

## Oral Abstract Session:

### 181. Challenges in *C. difficile* Infection Surveillance

Saturday: 10:30 a.m. - 12:00 p.m.  
Room: SDCC 29 ABCD

#### Moderators:

ERIK DUBBERKE, MD, MSPH; Washington University School of Medicine  
KAREN CARROLL, MD, FIDSA; John Hopkins University School of Medicine

#### Presenters:

- 1312** 10:30 a.m. **Risk Adjustment for Healthcare Facility-Onset *C. difficile* Infection and MRSA Bacteremia Reporting in NHSN**  
MAGGIE DUDECK, MPH, CPH<sup>1</sup>, PAUL MALPIEDI, MPH<sup>2</sup>, JONATHAN EDWARDS, MSTAT<sup>1</sup>, SCOTT FRIDKIN, MD<sup>1</sup>, L. CLIFFORD MCDONALD, MD<sup>1</sup> and **DAWN SIEVERT, PHD<sup>1</sup>**;  
<sup>1</sup>Centers for Disease Control and Prevention, Division of Healthcare Quality Promotion (DHQP), Atlanta, GA, <sup>2</sup>Centers for Disease Control and Prevention, Division of Healthcare Quality Promotion, Atlanta, GA
- 1313** 10:45 a.m. **Defining “Nosocomial” - Differences in MDRO and *C. difficile* Rates Using 2-Day vs. 3-Day Definitions**  
**ADRIJANA GOMBOSEV, BS<sup>1</sup>**, SALAH FOUAD, MS<sup>2</sup>, ERIC CUI, BS<sup>3</sup>, LEAH TERPSTRA, BA<sup>1</sup>, DIANE KIM, BS<sup>3</sup>, HILDY MEYERS, MD MPH<sup>4</sup>, MICHELE CHEUNG, MD MPH<sup>4</sup> and SUSAN S. HUANG, MD, MPH, FIDSA<sup>3</sup>; <sup>1</sup>University of California Irvine, School of Medicine, Irvine, CA, <sup>2</sup>Saddleback Memorial Medical Center, Laguna Hills, CA, <sup>3</sup>University of California Irvine, Irvine, CA, <sup>4</sup>Orange County Health Care Agency (OCHCA), Santa Ana, CA
- 1314** 11:00 a.m. **Evaluation of Differences in Population-Based Incidence of *Clostridium difficile* Infection across Diverse U.S. Geographic Locations, 2010**  
**FERNANDA LESSA, MD<sup>1</sup>**, YI MU, PHD<sup>2</sup>, JESSICA COHEN, MPH<sup>1</sup>, GHINWA DUMYATI, MD, FSHEA<sup>3</sup>, MONICA M. FARLEY, MD<sup>4</sup>, LISA WINSTON, MD<sup>5</sup>, KELLY KAST, MPH<sup>6</sup>, STACY HOLZBAUER, DVM<sup>7</sup>, JAMES MEEK, MPH<sup>8</sup>, ZINTARS G. BELDAVS, MS<sup>9</sup>, L. CLIFFORD MCDONALD, MD<sup>1</sup>, SCOTT FRIDKIN, MD<sup>1</sup> and EIP CDI SURVEILLANCE INVESTIGATORS;  
<sup>1</sup>Division of Healthcare Quality Promotion, Centers for Disease Control and Prevention, Atlanta, GA, <sup>2</sup>Centers for Disease Control and Prevention, Division of Healthcare Quality Promotion, Atlanta, GA, <sup>3</sup>University of Rochester, Rochester, NY, <sup>4</sup>Emory University School of Medicine, Atlanta, GA, <sup>5</sup>University of California, San Francisco/San Francisco General Hospital, San Francisco, CA, <sup>6</sup>Colorado Department of Public Health and Environment, Denver, CO, <sup>7</sup>CDC CEFO assigned to the MN Dept. of Hlth, St. Paul, MN, <sup>8</sup>CT EIP, New Haven, CT, <sup>9</sup>Oregon Health Authority, Portland, OR
- 1315** 11:15 a.m. **Effect of Nucleic Acid Amplification Testing on Population-based Incident Rates of *Clostridium difficile* Infection (CDI)**  
**CAROLYN GOULD, MD<sup>1</sup>**, JONATHAN EDWARDS, MSTAT<sup>1</sup>, JESSICA COHEN, MPH<sup>1</sup>, L. CLIFFORD MCDONALD, MD<sup>1</sup>, MONICA M. FARLEY, MD<sup>2</sup>, HELEN JOHNSTON, MPH<sup>3</sup>, LUCY WILSON, MD<sup>4</sup>, SAMIR HANNA, MD<sup>5</sup>, LISA WINSTON, MD<sup>6</sup>, STACY HOLZBAUER, DVM<sup>7</sup>, CAROL LYONS, MPH<sup>8</sup>, ERIN PHIPPS, DVM<sup>9</sup>, GARY HOLLICK, PHD<sup>10</sup>, ZINTARS G. BELDAVS, MS<sup>11</sup>, DALE GERDING, MD, FIDSA<sup>12</sup>, FERNANDA LESSA, MD<sup>1</sup> and CDC'S CLOSTRIDIUM DIFFICILE INFECTION SURVEILLANCE INVESTIGATORS; <sup>1</sup>Centers for Disease Control and Prevention, Atlanta, GA, <sup>2</sup>Atlanta Veterans Medical Center, Atlanta, GA, <sup>3</sup>Colorado Department of Public Health and Environment, Denver, CO, <sup>4</sup>Maryland Department of Health and Mental Hygiene, Baltimore, MD, <sup>5</sup>Tennessee Department of Health, Nashville, TN, <sup>6</sup>University of California, San Francisco/San Francisco General Hospital, San Francisco,

