October 2018 and will conclude in June 2019. BCID2 Panel performance is compared with reference methods of microbial culture as well as PCR/sequencing for AMR genes. In addition, BCID2 Panel MRSA results are compared with the FDA-cleared Xpert MRSA/SA BC system (Cepheid, Inc). Relevant bacterial isolates recovered from PBCs are also evaluated by various phenotypic antimicrobial susceptibility testing (AST) methods. The prospective evaluation is supplemented with a second study that involves testing of ~300 pre-selected, archived PBCs containing rare organisms. The third study includes over 500 seeded blood cultures containing very rare organisms with an evaluation of co-spiked samples.

Results. With over 1,200 samples tested to date (out of an anticipated 1,800 total), the BCID2 Panel has demonstrated an overall sensitivity of >98% and specificity of >99% for identification of microorganisms compared with culture. Concordance between the BCID2 Panel and the Xpert MRSA/SA BC test is >99% for identification of MRSA. Evaluation of BCID2 Panel AMR gene detection relative to AST and PCR is ongoing.

Conclusion. The FilmArray® BCID2 Panel appears to be a sensitive, specific, and robust test for rapid detection of microorganisms and MRSA in PBCs. With the use of this comprehensive test, improved antimicrobial stewardship is anticipated.

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

652. Impact of FilmArray Meningitis Encephalitis Panel on HSV Testing and Acyclovir Use in Children Beyond the Neonatal Period

Kevin Messacar, MD[1]; Kevin Messacar, MD[1]; James Gaensbauer, MD[1]; Meghan Birkholz, MSPH[2]; Claire Levek, MS[3]; James Todd, MD[1]; Ken Tyler, MD[3]; Samuel Dominguez, MD PhD[1]; [1]University of Colorado/Children's Hospital Colorado, Aurora, Colorado; [2]Children's Hospital Colorado, Aurora, Colorado; [3]University of Colorado, Aurora, Colorado

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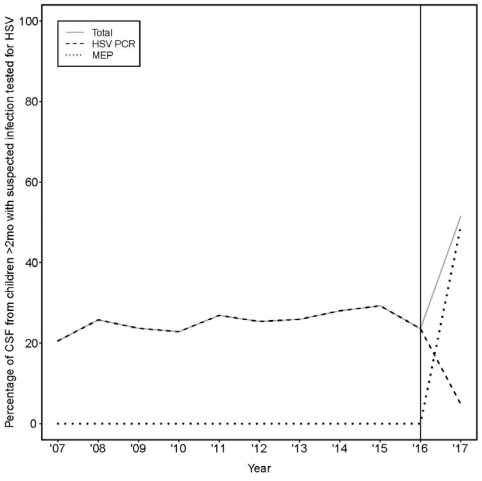
Background. Testing and empiric use of acyclovir for herpes simplex virus (HSV) in children beyond the neonatal period undergoing lumbar puncture for suspected central nervous system (CNS) infection doubled in the past decade, while the incidence of HSV CNS infection is unchanged. A new syndromic multiplex PCR panel (FilmArray Meningitis Encephalitis Panel [MEP]) rapidly detects 14 pathogens in cerebrospinal fluid (CSF), including HSV. The impact of MEP implementation on HSV testing and acyclovir use is unknown.

Methods. We retrospectively compared CSF testing and acyclovir use in the pre-MEP era January 1, 2007–January 22, 2017 to post-implementation era of MEP January 23, 2017–December 31, 2017 amongst children >60 days with a CSF specimen sent to the Children's Hospital Colorado microbiology laboratory. HSV singleplex PCR testing was available in both the pre-MEP and MEP eras.

Results. The proportion of CSF specimens from children with suspected CNS infection undergoing HSV testing (MEP or HSV PCR) doubled from 25% in the pre-MEP era to 54% in the MEP era ($P < 0.01$; Figure 1). In the MEP era, HSV testing was conducted by MEP in 96% of cases and HSV PCR in 8% of cases. In both eras, a majority of CSF specimens undergoing HSV testing had no pleocytosis (63% vs. 59%, $P = 0.27$). Children with negative HSV testing by MEP were less likely to be started on acyclovir than those with negative HSV testing by singleplex PCR (18% vs. 50%, $P < 0.01$) and, amongst those started, acyclovir was discontinued sooner, after a median 3 vs 5 doses ($P = 0.05$). Overall, however, a similar proportion of children with suspected CNS infection received acyclovir in the MEP and pre-MEP eras (13% vs. 12%), despite a low rate of HSV positivity (0.5% vs. 0%).

