin Lf in body fluids. To overcome this iron limitation bacteria have evolved mechanisms of another kind of iron binding. By either synthesis of extracellular iron chelating molecules called sidrophores or by presence of specific receptors for Tf and Lf on their surfaces. Understanding of how bacteria can steal our body iron can improve the way in how to provide treatment to fight bacterial infection.

We investigated whether *Pseudomonas aeruginosa,* one of the major human pathogen that can cause severe illness in cystic fibrosis patients, have specific receptors for human Tf and Lf. Early findings revealed that one of *P. aeruginosa* major protein (porin), located in their outer membrane, called OprF is both Tf and Lf binding and this is uncommon in other bacteria like *Neisseriaceae* which was found to have two separate receptors one for Tf and another for Lf. Morover, there is a correlation between high OprF level and less iron sequestering sidrophores (pyoverdine) also it was found that more OprF means bacteria can grow better in iron restricted media such as serum medium. Knockout the gene which express this protein can improve our understanding of the role that it may play in iron biology and virulence of *P. aeruginosa*.

Shamim AL-Husseini, Dr Primrose Freestone

University of Leicester, Leicester, United Kingdom

Poster Number: 38

Identification of novel bacterial adhesins for use in vaccination to prevent Verocytotoxigenic Escherichia coli (VTEC) infections in children.

Abstract

VTEC are a group of strains of E. coli, which cause severe bloody diarrhoea. VTEC infections can lead to haemolytic uraemic syndrome (HUS), which is the main cause of kidney failure among children. HUS can also cause other life-long complications including seizures, bowel perforation and blindness. There are no vaccines available to protect children from VTEC infections. Due to a high proportion of patients experiencing severe symptoms and complications, there is an urgent need for a protective vaccine against this infection.

We have investigated the epidemiology of VTEC in an Irish paediatric patient population 2011-2016 and found that, O157 and O26 were the most common serotypes among Irish children, contributing to 45% and 25% of all paediatric VTEC infections. Both strains were associated with comparable levels of hospital admissions.

Bacterial proteins involved in host cell attachment have previously been shown to be effective vaccines. We have developed a method which identifies bacterial proteins involved in host cell attachment which has enabled us to identify vaccine antigens with excellent preclinical efficacy (McClean et al., 2016). Using this proteomic approach, we have identified that VTEC uses unique bacterial adhesins to attach to gastrointestinal cells in vitro (Caco-2 and HT-29 cells), compared to commensal E. coli strains. In particular, we have identified eight host cell attachment proteins unique to VTEC strains only, and not identified in attachment of commensal strains.

Protection of children and immunocompromised adults against VTEC infection will reduce the risk of life-threatening complications from this difficult pathogen.

Conor Quinn, Susanna Frost, Siobhan McClean

School of Biomolecular and Biomedical Science, University College Dublin, Belfield, Dublin 4, Ireland

Poster Number: 39

The investigation of the mechanisms of adaptation during chronic colonisation in cystic fibrosis

Abstract

Cystic fibrosis (CF) is a recessive genetic disease characterised by a production of abnormal thick mucus in the lungs and other organs. CF patients are at risk of developing chronic lung infections caused by opportunistic pathogens such as members of the Burkholderia cepacia complex (Bcc). Bcc is a group of 22 highly antibiotic resistant Gram negative bacteria. Colonisation by Bcc is rarely eradicated and can reduce a patient's lifespan by up to 10 years.

The mechanisms of adaptation by Bcc during chronic colonisation are poorly understood. Recently we identified an immunogenic protein which we hypothesise plays a role in the chronic infection process. It was found to be dramatically upregulated in chronically infecting strains of Bcc during stationary phase and low-oxygen conditions. Recently we have shown that it is indirectly involved in host cell attachment, antibiotic resistance and supressing host inflammatory responses.

Whole proteomic comparison of a mutant strain showed that 1068 proteins were significantly changed in abundance, suggesting this protein is a global regulator. Structural modelling shows that the protein has an overall negative surface charge characteristic of DNA mimics, indicating it may mediate its regulatory effects via interaction with positively charged DNA-binding proteins. Proteomic analysis identified potential candidate interacting proteins: HU-DNA binding protein, a nucleoid-associated protein and NUDIX hydrolase protein, a member of a family of oxidative stress response proteins.