CA, <sup>7</sup>CDC CEFO assigned to the Minnesota Department of Health, St. Paul, MN, <sup>8</sup>Connecticut Emerging Infections Program, New Haven, CT, <sup>9</sup>New Mexico Emerging Infections Program, Albuquerque, NM, <sup>10</sup>University of Rochester Medical Center, Rochester, NY, <sup>11</sup>Oregon Health Authority, Portland, OR, <sup>12</sup>Edward Hines, Jr. Veterans Affairs Hospital, Hines, IL

**1316** 11:30 a.m. **The Biology and Epidemiology of *Clostridium difficile* in Oxfordshire Hospitals 2007-2010**

**MADELEINE CULE, PHD<sup>1</sup>**, RORY BOWDEN, PHD<sup>2</sup>, DAVID EYRE, BM, BCH<sup>3</sup>, A. SARAH WALKER, PHD<sup>1</sup>, DAVID GRIFFITHS, BSC<sup>4</sup>, JOHN FINNEY<sup>1</sup>, DAVID WYLLIE, PHD<sup>1</sup>, DERRICK CROOK, MB, BCH<sup>2</sup>, TIM PETO, MB BS, DPHIL<sup>2</sup>, PETER DONNELLY<sup>2</sup> and INFECTIONS IN OXFORDSHIRE RESEARCH DATABASE; <sup>1</sup>NIHR Oxford Biomedical Research Centre, Oxford, United Kingdom, <sup>2</sup>University of Oxford, Oxford, United Kingdom, <sup>3</sup>Nihr Oxford Biomedical Research Centre, Oxford, United Kingdom, <sup>4</sup>National Institute for Health Research Oxford Biomedical Research Centre, Oxford, United Kingdom

**1317** 11:45 a.m. **Prevalence and Risk Factors for Asymptomatic *Clostridium difficile* Carriage**  
**ERIK DUBBERKE, MD, MSPH<sup>1</sup>**, FAISAL ALASMARI, MD<sup>2</sup>, SONDRASEILER, BA<sup>1</sup>, TIFFANY HINK, BS<sup>3</sup> and CAREY-ANN BURNHAM, PHD<sup>4</sup>; <sup>1</sup>Washington University School of Medicine, St. Louis, MO, <sup>2</sup>Washington University School of Medicine, St. Louis, MO, <sup>3</sup>Washington University School of Medicine, St. Louis, MO, <sup>4</sup>Washington University School of Medicine, St. Louis, MO

**Session #181 Presentations:**

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**1312. Risk Adjustment for Healthcare Facility-Onset *C. difficile* Infection and MRSA Bacteremia Reporting in NHSN**

Part of Session: 181. Challenges in *C. difficile* Infection Surveillance

10:30 a.m.

