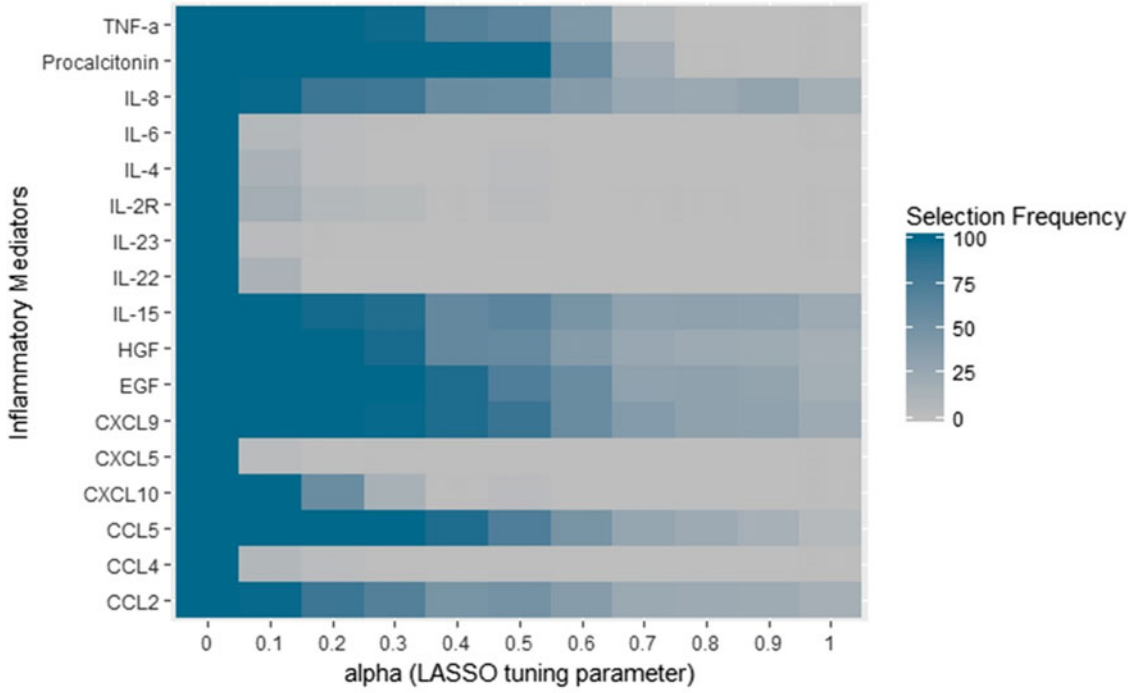

Figure 1. Heat map of frequency of inflammatory mediator selection at varying alpha levels in Ridge-/LASSO-/elastic net-regularized regression modeling of rCDI across 100 iterations of the cross-validation selection procedure. Setting alpha=0.1 yielded the optimal AuROCs (range 0.74–0.8).

Fig. 1.

the serum panel was associated with rCDI ($P = .007$) but the stool panel was not. Serum procalcitonin, IL-8, IL-6, CCL5, and EGF were associated with recurrence. The machine-learning models using the serum panel predicted rCDI with AuROCs between 0.74 and 0.8 (Fig. 1). No stool inflammatory mediators independently predicted rCDI. However, stool IL-8 interacted with toxin activity to predict rCDI (Fig. 2). These results did not change significantly upon sensitivity analysis. **Conclusions:** A panel of serum inflammatory mediators predicted rCDI with up to 80% accuracy, but the stool panel alone was less successful. Incorporating toxin activity levels alongside inflammatory mediator measurements is a novel, promising approach to studying stool-derived biomarkers of rCDI. This approach revealed that stool IL-8 is a potential biomarker for rCDI. These results need to be confirmed both with a larger dataset and after adjustment for clinical covariates.

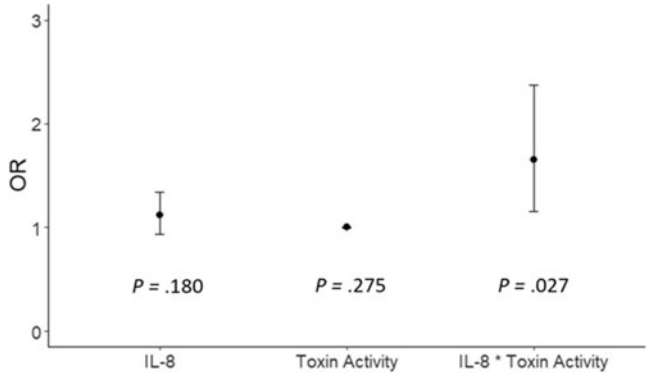

Figure 2: Neither IL-8 nor toxin activity alone predict rCDI, but modeling the interaction between both is significant. Abbreviations: OR, odds ratio for rCDI for every 1-log increase in predictor.

