

Figure 1. *Candida auris* colonized patient transfer network- Orange County, CA healthcare facilities

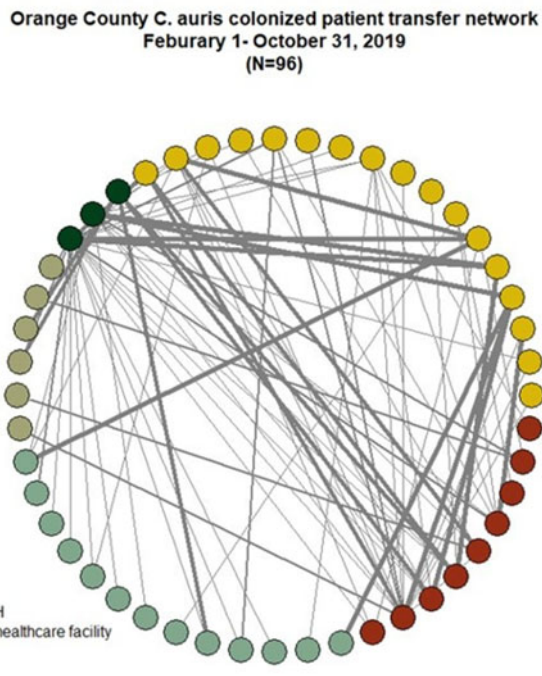


Fig. 1.

surveillance testing of exposed patients and implemented appropriate environmental and contact precautions. **Result:** From February–October 2019, 192 colonized patients were identified. All 3 LTACHs and 6 of 14 VSNFs had at least 1 *C. auris*–colonized patient identified on initial PPS, and 2 facilities had ongoing transmission identified on serial PPS. OCHCA followed 96 colonized patients transferred a total of 230 times (an average of 2.4 transfers per patient) (Fig. 1) and 677 exposed patients discharged from facilities with ongoing transmission (Fig. 2). Admission screening of 252 exposed patients on transfer identified 13 (5.2%) *C. auris*–colonized patients. As of November 1, 2019, these 13 patients were admitted 21 times to a total of 6 acute-care hospitals, 2 LTACHs, and 3 vSNFs. Transferring facilities did not consistently communicate the colonized patient’s status and the requirements for isolation and testing of exposed patients. **Conclusion:** OCHCA oversight of interfacility transfer, though labor-intensive, improved identification of patients colonized with *C. auris* and implementation of appropriate environmental and contact precautions, reducing the risk of transmission in receiving healthcare facilities.

Funding: None

Disclosures: None

Doi:10.1017/ice.2020.567

Presentation Type:

Top Rated Posters

Recurrent *Clostridioides difficile* infection can be predicted using inflammatory mediator and toxin activity levels Jonathan Motyka, University of Michigan Medical School; Aline Penkevich, University of Michigan Medical School; Vincent Young, University of Michigan Medical School; Krishna Rao, University of Michigan

Figure 2. Flow chart of exposed patient tracking for patients discharged from facilities with ongoing transmission

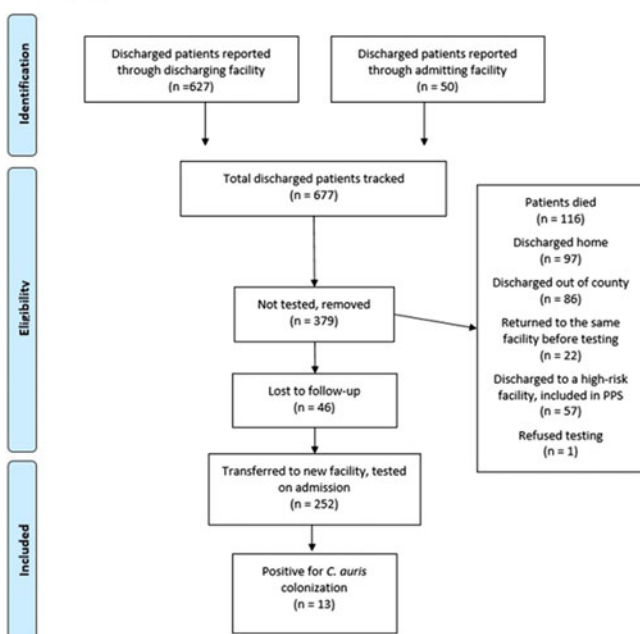


Fig. 2.