MAGGIE DUDECK, MPH, CPH<sup>1</sup>, PAUL MALPIEDI, MPH<sup>2</sup>, JONATHAN EDWARDS, MSTAT<sup>1</sup>, SCOTT FRIDKIN, MD<sup>1</sup>, L. CLIFFORD MCDONALD, MD<sup>1</sup> and **DAWN SIEVERT, PHD<sup>1</sup>**; <sup>1</sup>Centers for Disease Control and Prevention, Division of Healthcare Quality Promotion (DHQP), Atlanta, GA, <sup>2</sup>Centers for Disease Control and Prevention, Division of Healthcare Quality Promotion, Atlanta, GA

**Background:** The Multidrug-Resistant Organism and *Clostridium difficile* Infection (MDRO/CDI) Module was implemented in the National Healthcare Safety Network (NHSN) in March 2009 to allow reporting of CDI, methicillin-resistant *Staphylococcus aureus* (MRSA), and other MDROs. State mandated reporting drove initial participation, but the Centers for Medicare and Medicaid Services will incentivize reporting of these two infections from acute care hospitals beginning in 2013. The use of these data for inter-facility comparisons and public reporting highlight the immediate need for adequate risk adjustment methods.

**Methods:** During 2010-2011, participating facilities reported all unique positive specimens (collected >14 days after a previous positive specimen) for CDI and MRSA bacteremia (blood specimens) to NHSN. Events were categorized as community-onset (CO, collected ≤3 days after admission) or healthcare facility-onset (HO, collected >3 days after admission). HO CDI and HO MRSA bacteremia incidence rates (per 10,000 and 1,000 patient-days, respectively) were calculated and compared by facility characteristics to identify potential risk adjustment variables using negative binomial testing.

**Results:** In 2010, 715 facilities from 28 states monitored CDI events in NHSN. A total of 20,803 HO CDI events were reported from 5,757,846 admissions and 28,279,284 patient-days. CDI incidence rates differed significantly by facility teaching type, bedsize, test type, and CO prevalence (Table). MRSA bacteremia was monitored in 548 facilities from 29 states. A total of 1,078 HO MRSA bacteremia events were reported from 3,807,920 admissions and 17,427,005 patient-days. MRSA bacteremia incidence rates differed significantly by teaching type and bedsize.

**Conclusion:** These facility characteristics will be assessed using multivariable analysis to determine risk adjustment for the HO CDI and HO MRSA bacteremia Standardized Infection Ratios (SIRs).

**CDI**

**MRSA**

## Bacteremia

	Rate	p-value	Rate	p-value
Teaching Type				
Major	8.6	---	0.1	---
All Other	6.7	<0.0001	0.05	<0.0001
Facility Bedsize				
≤200	6.7	---	0.05	---
201-500	7.0	0.0005	0.05	---
501+	8.9	<0.0001	0.1	<0.0001
Test Type				
PCR	8.3	---	---	---
All Other	6.6	<0.0001	---	---
CO Prevalence	Continuous	<0.0001	---	---

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### 1313. Defining “Nosocomial” - Differences in MDRO and *C. difficile* Rates Using 2-Day vs. 3-Day Definitions

Part of Session: 181. Challenges in *C. difficile* Infection Surveillance

10:45 a.m.

**ADRIJANA GOMBOSEV, BS<sup>1</sup>**, SALAH FOUAD, MS<sup>2</sup>, ERIC CUI, BS<sup>3</sup>, LEAH TERPSTRA, BA<sup>1</sup>, DIANE KIM, BS<sup>3</sup>, HILDY MEYERS, MD MPH<sup>4</sup>, MICHELE CHEUNG, MD MPH<sup>4</sup> and SUSAN S. HUANG, MD, MPH, FIDSA<sup>3</sup>; <sup>1</sup>University of California Irvine, School of Medicine, Irvine, CA, <sup>2</sup>Saddleback Memorial Medical Center, Laguna Hills, CA, <sup>3</sup>University of California Irvine, Irvine, CA, <sup>4</sup>Orange County Health Care Agency (OCHCA), Santa Ana, CA

**Background:** The CDC's 48-hour nosocomial definition is commonly implemented as a two or three-calendar day rule by hospitals. Our prior work in Orange County (OC), CA hospitals found that one-third of hospitals each used 48-hour, >2 day, >3 day case definitions. We now assess the impact of definition choice on acquisition and infection rates.