Conclusion. Implementation of MEP for syndromic CSF testing in children >60 days with suspected CNS infection doubled HSV testing without affecting the rate of empiric acyclovir initiation. Patients with negative HSV testing on MEP were less likely to be started on acyclovir, and if started, received fewer doses than those who tested negative on HSV singleplex PCR, likely due to more rapid turnaround time. However, increased MEP testing offset this, suggesting increased use of newer rapid syndromic tests will not cure creeping empiricism. Diagnostic stewardship targeting MEP use toward children with pleocytosis to decrease unnecessary test utilization are warranted.



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

653. Diagnosis of Burn Sepsis Using the FcMBL ELISA: A Pilot Study in Critically Ill Burn Patients

Kevin S. Akers, MD[1]; Taylor Schlotman, PhD[1]; Lee C. Mangum, PhD[1]; Gerardo Garcia, MS[1]; Amanda Wagner, RN[1]; Benjamin Seiler, BS[2]; Mark Cartwright, PhD[2]; Sami Rifai, BA[3]; Donald Ingber, MD,PhD[4]; Michael Super, PhD[2]; [1]US Army Institute of Surgical Research, JBSA Fort Sam Houston, Texas; [2]Wyss Institute at Harvard University, Boston, Massachusetts; [3]Wyss Institute for Biologically Inspired Engineering, Brookline, Massachusetts; [4]Harvard University, Boston, Massachusetts

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Background. Infection is the leading cause of death among burn survivors, with sepsis associated with more extensive burns. Conventional diagnostic criteria are insensitive in this population. We examined a novel diagnostic ELISA based on Mannose-Binding Lectin (MBL) linked to an immunoglobulin Fc domain, which measures the concentration of Pathogen-Associated Molecular Patterns (PAMPs) across a broad range of bacterial and fungal organisms, for diagnosis and antimicrobial management of sepsis in burn patients.

Methods. We prospectively enrolled burn patients with ≥15% Total Body Surface Area (TBSA) burns into groups of noninfected, sepsis, or incipient infection, and healthy volunteers. Sepsis was defined by clinical actions responsive to sepsis. The FcMBL ELISA was performed daily using fresh whole blood. Burn subjects were sampled daily until completing antimicrobials, for 14 days if noninfected, and once for healthy controls. Differences in median PAMP concentrations between groups were assessed with the Kruskal–Wallis test, including multiple comparisons between categories.

Results. 14 burn patients (3 noninfected, of whom 1 died prior to sampling, 4 Sepsis, 7 Incipient) were enrolled. The median (25–75% CI) PAMP concentration was 0.53 (0.12–1.34) ng/mL in healthy controls, 3.725 (2.53–5.94) ng/mL in noninfected, 2.22 (1.42–4.62) ng/mL in incipient, and 1.59 (0.83–2.29) ng/mL in sepsis groups. PAMP concentrations in sepsis were different ($P = 0.0057$) from noninfected, but incipient did not differ from noninfected ($P = 0.2025$). The dynamic range was lower in healthy controls (2.69 ng/mL) than incipient (4.57 ng/mL), sepsis (4.70 ng/mL), or noninfected (5.90 ng/mL). PAMP elevations correlated with clinical deterioration from infection, and were not associated with OR visits for debridement and grafting. 7 of 11 infected patients had declining PAMP levels at completion of antimicrobial therapy. 2 subjects had PAMP elevations associated with *Aspergillus* molds in their burn wounds.

Conclusion. The FcMBL ELISA assay may be useful for diagnosis of infection in burn patients, and may facilitate earlier discontinuation of antimicrobials. This assay may also have a novel utility for early diagnosis of Invasive Fungal Infection.

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

654. Evaluation of the Febridx Host Response Point-of-Care Test to Differentiate Viral From Bacterial Etiology in Adults Hospitalized with Acute Respiratory Illness During Influenza Season

Kate Beard, BMBS[1]; Cathleen Chan, MBBS[2]; Samuel Mills, BM, BCh[2]; Stephen Poole, MBBS[3]; Nathan Brendish, MBBS[1]; Tristan William. Clark, BM, MRCP, DTM&H, MD[1]; [1]Universitty of Southampton, Southampton, UK; [2]University Hospital Southampton Foundation NHS Trust, Southampton, UK, ; [3]NIHR Southampton Biomedical Research Centre, Southampton, UK

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Background. Antibiotics are overused in patients hospitalized with acute respiratory illness (ARI). Diagnostic uncertainty regarding microbial etiology contributes to this practice and so a host response test that can distinguish between viral and bacterial infection has the potential to reduce unnecessary antibiotic use. The FebriDx is a low cost, rapid, host response POCT that uses fingerpick blood samples to distinguish between viral and bacterial infection but has not been evaluated in hospitalized adults with ARI.