Fig. 2.

Funding: None
Disclosure: Vincent Young is a consultant for Bio-K+ International, Pantheryx, and Vedanta Biosciences.
Doi:10.1017/ice.2020.568

Presentation Type:
Top Rated Posters

Risk of Hospital-Onset *C. difficile* Infection Increases With Prior Inpatient and Outpatient Visits

Aaron Miller, University of Iowa; Alberto Segre, Department of Computer Science; Daniel Sewell, University of Iowa; Sriram Pemmaraju, University of Iowa; Philip Polgreen, University of Iowa

Background: *Clostridioides difficile* is a leading cause of healthcare-associated infections, and greater healthcare exposure is a primary risk factor for *Clostridioides difficile infection* (CDI). Longer hospital stays and greater CDI pressure, both at the hospital level and the level, have been linked to greater risk. In addition, symptoms associated with healthcare-associated CDI often do not present until a patient has been discharged. Our study objective was to estimate the extent to which exposure to different types of healthcare settings (eg, prior hospitalization, emergency department [ED], outpatient or long-term care) increase risk for hospital-onset CDI. **Methods:** We conducted a case-control study using the Truven Marketscan Commerical Claims and Medicare Supplemental databases from 2001 to 2017. Case patients were selected as all inpatient visits with a secondary diagnosis of CDI and no previous CDI diagnosis in the prior 90 days. Controls were selected from all inpatient admissions without any CDI diagnosis during the current admission or prior 90

https://doi.org/10.1017/ice.2020.569 Published online by Cambridge University Press

Table 1 – Selected odds ratios (95% CI) from regression analysis

Prior Exposure Window	Prior Hospitalization	Prior ED Visit	Prior Outpatient Clinic	Prior Nursing Home / LTC	Low-Risk Antibiotics (Outpatient)	High-Risk Antibiotics (Outpatient)	Prior Family Exposure
0 Days[a]	3.83 (3.74-3.93)	1.23 (1.21-1.25)	1.03 (1.02-1.05)	1.19 (1.14-1.25)			
1-30 Days[b]	2.62 (2.58-2.67)	1.20 (1.18-1.22)	1.20 (1.17-1.23)	2.79 (2.73-2.85)	1.20 (1.18-1.23)	1.70 (1.67-1.73)	4.68 (3.73-5.88)
31-60 Days	2.18 (2.13-2.24)	1.12 (1.09-1.15)	1.23 (1.19-1.27)	1.98 (1.89-2.08)	1.09 (1.06-1.12)	1.60 (1.56-1.64)	2.75 (1.91-3.97)
61-90 Days	1.71 (1.66-1.76)	1.11 (1.08-1.15)	1.21 (1.16-1.27)	1.72 (1.62-1.82)	1.01 (0.98-1.04)	1.38 (1.34-1.42)	2.56 (1.74-3.76)

Notes: [a] 0 Days denote visits on the same day as the hospitalization (e.g., transfers); [b] for antibiotics and prior family exposure a 0-30 day window was used; [c] low-risk includes penicillins, macrolides, sulfonamides or trimethoprim; [d] high-risk include clindamycin, fluoroquinolones, cephalosporins

Fig. 1.