**Methods:** We conducted a prospective survey of Infection Prevention Programs in OC hospitals to assess the impact of using a >2 day vs. >3 day definition for nosocomial rates of MRSA, VRE, ESBL (*Klebsiella* and *E. coli*), and MDR *Acinetobacter* acquisition, as well as MRSA bacteremia and *C. difficile* infection. Respondents provided monthly data using both definitions from January-December 2010. Total patient day denominators were retrieved from a mandatory state discharge dataset. Differences in mean rates between the two nosocomial definitions were assessed using two-tailed t-tests.

**Results:** Nineteen of 31 countywide hospitals participated, with a total of 1,062,242 patient days in 2010. Across pathogens, we found that use of the >3-day nosocomial definition resulted in acquisition rates that were, on average, 17% (range 9-24%) lower than use of a >2-day definition.

**Table. Percent Lost to Capture by using >3d vs. >2d Day Nosocomial Definitions**

Pathogen	Mean Hospital Nosocomial Rate Using >2d Definition (Events/10,000 total patient days)	Mean Hospital Nosocomial Rate Using >3d Definition (Events/10,000 total patient days)	Paired t-test p-value	% Countywide Events Lost by Using >3d vs. >2d Definition
<b>Acquisition</b>				
MRSA	5.74	4.36	0.002	24%
VRE <sup>§</sup>	3.20	2.93	0.01	9%
ESBL*	2.65	2.18	0.001	17%
MDR <i>Acinetobacter</i>	1.12	0.99	0.03	12%
<b>Infection</b>				
MRSA Bacteremia	0.39	0.29	0.03	24%
<i>C. difficile</i> Infection	6.24	5.29	<0.001	15%

\*ESBL (*Klebsiella* and *E. coli*) combined; <sup>§</sup>Data from 18 hospitals

**Conclusion:** The common use of a >3-day definition for reporting nosocomial acquisition and infections produces significantly lower rates than a >2-day definition. This difference could substantially impact hospital rankings for public reports based upon differences in definitions alone. These data support the CDC's decision to standardize nosocomial assessment using a >2 calendar day rule in January 2013.

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## 1314. Evaluation of Differences in Population-Based Incidence of *Clostridium difficile* Infection across Diverse U.S. Geographic Locations, 2010

Part of Session: 181. Challenges in *C. difficile* Infection Surveillance

11:00 a.m.

**FERNANDA LESSA, MD<sup>1</sup>**, YI MU, PHD<sup>2</sup>, JESSICA COHEN, MPH<sup>1</sup>, GHINWA DUMYATI, MD, FSHEA<sup>3</sup>, MONICA M. FARLEY, MD<sup>4</sup>, LISA WNSTON, MD<sup>5</sup>, KELLY KAST, MPH<sup>6</sup>, STACY HOLZBAUER, DVM<sup>7</sup>, JAMES MEEK, MPH<sup>8</sup>, ZINTARS G. BELDAVS, MS<sup>9</sup>, L. CLIFFORD MCDONALD, MD<sup>1</sup>, SCOTT FRIDKIN, MD<sup>1</sup> and EIP CDI SURVEILLANCE INVESTIGATORS; <sup>1</sup>Division of Healthcare Quality Promotion, Centers for Disease Control and Prevention, Atlanta, GA, <sup>2</sup>Centers for Disease Control and Prevention, Division of Healthcare Quality Promotion, Atlanta, GA, <sup>3</sup>University of Rochester, Rochester, NY, <sup>4</sup>Emory University School of Medicine, Atlanta, GA, <sup>5</sup>University of California, San Francisco/San Francisco General Hospital, San Francisco, CA, <sup>6</sup>Colorado Department of Public Health and Environment, Denver, CO, <sup>7</sup>CDC CEFO assigned to the MN Dept. of Hlth, St. Paul, MN, <sup>8</sup>CT EIP, New Haven, CT, <sup>9</sup>Oregon Health Authority, Portland, OR

**Background:** *C. difficile* infection (CDI) diagnosis and treatment are no longer restricted to hospital settings. Accurate estimates of CDI nationally will require accounting for differences in population- and diagnostic-specific factors (e.g., age, testing practices) that influence incidence measures. We analyzed population-based data to identify these factors.