Methods. We took fingerpick blood samples from adult patients with ARI, hospitalized during influenza season, and tested them on the FebriDx. Respiratory samples were tested for viruses on the FilmArray Respiratory Panel (FARP). The FebriDx was evaluated for ease of use, failure rate and accuracy of the results (Viral, Bacterial, Negative).

Results. 149 patients were approached and 10 patients declined fingerpick testing. A valid result was obtained from 124/139 (89%) overall. Common user comments included test failure due to difficulty of getting blood to fill the capillary tube and difficulty in interpreting the results lines due to the variability of color change. 111/124 (89%) were tested for viruses by FARP. 69/111 (62%) had viruses detected. Of 69 patients with viruses detected, 41 (59%) had influenza, 12 (17%) rhino/enterovirus and 16 (23%) other viruses. 44/69 (64%) had a viral FebriDx result. For influenza-positive patients 34/41 (83%) had a viral FebriDx result, 1/12(8%) of rhinovirus-positive patients had a viral FebriDx result and 9/16 (56%) of patients with other viruses detected had a viral FebriDx result. These are interim results. Full results for 200 patients will be available at presentation.

Conclusion. The use of the FebriDx POC was associated with a failure rate of ~10% and problems with the interpretation of result lines. FebriDx was not sufficiently accurate in differentiating viral and bacterial infection when using detection of virus by PCR as the definition of viral infection; however, FebriDx had a high PPV for all viral

detection and for influenza detection in this cohort and could have utility in hospital emergency departments.

Table 1. Diagnostic accuracy of FebriDx in distinguishing viral ARI from non-viral ARI

	All patients, n=111	Excluding Rhinovirus and Mycoplasma, n=97
Sensitivity	64%	75%
Specificity	88%	91%
PLR	4.5	7.5
NLR	0.4	0.3
PPV	88%	92%
NPV	59%	72%
Accuracy	72%	81%

PLR, positive likelihood ratio. NLR, negative likelihood ratio.
PPV, positive predictive value. NPV, negative predictive value

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

655. Detection of Antibiotic Resistance Genes in Clinical Samples using T2 Magnetic Resonance

Jessica L. Snyder, PhD[1]; Brendan Manning, PhD[1]; Robert Shivers, PhD[1]; Daniel Gamero, BS[1]; Heidi Giese, PhD[1]; Nu Phung, BS[1]; Benjamin Chang, BS[1]; Heather Lapp, MS[1]; Riccardo Paggi, PhD[2]; Brunella Posteraro, MD[3]; Maurizio Sanguinetti, PhD[3]; Giulia De Angelis, MD, PhD[3]; Antonella Mencacci, MD[2]; Thomas J. Lowery, PhD[1]; [1]T2 Biosystems, Lexington, Massachusetts; [2]University of Perugia, Perugia, Umbria, Italy; [3]Policlinico Universitario Agostino Gemelli, Rome, Lazio, Italy

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Background. Antibiotic-resistant bacteria are spread through selective pressure from the use of broad-spectrum empirical therapies, mobile genetic elements that pass resistance genes between species, and the inability to rapidly and appropriately respond to their presence. Resistance gene identification is often performed with post culture molecular diagnostic tests. The T2Resistance Panel, which detects methicillin resistance genes *mecA/C*; vancomycin resistance genes *vanA/B*; carbapenemases bla_{KPC}, bla_{OXA-48},bla_{NDM}, bla_{VIM}, and bla_{IMP}; AmpC β-lactamases bla_{CMY} and bla_{DHA}; and extended-spectrum β-lactamases bla_{CTX-M} directly from patient blood samples, is based on T2 magnetic resonance (T2MR), an FDA-cleared technology with demonstrated high sensitivity and specificity for culture-independent bacterial and fungal species identification. Here we report the clinical performance of T2MR detection of resistance genes directly from patient blood samples.

