Table 1. Clinical and microbiological characteristics, and treatment outcomes of patients infected by MRSA with mixed hemolytic phenotype

| Variable | 1 | 2 | 3 | 4 | 5 | 6 |
|----------|---|---|---|---|---|---|
| Age (yrs) | 66M | 65F | 68F | 65M | 64F | 63F |
| Underlying disease | leukaemia | CS | diabetes | end-stage renal disease | rheumatoid arthritis | lymphoma | Liver transplant |
| Previous use of antibiotics | Yes | Yes | No | No | No | Yes |
| Acquisitions | hospital-onset | hospital-onset | hospital-onset | community-onset | hospital-onset | hospital-onset | hospital-onset |
| Type of infection | infective endocarditis | septic arthritis | septicemia | infective endocarditis | primary bacteremia | primary bacteremia |
| Microbiological data | MDR | MDR | MDR | MDR | MDR | MDR |
| Nature of mixed infection | mixed aero | mixed aero | mixed aero | mixed aero | mixed aero | mixed aero |
| Phenotype | 1A | 1B | 1A | 1A | 1A | 1A |
| Stability of mixed infections | no - | no - | no - | no - | no - | no - |
| Vancocin MIC (mg/L) | 1 | 2 | 1 | 1 | 1 | 1 |
| Clinical status | Treatment | Vancocin | Vancocin | Vancocin | Vancocin | Vancocin |
| Provenetum | Yes | No | Yes | No | No | No |
| Vaccine therapy (GST 74) | improved | improved | improved | improved | improved | dead |

Figure 1. Mixed hemolytic pattern observed in single colony evaluation. A. The mixed hemolytic phenotypes of ST72-MRSA strain. B. The mixed hemolytic phenotypes of ST5-MRSA strain. All isolates were inoculated onto sheep blood agar plates containing RN4220 supernatants (β-hemolysin), and hemolysis phenotype was evaluated in each single colony after overnight culture. Arrows indicate hemolytic (black) and non-hemolytic (white) colony.

Figure 2. Comparison of the transcriptional expression of genes encoding hemolysins and saeR/saeS in two-component regulatory system between hemolytic (H) and non-hemolytic (NH) colonies in four ST72 isolates showing mixed hemolytic pattern. * P < 0.05 by Mann-Whitney U-test.

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2606. A Divergent Ferrichrome Receptor Associated with an Insertional Element (IS3) Identified on Novel Loci in Clinical Strain of Pseudomonas aeruginosa

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Session: 269. Pathogenesis and Host-Response Interactions
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Background: Pseudomonas aeruginosa is an aerobic Gram-negative bacterium that causes life-threatening acute and chronic infections in humans. Genotypic mutations and phenotypic variations are key features of its antimicrobial resistance and adaptation to the host environment. Pyoverdine associated genes and divergent receptors play a key role in acute Pseudomonas infections. This study seeks to address the heterogeneity of ferrichrome-iron receptor (fpvA) expression, its effect on pathogenicity and its propensity to cause acute infections clinically. Genetic and phenotypic variation of a clinical isolates of P. aeruginosa (PA097 and PA115) were identified by complete genome sequencing method.

Methods: An IRB-approved prospective study collected 38 P. aeruginosa clinical isolates and stored at Carilion Medical Center. Two genetically unrelated clinical strains were selected from tracheal aspirates: PA097 and PA115. These isolates were characterized by pyoverdine (pvd) quantification in planktonic culture filtrate at OD650 nm. Multiplex PCR was carried out using primers for fpvA receptors. Quantification of iron acquisition was done on chrome azurol S (CAS) agar. Genomes for PA115 and PA097 were sequenced by Illumina Next-generation DNA sequencing.

Results: Genome assembly shows a 6.3 Mb genome size in PA115 with G+C content of 66.4%. Seven insertion sequence elements were located. We found a 101 kb locus for pvd and a highly diversified fpvA associated with an insertional element (IS3). PA115, exhibits rich green pigment of pvd followed by PAO1 and PA097 in LB media (Figure 1A) and also in Planktomic culture filtrate (Fig 1B) for quantitative estimation of pvd (Figure 1C). On CAS agar, PA115 showed high uptake of iron by orange pigment compared with lower pigmentation in PAO1 and PA097 (Fig 1D). We confirmed the ferrichrome-iron receptor as fpvA in PA115 in Multiplex PCR seen in sequencing of PA115 (Figure 2).

Conclusion: We found high genetic and phenotypic variation in clinical isolate of P. aeruginosa (PA115) from an acute pneumonia patient. The novel IS element found in its receptor gene locus suggests an increased role in pvd expression and iron uptake from the host. Increased pvd expression and diversified fpvA/Ab association with an IS3 element may indicate higher virulence in the PA115 strain.
(AMB) or posaconazole (PCZ) was added to the medium of *R. arrhizus*-infected larvae at 16 h post-infection.

**Results:** In MTZ-treated GET larvae, inoculum-dependent mortality was found for both *R. arrhizus* (panel A) and *C. albicans* (panel B). High inter-experiment reproducibility of survival rates was seen (CV < 0.3). Using a GPP-expressing *R. arrhizus* strain, fungal invasion of the larval tissue was verified by fluorescence microscopy (panel C). PCZ and AMB improved survival rates of *R. arrhizus*-infected (5 x 10^7/ml) larvae from 46% to 85% and 51% to 86%, respectively (P < 0.001). Similarly, significantly reduced fungal burden in AMB and PCZ-treated larvae was documented by qPCR (panel D) and histopathology. In additional validation experiments, the hypo-virulent phenotypes of a CotH-depleted *R. arrhizus* strain and filamentation-defective *C. albicans* mutants (ΔeG2 and Δcph1) were recapitulated in zebrafish larvae with epithelial cell loss.