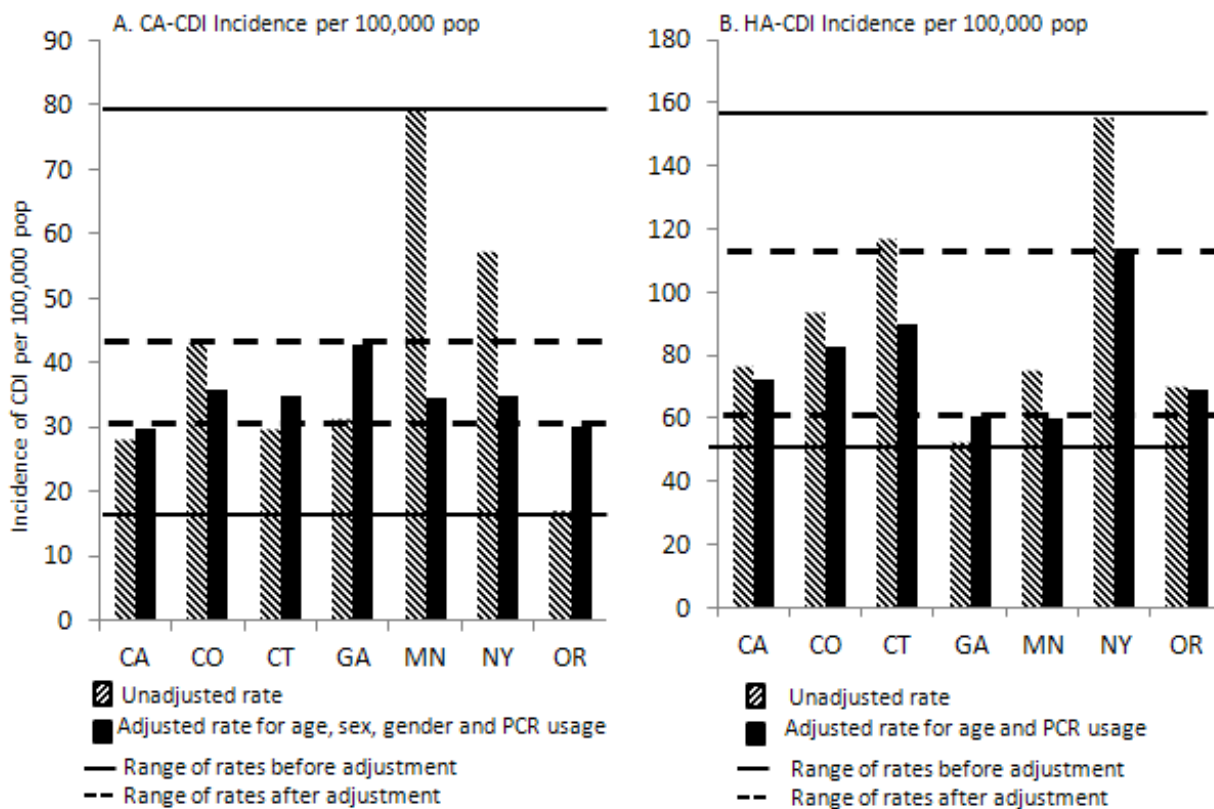
**Methods:** Population-based surveillance for persons  $\geq 1$  year of age was conducted in 21 counties in 7 U.S. states (8.5 million pop) in 2010. A CDI case was defined as a positive *C. difficile* toxin or molecular assay on a stool specimen from a person without a prior positive assay in the past 8 weeks. Cases were classified as community-associated (CA) if stool was collected as an outpatient or  $\leq 3$  days of admission in a person with no overnight stay in a healthcare facility in the

past 12 weeks; otherwise they were classified as healthcare-associated (HA). We queried participating laboratories about molecular diagnostics (e.g., PCR) utilization. The U.S. Census and Area Resource File provided county-level demographics and healthcare utilization data. Two regression models (CA- and HA-CDI) were built to evaluate factors associated with higher CDI incidence. Site-specific incidence was calculated using 2010 U.S. Census and adjusted based on the regression models.