Methods. Patients with a clinical diagnosis of sepsis and an order for blood culture (BC) were enrolled in the study at two sites. BCs were managed using standard procedures and MALDI-TOF for species identification. Resistance testing with the T2MR assay was performed on a direct patient draw and compared with diagnostic test results from concurrent BC specimen and BC specimen taken at other points in time. The potential impact on therapy was evaluated through patient chart review.

Results. T2MR detected the same resistance genes as detected by post culture diagnostics in 100% of samples from concurrent blood draws. Discordant results occurred when T2MR was taken ≥48 hours after BC for patients on antimicrobial therapy. The average time to positive result was 5.9 hours with T2MR vs. 30.6 hours with post-culture molecular testing.

Conclusion. The T2Resistance Panel detected antibiotic resistance genes in clinical samples and displayed agreement with post culture genetic testing. T2MR results were achieved faster than culture-dependent diagnostic testing results and may allow for an earlier change from empiric to directed therapy. The use of culture-independent diagnostics like T2MR could enable a quicker response to antibiotic-resistant organisms for individual patients and developing outbreaks.

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

656. Prioritizing Gram-Negative Bacteremia (GNB) Cases for Rapid Detection by β-Lactam Resistance (BLR) and Patient Outcomes

Erin K. McCreary, PharmD, BCPS, BCIDP[1]; Lloyd Clarke, BSc[1]; Rachel V. Marini, PharmD[2]; Minh-Hong Nguyen, MD[3]; Cornelius J. Clancy, MD[3]; Ryan K. Shields, PharmD, MS[3]; [1]University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania; [2]University of Pittsburgh Medical Center Presbyterian, Pittsburgh, Pennsylvania; [3]University of Pittsburgh, Pittsburgh, Pennsylvania

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Background. GNB is associated with significant morbidity and mortality. The availability of rapid diagnostic tests (RDTs) provides an opportunity to improve outcomes. Our goal was to review GNB and its empiric treatment at our center in order to devise rational approaches to diagnostic stewardship and use of RDTs.

Methods. All patients with GNB from 2010 to 2018 were evaluated. BLR was defined by 2019 CLSI breakpoints; phenotypes are shown in Table 1.

Results. A total of 2795 GNB cases were included (Table 2); 57% occurred within the first 24 hours of hospitalization and 29.3% in the ICU. The median length of stay (LOS) was 12 days; 17.2% of patients were re-admitted within 30 days. Fourteen- and 30-day mortality rates were 13.7% and 19.5%, respectively. Rates of death were higher (30 days; 26.3% vs. 17.1%; $P < 0.001$) and median LOS longer (17 vs. 11 days; $P < 0.001$) among patients with BLR compared with susceptible GNB. Thirty-day mortality rates were highest for CRE (30.1%) and BLR *P. aeruginosa* (BLR-Pa; 32.8%, Figure 1). 47.7% of BLR GNB were non-CRE/ESBL, which demonstrated higher mortality rates than CRE/ESBL (30 days; 27.6% vs. 21.2%; $P = 0.048$). Most common empiric regimens prescribed were piperacillin–tazobactam (TZP; 50.3%), cefepime (FEP; 24.2%), carbapenem (9.3%), or other agents (16.2%). 21.6% of GNB patients received inactive empiric treatment (IET). Empiric TZP (21.9%) was more likely to be inactive than FEP (17.5%; $P = 0.05$), but not a carbapenem (20.7%; P = NS). 57.6% of patients with inhibitor-resistant Enterobacteriaceae (IRE) received TZP empirically. Receipt of IET was associated with higher rates of death (30 days; 22.5% vs. 16.7, $P = 0.03$) and longer LOS (14 vs. 11 days; $P < 0.001$) than receipt of active ET. Rates of IET varied by pathogen (Figure 1).

Conclusion. IET is common against BLR GNB and associated with poor pt outcomes, highlighting the potential for RDTs and diagnostic stewardship teams (DSTs) to improve care. Genotypic RDTs detect most CRE/ESBL, but may miss nearly 50% of BLR GNB cases at our center. BLR-Pa and IRE are pathogens associated with prolonged LOS, and high rates of IET and death. These pathogens could be detected earlier by phenotypic RDTs and prioritized by DSTs to optimize early treatment regimens.