**Conclusion:** We have established a robust and reliable model of invasive mycoses by controlled ablation of epithelial cells in zebrafish larvae, allowing for rapid immunosuppression-based interrogation of different infection and treatment options. Our proof-of-concept experiments suggest that GET zebrafish larvae are positioned as an appealing high-throughput in vivo system for antifungal drug screening or comparative virulence studies.

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2606. Restriction of Rhinovirus Infection Depends on Virus Sensing and Early IFN Induction
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**Background:** Human rhinovirus (RV) infections are ubiquitous, underestimated, and costly. RV causes 3–12 infections per year per individual with a wide range of clinical presentations from mild upper respiratory infections to severe viral pneumo-nias (1). The virus-host interactions that control RV infection are poorly understood. Thus, there are no vaccines or antiviral medications available. RV infection begins at the airway epithelial surface where the virus first encounters the host cell immune defenses, including type I and III interferon (IFN). IFN actions are critical for defense against RV infection wherein interferon-stimulated genes (ISGs) direct antiviral actions to limit RV infection.

**Methods:** We hypothesized that the timing of IFN induction is a critical deter-minant of RV restriction by host innate immune defenses in the human respiratory tract. Thus, an immortalized bronchial epithelial cell line was infected with RV-14 with multiplicity of infection (MOI) ranging from 0.1 – 10 and under conditions of pre-/post infection treatment with IFN-β or IFN-λ. Host and viral RNA, protein, and RV infectious particle levels were analyzed.

**Results:** We found that RV infection induces IFN-β and IFN-λ production and subsequent ISG induction, including expression of IFIT-1, OAS1, and MX1. RV-14 infection induced IFN-β and IFN-λ in a dose-dependent manner, with a maximum fold increase of IFN expression at 48hours post infection. ISGs were induced in a similar pattern to IFNs. Viral titers increased significantly over the first 24hours post infection and then plateaued through 96hours. IFN-β and IFN-λ pre- and posttreatment condi-tions significantly decreased maximum viral titers achieved but with continued viral plateaus 24–96 hours post infection.

**Conclusion:** Our observations demonstrate that RV induces innate immune activa-tion and the production of type I and III IFN during acute infection of airway cells. Sustained viral titer plateaus, despite antiviral ISG induction, suggests viral blocking of IFN pathway mechanisms that can be overcome by early IFN induction to significantly restrict RV viral replication.

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2609. *Escherichia coli* Clonal Lineages and Virulence Factors Predict Fecal Colonization within Households
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**Background:** Extraintestinal *Escherichia coli* infections are an ever-growing threat, to which specific clonal lineages and virulence factors contribute disproportionately. Despite the gut being the main reservoir for such *E. coli* strains, relationships between clonal lineages, virulence factors, and fecal colonization patterns are poorly understood. Accordingly, we defined *E. coli* fecal colonization patterns within households (HHs) and assessed specific lineages and virulence genes (VGs) as predictors of colonization behaviors.

**Methods:** Veterans with an *E. coli* clinical isolate (*n* = 22; 11 fluoroquinolone [FQ]-resistant, 11 FQ-susceptible) and their HH members provided stool samples on 2–6 occasions each. Stools were screened for total and FQ-resistant *E. coli*. Distinct *E. coli* strains were resolved by genomic profiling of 10 colonies/sample. Strains underwent molecular lineage identification, VG detection, and comparison with the veteran's clinical isolate. Clonal lineages and VGs were assessed (Wilcoxon rank-sum test) as predictors of strains' (i) predominance within the fecal sample, (ii) persistence across serial fecal samples, (iii) within-HH strain sharing, and (iv) overall within-HH colonization prevalence.

**Results:** From the 22 veterans and 46 HH members (27 humans, 19 pets) we recovered 139 unique HH-by-household fecal *E. coli* strains. Sixty-four traits were evaluated (36 clonal lineages, 48 VGs). Of these, 44 exhibited n ≥ 5, so could be analyzed statistically. Among these 44 traits, the proportion significantly associated with ≥ 1 outcome variable was 5/6 (83%) for clonal lineages and 18/38 (47%) for VGs. Additionally, fecal strains that matched the veteran's clinical isolate exhibited significantly greater sharing, persistence, and overall colonization.

**Conclusion:** The studied *E. coli* traits – known for their associations with clinical infec-tions – were significantly associated with within-HH colonization behavior. These find-ings support that “virulence factors” may be regarded also (or perhaps best) as “colonization factors,” and “virulent lineages” as “colonizing lineages.” This suggests the possibility that future interventions that disrupt colonization behavior also could prevent *E. coli* infections.

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2610. A Deadly Intrusion: Competitive Strain Displacement among Dengue Virus Strains in Sri Lanka
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**Background:** Mosquito-borne dengue virus (DENV), the agent of dengue hemorrhagic fever (DHF), is genetically diverse, and new strains regularly invade distant locations and displace existing strains. Invasive strains often cause higher rates of DHF