**Results:** Of 10,062 cases identified, 32% were CA. Overall CDI incidence per 100,000 was higher among persons who were female (137 vs. 99;  $P=0.01$ ), white (140 vs. 75;  $P<.001$ ), or > 64 years (632 vs. 59;  $P<.001$ ). Unadjusted incidence varied by site; CA-CDI ranged from 28-79/100,000 and HA-CDI ranged from 70-155/100,000. By multivariate analysis independent predictors of higher CA-CDI incidence were age, race, sex, and PCR usage; for HA-CDI only age was a statistically significant predictor. After adjusting for relevant factors, the range of incidence narrowed greatly; CA-CDI ranged from 29-42/100,000 and HA-CDI ranged from 59-111/100,000 (Figure).

**Conclusion:** Differences in CDI incidence across sites can be partially explained by differences in PCR usage, age, race and gender, especially for CA-CDI cases. Variation in antimicrobial use and infection control practices, not captured in this analysis, may contribute to the remaining differences in CDI incidence.

Figure: Unadjusted and Adjusted Community- and Healthcare-Associated Population-Based Incidence



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### 1315. Effect of Nucleic Acid Amplification Testing on Population-based Incident Rates of *Clostridium difficile* Infection (CDI)

Part of Session: 181. Challenges in *C. difficile* Infection Surveillance

11:15 a.m.

**CAROLYN GOULD, MD<sup>1</sup>**, JONATHAN EDWARDS, MSTAT<sup>1</sup>, JESSICA COHEN, MPH<sup>1</sup>, L. CLIFFORD MCDONALD, MD<sup>1</sup>, MONICA M. FARLEY, MD<sup>2</sup>, HELEN JOHNSTON, MPH<sup>3</sup>, LUCY WILSON, MD<sup>4</sup>, SAMIR HANNA, MD<sup>5</sup>, LISA WINSTON, MD<sup>6</sup>, STACY HOLZBAUER, DVM<sup>7</sup>, CAROL LYONS, MPH<sup>8</sup>, ERIN PHIPPS, DVM<sup>9</sup>, GARY HOLLICK, PHD<sup>10</sup>, ZINTARS G. BELDAVS, MS<sup>11</sup>, DALE GERDING, MD, FIDSA<sup>12</sup>, FERNANDA LESSA, MD<sup>1</sup> and CDC'S CLOSTRIDIUM DIFFICILE INFECTION SURVEILLANCE INVESTIGATORS; <sup>1</sup>Centers for

Disease Control and Prevention, Atlanta, GA, <sup>2</sup>Atlanta Veterans Medical Center, Atlanta, GA, <sup>3</sup>Colorado Department of Public Health and Environment, Denver, CO, <sup>4</sup>Maryland Department of Health and Mental Hygiene, Baltimore, MD, <sup>5</sup>Tennessee Department of Health, Nashville, TN, <sup>6</sup>University of California, San Francisco/San Francisco General Hospital, San Francisco, CA, <sup>7</sup>CDC CEFO assigned to the Minnesota Department of Health, St. Paul, MN, <sup>8</sup>Connecticut Emerging Infections Program, New Haven, CT, <sup>9</sup>New Mexico Emerging Infections Program, Albuquerque, NM, <sup>10</sup>University of Rochester Medical Center, Rochester, NY, <sup>11</sup>Oregon Health Authority, Portland, OR, <sup>12</sup>Edward Hines, Jr. Veterans Affairs Hospital, Hines, IL

#### **Background:**

Nucleic acid amplification tests (NAATs) targeting toxin genes of *C. difficile* have higher sensitivity than enzyme immunoassays (EIA) and are being adopted by clinical laboratories. The use of NAAT is likely to increase detection of CDI, but the magnitude of the increases on incidence rates is unknown.

