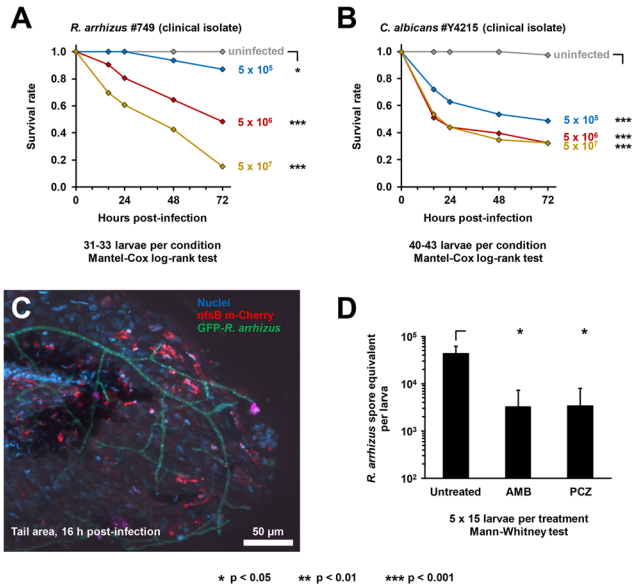
(AMB) or posaconazole (PCZ) was added to the medium of *R. arrhizus*-infected larvae at 16 h post-infection.

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 GFP-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 (5×10^6/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 (Δefg1 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 immersion-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.



Disclosures. **All authors:** No reported disclosures.

2608. 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 pneumonias (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 (ISG)s direct antiviral actions to limit RV infection.

Methods: We hypothesized that the timing of IFN induction is a critical determinant 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 conditions 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 activation 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 fluoroquinoline [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-by-household fecal *E. coli* strains. Sixty-four traits were evaluated (16 clonal lineages, 48 VGs). Of these, 44 exhibited $n \geq 5$, 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 infections –here were significantly associated with within-HH colonization behavior. These findings 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.

Table 1: Correspondence of *E. coli* molecular characteristics with colonization behaviors.

Characteristic	Strains, no. (% of 139)	*P* value: Predominance	Persistence	Sharing	Colonization
Matches clinical isolate	16 (12)	–	< 0.001	0.005	< 0.001
Clonal lineage					
ST131	14 (10)	–	0.002	–	0.04
ST131-*H30*	13 (9)	–	0.001	–	0.02
ST95	16 (12)	–	–	0.02	–
ST73	8 (6)	–	–	–	0.01
ST648	5 (4)	0.02	–	–	–
Virulence gene					
Adhesin					
papAH	37 (27)	0.001	–	–	0.05
papC	37 (27)	0.001	–	–	0.05
papEF	36 (26)	0.002	–	–	–
papG	39 (28)	0.04	–	–	–
papGII	25 (18)	0.01	–	–	–
sfaS	20 (21)	0.04	–	–	–
iha	23 (17)	–	0.02	–	0.04
Toxin					
hlyD	22 (16)	0.02	–	–	–
hlyF	9 (6)	0.01	–	–	–
sat	27 (19)	–	0.008	–	–
vat	47 (34)	–	–	0.04	–
Siderophore					
fyuA	78 (56)	–	0.04	–	–
ireA	21 (15)	0.02	–	–	–
Protectin, invasin					
kpsMII	70 (50)	–	0.004	0.04	0.004
iss	6 (4)	0.03	–	–	–
Miscellaneous					
usp	59 (42)	–	0.02	–	0.02
ompT	87 (63)	–	0.02	–	–
H7 *fliC*	18 (13)	0.01	–	–	–

[a]The symbol "–" indicates P > 0.05 (lack of statistical significance) for the particular comparison.

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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