Background: *Clostridioides difficile* infection (CDI) frequently recurs after initial treatment. Predicting recurrent CDI (rCDI) early in the disease course can assist clinicians in their decision making and improve outcomes. However, predictions based on clinical criteria alone are not accurate and/or do not validate other results. Here, we tested the hypothesis that circulating and stool-derived inflammatory mediators predict rCDI. **Methods:** Consecutive subjects with available specimens at diagnosis were included if they tested positive for toxigenic *C. difficile* (+enzyme immunoassay [EIA] for glutamate dehydrogenase and toxins A/B, with reflex to PCR for the *tcdB* gene for discordants). Stool was thawed on ice, diluted 1:1 in PBS with protease inhibitor, centrifuged, and used immediately. A 17-plex panel of inflammatory mediators was run on a Luminex 200 machine using a custom antibody-linked bead array. Prior to analysis, all measurements were normalized and log-transformed. Stool toxin activity levels were quantified using a custom cell-culture assay. Recurrence was defined as a second episode of CDI within 100 days. Ordination characterized variation in the panel between outcomes, tested with a permutational, multivariate ANOVA. Machine learning via elastic net regression with 100 iterations of 5-fold cross validation selected the optimal model and the area under the receiver operator characteristic curve (AuROC) was computed. Sensitivity analyses excluding those that died and/or lived >100 km away were performed. **Results:** We included 186 subjects, with 95 women (51.1%) and average age of 55.9 years (± 20). More patients were diagnosed by PCR than toxin EIA (170 vs 55, respectively). Death, rCDI, and no rCDI occurred in 32 (17.2%), 36 (19.4%), and 118 (63.4%) subjects, respectively. Ordination revealed that

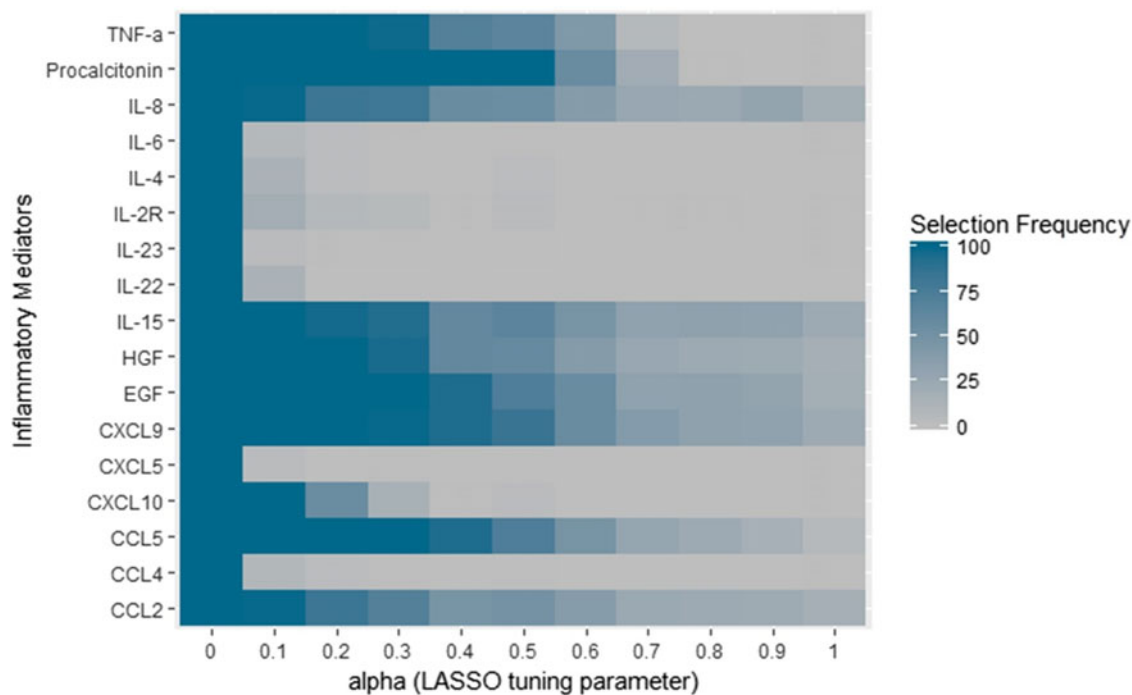


Figure 1. Heat map of frequency of inflammatory mediator selection at varying alpha levels in Ridge-/LASSO-/elastic net-regularized regression modeling of rCDI across 100 iterations of the cross-validation selection procedure. Setting alpha=0.1 yielded the optimal AuROCs (range 0.74–0.8).

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.

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

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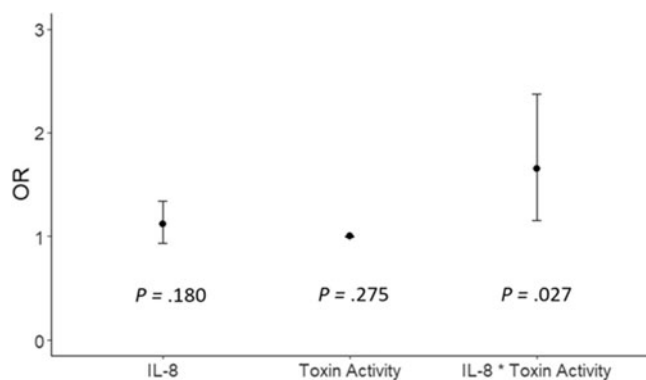


Figure 2: Neither IL-8 nor toxin activity alone predict rCDI, but modeling the interaction between both is significant. Abbreviations: OR, odds ratio for rCDI for every 1-log increase in predictor.

Fig. 2.

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 Commercial 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