#### **Methods:**

CDI case counts (case defined as a positive *C. difficile* stool specimen by toxin or molecular assay from a resident of the surveillance catchment area without a prior positive assay in the past 8 weeks) and laboratory testing methods from population-based surveillance operating across 35 counties in 10 U.S. states during 2009-2011 were evaluated. Labs that changed from EIA to NAAT as first line testing ("switch labs") were compared to labs that only used EIA during the evaluation period ("non-switch labs") which served as controls. The median ratio of CDI case counts for switch labs during equivalent bimonthly time intervals post- and pre- switch, to control for seasonal variation, was compared to the median ratio for non-switch labs (same catchment area, same time). A one-sided non-parametric median test was used for comparison. The change in CDI incidence in each catchment area attributable to NAAT was calculated as the ratio of the switch lab median ratio over the non-switch lab median ratio, by EIP site. The proportions of stools tested that were *C. difficile* positive in the 3 months pre- and post- NAAT in switch labs were compared using a Mid-P exact test.

#### **Results:**

Five switch labs from 3 states (CA, GA, CO) were compared to a total of 43 non-switch labs. The number of months evaluated ranged from 14 to 24. The post/pre median ratios of case counts for switch labs were greater than the median ratios for non-switch labs in all states: CA: 1.78 vs. 1.0 ( $P=0.008$ ); GA: 1.59 vs. 1.0 ( $P=0.01$ ); CO: 1.87 vs. 0.99 ( $P=0.006$ ), respectively. The relative percent increases in CDI incidence attributed to NAAT were 78% in CA, 59% in GA and 89% in CO. Percent of specimens testing positive increased from 8% to 19% ( $P<0.0001$ ) in the 3 months after implementation of NAAT.

#### **Conclusion:**

We expect that labs switching to NAAT will increase population-based incidence of CDI by 59%-89%. Analysis and interpretation of CDI rates require adjustment for more sensitive testing methods.

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## **1316. The Biology and Epidemiology of *Clostridium difficile* in Oxfordshire Hospitals 2007-2010**

Part of Session: 181. Challenges in *C. difficile* Infection Surveillance

11:30 a.m.

**MADELEINE CULE, PHD<sup>1</sup>**, RORY BOWDEN, PHD<sup>2</sup>, DAVID EYRE, BM, BCH<sup>3</sup>, A. SARAH WALKER, PHD<sup>1</sup>, DAVID GRIFFITHS, BSC<sup>4</sup>, JOHN FINNEY<sup>1</sup>, DAVID WYLLIE, PHD<sup>1</sup>, DERRICK CROOK, MB, BCH<sup>2</sup>, TIM PETO, MB BS, DPHIL<sup>2</sup>, PETER DONNELLY<sup>2</sup> and INFECTIONS IN OXFORDSHIRE RESEARCH DATABASE; <sup>1</sup>NIHR Oxford Biomedical Research Centre, Oxford, United Kingdom, <sup>2</sup>University of Oxford, Oxford, United Kingdom, <sup>3</sup>Nihr Oxford Biomedical Research Centre, Oxford, United Kingdom, <sup>4</sup>National Institute for Health Research Oxford Biomedical Research Centre, Oxford, United Kingdom

**Background:** *Clostridium difficile* is a major cause of healthcare-associated diarrhoea and controlling its spread is the focus of significant public health effort. However, its biology and transmission epidemiology are incompletely understood.

**Methods:** Admission records and ward movements for inpatient stays in hospitals in Oxfordshire were combined with results of *C. difficile* EIA testing and culture between 1 September 2007 and 1 March 2010 (ca. 750,000 hospital admissions, 931 culture-positive patients). *C. difficile* cases were resolved into 70 distinct types using Multi-Locus Sequence Typing. A stochastic compartmental model for transmission of *C. difficile* between hospital contacts (EIA positive, EIA negative and not tested), including potential transmission within and between wards, and ward contamination, was fitted to the available data using Markov Chain Monte Carlo. Whole genome sequencing was used to validate inferences from the model and confirmed it was well-calibrated.