days. A logistic regression model was used to estimate risk associated with prior healthcare exposure. Indicators were created for prior exposure to different healthcare settings: separate indicators were used to indicate transfer, exposure to that setting in the prior 1–30 days, 31–60 days and 61–90 days. Separate indicators were created for prior hospitalization, ED, outpatient clinic, nursing home or long-term care facilities (LTCFs), psychiatric or substance-abuse facility or other outpatient facility. We also included an indicator for prior exposure to a family member with CDI and prior outpatient antibiotics. **Results:** Estimates for selected variables (odds ratios) are presented in Table 1. Prior hospitalization, ED visits, outpatient clinics, nursing home and LTCFs were all associated with increased risk of secondary diagnosed CDI. Prior hospitalization and nursing home/LTCF conveyed the greatest risk. In addition, a 'dose-–response' relationship occurred for each of these exposure settings, with exposure nearest the admission date having the largest risk. Prior exposure to psychiatric , substance abuse, or other outpatient facilities were not risk factors for CDI. Having a family member with prior CDI and both low-risk and high-risk outpatient antibiotics were associated with increased risk. These factors also exhibited a 'dose–response' pattern. **Conclusions:** Exposure to various healthcare settings significantly increased risk for secondary CDI. Prior healthcare exposures occurring nearest to the point of admission conveyed the greatest risk. These results suggest that many hospital-associated CDI cases attributed to a current hospital stay may actually be acquired from prior healthcare settings.

Funding: CDC Modeling Infectious Diseases (MInD) in Healthcare Network

Disclosures: None

Doi:10.1017/ice.2020.569

Presentation Type:

Top Rated Posters

Six Years of Admission Screening for Carbapenemase-Producing Organisms at the NIH Clinical Center

Robin T. Odom, Hospital Epidemiology Service, NIH Clinical Center, NIH; Michele E. Woolbert, Hospital Epidemiology Service, NIH Clinical Center, NIH; Anna F. Lau, Sterility Testing Service, Department of Laboratory Medicine, National Institutes of Health Clinical Center; John P. Dekker, Laboratory of Clinical Immunology and Microbiology, National Institute of Allergy and Infectious Diseases, NIH; Angela V. Michelin, Hospital Epidemiology Service, NIH Clinical Center, NIH; MaryAnn Bordner, Hospital Epidemiology Service, NIH Clinical Center, NIH; David Henderson, NIH Clinical Center, NIH; Karen M. Frank, Department of Laboratory Medicine, National Institutes of Health Clinical Center; Tara Palmore, NIH

Background: Transmission of carbapenemase-producing organisms (CPO) threatens patient safety in healthcare facilities. As a result of a 2011 outbreak of *bla*KPC+ *Klebsiella pneumoniae*, the NIH Clinical Center (NIHCC) has prioritized early detection and isolation of CPO carriers, using point-prevalence surveys and targeted high-risk ward surveillance since 2011 and admission surveillance since 2013. We describe our experience over 6 years of admission surveillance. **Methods:** The NIHCC is a 200-bed research hospital that provides care for a highly immunocompromised patient population. From September 2013 to September 2019, perirectal swabs were ordered automatically for all patients on admission to nonbehavioral health wards. Swabs were ordered twice weekly for ICU patients, weekly in other high-risk wards, and monthly for hospital-wide point prevalence (excluding behavioral health). Patients hospitalized in the United States in the previous week or abroad in the previous 6 months were considered high risk for carriage and isolated pending results from 2 swabs. Most swabs (n = 37,526) were cultured onto HardyCHROM CRE. If gram-negative bacilli (GNB) were present, a molecular screen for carbapenemases was performed on a sweep of cultured material (day 1) pending organism isolation. GNB were identified by MALDI-TOF MS. Prior to June 2019, isolates were screened by *bla*KPC/*bla*NDM PCR. Starting in June 2019, Enterobacteriaceae and *Pseudomonas aeruginosa* were screened using the phenotypic modified carbapenem inactivation method (mCIM), reflexing to the GeneXpert CARBA-R molecular assay if positive; other GNB were tested directly with CARBA-R. Selected GNB underwent susceptibility testing (Sensititre). Whole-genome sequencing was used to assess relatedness among CPO isolates. Swabs from high-risk patients were tested directly by *bla*KPC PCR (n = 699) until August 2019 (most in parallel with culture) and thereafter by CARBA-R (n = 13). **Results:** Among 54,188 orders for perirectal swabs, 38,238 were collected from 14,497 patients (compliance 71%). Among 33 CPO-colonized patients identified from September 2013 through September 2019, 15 were identified on admission, 6 were identified in point-prevalence surveys, 8 were identified from high-risk ward surveillance, and 4 were identified from clinical cultures. Sequencing demonstrated no relatedness among CPO isolates. Although only 1.4% of patients sampled on admission were colonized with CPO, those meeting high-risk criteria were 21 times as likely to be colonized. **Conclusion:** Admission surveillance for CPO identified a low rate of colonization, but it detected