We have named this protein ERP (Electronegative Regulatory Protein) and hypothesise it is involved in early stage adaptations crucial for chronic colonisation of the CF lung.

Sarah Mc Cormack, Simon Dignam, Ruth Dennehy, Siobhán McClean

niversity College Dublin, Dublin, Ireland

Poster Number: 40

Acetate, Propionate and Butyrate regulation of intestinal epithelial cell responses

Abstract

Background: Dietary fibers are fermented by commensal microbiota and generate short chain fatty acids (SCFAs) which can act as a source of metabolic energy for intestinal epithelial cells (IEC) and influence inflammatory cellular responses. Current knowledge regarding SCFA effects on IEC responses emanates primarily from IECs exposure to butyrate. This study aims to examine IEC responses to acetate, propionate and butyrate.Methods: CaCO2Bbe1 cells were treated with different individual SCFAs concentrations and cell viability, cytotoxicity and gene expression (qRT-PCR) analysed. Acetate-, propionate- or butyrate-treated cells were challenged with a pro-inflammatory cytokine cocktail and chemokine responses measured. Murine small intestinal organoids were differentiated in media supplemented with individual SCFAs for 7 days. Results: Acetate moderately affected IEC viability and proliferation while high propionate concentration was toxic for IECs with a dose dependent reduction in proliferation. In contrast, butyrate minimally effected cell viability and cytotoxicity, but induced proliferation. At the gene expression level, acetate moderately reduced genes involved in cell cycle but enhanced NFKB1, while propionate and butyrate enhanced IL18 and HDAC3, but attenuated NFKB1. Propionate and butyrate pre-treatment attenuated cytokine-induced IL-8 secretion, whereas acetate pre-treatment enhanced IL-8 and butyrate enhanced CCL20 response. Organoids differentiated with acetate demonstrated an increased area and budding while propionate and butyrate didn't. Conclusion: Our data shows that individual SCFA have differential effects on IEC. Acetate moderately modulate gene expression or toxicity in IECs while increasing budding in organoids. Propionate reduced proliferation while butyrate increased it and both present anti-inflammatory potential and differential gene regulation.

Aine Fanning, Keith O'Donoghue, Mary Ahern, Orlaith O'Connell, Fergus Shanahan, Silvia Melgar

APC Microbiome Institute, Cork, Ireland

Poster Number: 41

Characterisation of microorganisms from the lower respiratory tract of asthmatic patients and healthy individuals using extended culture

Abstract

The healthy lower respiratory tract was once thought to be a sterile environment. Molecular based investigations however have resulted in acceptance that a healthy lung microbiome exists and that this microbiome is altered in patients suffering from lung diseases.

Using a range of different media including; Columbia blood agar, chocolate agar, MacConkey's agar, and minimal medium supplemented with 0.5 % (w/v) mucin; we are assembling a culture collection of bacteria isolated from lung brushings taken from asthmatic and healthy individuals.

Plates were incubated in both aerobic and anaerobic atmospheres. Colonies were selected from source plates over a period of seven days to ensure the inclusion of slower growing organisms. To ensure purity, isolates were streaked twice prior to storage on Microbank beads.

From three asthmatic patients and two healthy controls over 600 bacterial isolates have been collected. A database of the isolates has been created including bacterial characteristics such as Gram stain, oxidase and catalase types. 16S rRNA analysis has revealed 16 OTU types, however, we anticipate variation within these OTUs. To date, eighty-five strains have been selected for whole genome sequencing with genomes assembled using the automated microbial genome analysis pipeline BugBuilder.

Further work on these isolates will include characterisation of their biochemical properties, identification of virulence factors and antimicrobial resistance genes, as well as comparisons between bacterial isolates obtained from asthmatic versus healthy lungs.

Colin P. Churchward, Leah Cuthbertson, Michael J. Cox, William O. C. Cookson, Miriam F. Moffatt

Imperial College London, London, United Kingdom

Charles Darwin House,
12 Roger Street,
London, WC1N 2JU, UK
www.microbiologysociety.org

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