**Results:** Use of a probabilistic modelling approach allows novel insights into the biology and epidemiology of *C. difficile* infection. We see strong evidence for heterogeneity between strains, particularly that NAP1/ST1/Ribotype 027 is more

transmissible, being responsible for 50% of transmissions but only 13% of new introductions to the hospital. We find that a minority (22%) of patients continue to transmit for several months after initial diagnosis, and identify a potentially significant role for a median of 14 days (IQR 6-30 days) post-ward-discharge contamination leading to post-ward-discharge transmission. We find limited evidence for onward transmission prior to EIA test (likely onset of symptoms). We find no evidence for the existence of “superspreader” patients (the largest observed number of onward transmissions is 6), and find evidence for reductions in the amount of transmission over calendar time, and differences between the hospitals in the study. We confirm earlier findings that the majority of *C. difficile* cases cannot be explained by contact with symptomatic EIA-positive patients.

**Conclusion:** Transmission declined over time and varied by genotype. Statistical modelling provides a useful metric for assessing case-to-case transmission.

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## 1317. Prevalence and Risk Factors for Asymptomatic *Clostridium difficile* Carriage

Part of Session: 181. Challenges in *C. difficile* Infection Surveillance

11:45 a.m.

**ERIK DUBBERKE, MD, MSPH<sup>1</sup>**, FAISAL ALASMARI, MD<sup>2</sup>, SONDRA SEILER, BA<sup>1</sup>, TIFFANY HINK, BS<sup>3</sup> and CAREY-ANN BURNHAM, PHD<sup>4</sup>; <sup>1</sup>Washington University School of Medicine, St. Louis, MO, <sup>2</sup>Washington University School of Medicine, St. Louis, MO, <sup>3</sup>Washington University School of Medicine, St. Louis, MO, <sup>4</sup>Washington University School of Medicine, St. Louis, MO

**Background:** *C. difficile* infection (CDI) incidence has increased dramatically over the last decade. Recent studies suggest asymptomatic carriers may be an important reservoir of *C. difficile* (CD) in healthcare settings. We sought to identify the prevalence and risk factors for asymptomatic CD carriage on admission to the hospital.

### Methods:

Patients admitted to medical and surgical wards at Barnes-Jewish Hospital (BJH) without diarrhea and anticipated length of stay of >48 hours were prospectively enrolled from Jun 21, 2010, through Oct 25, 2011. Stool, or rectal swabs were collected within 48 hours of admit and stored at -30C. Demographics, comorbidities, and healthcare and medication exposures 90 days prior to admission were collected. Specimens were heat shocked at 80C for 10 min and inoculated into cycloserine, cefoxitin, manitol broth with lysozyme and taurocholate (Anaerobe Systems). Growth was plated onto blood agar. CD was identified by colony and Gram stain morphology, and standard biochemical tests. CD isolates were subcultured in BHI, and culture supernatant tested for GDH and toxins A and B (C. DIFF QUIK CHEK COMPLETE). Chi-square/Fisher's exact test was used for data analysis.

### Results:

259 subjects had an admission stool/swab specimen. 204 (79%) were not colonized, 40(16%) had toxigenic CD (TCD), and 15(6%) had nontoxigenic CD. There were no differences between TCD colonized and uncolonized subjects for age (mean 56 v 58, p=.46) or proportion that were admitted to the medicine service (83% v 88%, p=.32), admitted from another healthcare facility (33% v 24%, p=.23), or reason for admission (p=.45). There were no differences in any of 12 comorbidities or past history of CDI (1% v 2%, p=.82). There were also no differences in antibiotics exposure in the 90 days prior to admission (55% v 56%, p= .91), or hospitalization in the prior 90 days (50% v 50%, p=.43). 4(2%) TCD colonized patients and 2(1%) uncolonized patients were diagnosed with CDI (p=.07).

**Conclusion:** There was a high prevalence of TCD colonization on admission to BJH. There were no associations between demographics or antibiotic or healthcare exposures between colonized and uncolonized patients. Asymptomatic TCD carriers may be an important source of TCD in acute care facilities.

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