Table 1. Definitions of β-lactam resistant (BLR) phenotypes

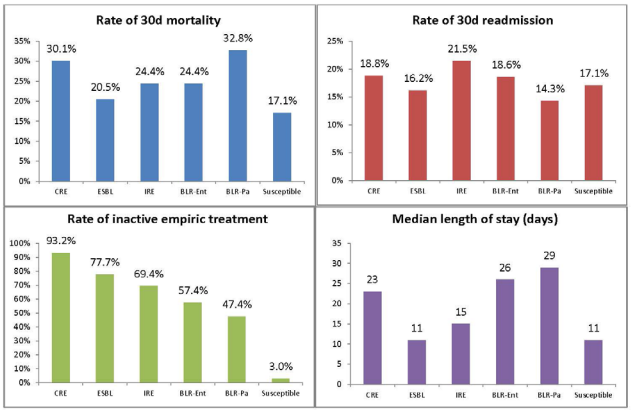
Resistance phenotype	Definition
Carbapenem-resistant Enterobacteriaceae (CRE)	Non-susceptible to ertapenem, meropenem, and/or imipenem
Extended-spectrum β-lactamase (ESBL)	Non-susceptible to aztreonam and/or ceftriaxone
Inhibitor-resistant Enterobacteriaceae (IRE)	Non-susceptible to piperacillin/tazobactam Susceptible to ceftriaxone
BLR Enterobacter species (BLR-Ent)	Non-susceptible to ceftriaxone and/or cefepime Susceptible to carbapenems
BLR Pseudomonas aeruginosa (BLR-Pa)	Non-susceptible to aztreonam, cefepime, ceftazidime, meropenem, and/or piperacillin/tazobactam
BLR Serratia marcescens (BLR-Sm)	Non-susceptible to ceftriaxone and/or cefepime Susceptible to carbapenems
Susceptible	Susceptible to aztreonam, ceftriaxone, cefepime, piperacillin/tazobactam, cefepime, and all carbapenems

Table 2. Patient outcomes stratified pathogen following GNRB

Pathogen (n)	Resistance phenotype	No. of cases	14d mortality, n (%)	30d mortality, n (%)	Median hospital length of stay (days)	30d hospital readmission, n (%)
E. coli (n=1079)	CRE	13	6 (46.2)	7 (53.9)	25	1 (7.7)
	ESBL	158	26 (16.5)	33 (20.9)	10	23 (14.6)
	IRE	57	10 (17.5)	14 (24.6)	15	12 (21.1)
	Susceptible	851	112 (13.2)	161 (18.9)	9	141 (16.6)
K. pneumoniae (n=835)	CRE	104	19 (18.3)	32 (30.8)	29	21 (20.2)
	ESBL	52	7 (13.5)	10 (19.2)	16	11 (21.1)
	IRE	78	12 (15.4)	19 (24.4)	16	17 (21.8)
	Susceptible	601	63 (10.5)	93 (15.5)	12	103 (17.1)
E. cloacae (n=281)	CRE	48	9 (18.8)	12 (25.0)	21	10 (20.8)
	FEP-NS	28	5 (17.9)	5 (17.9)	38	6 (21.4)
	FEP-S, CRO-NS	17	2 (11.8)	3 (17.6)	17	3 (17.6)
	Susceptible	188	14 (7.4)	25 (13.3)	15	46 (16.0)
E. aerogenes (n=90)	CRE	5	0 (0)	1 (20.0)	12	0 (0)
	FEP-NS	4	0 (0)	0 (0)	18	1 (25.0)
	FEP-S, CRO-NS	21	6 (28.6)	8 (38.1)	22	4 (19.0)
	Susceptible	60	10 (16.7)	12 (20.0)	16	12 (20.0)
S. marcescens (n=165)	CRE	6	1 (16.7)	2 (33.3)	19	1 (16.7)
	FEP-NS	5	1 (20.0)	1 (20.0)	51	0 (0)
	FEP-S, CRO-NS	11	3 (27.3)	4 (36.4)	20	2 (18.2)
	Susceptible	143	20 (13.9)	22 (15.4)	19	23 (16.1)
P. aeruginosa (n=345)	NS to ≥ 3 BLs	46	14 (30.4)	18 (39.1)	32	8 (17.4)
	NS to 2 BLs	21	6 (28.6)	7 (33.3)	23	2 (9.5)
	NS to 1 BL	64	10 (15.6)	18 (28.1)	28	8 (12.5)
	Susceptible	214	26 (12.2)	38 (17.8)	14	27 (12.6)
Total		2795	382 (13.7)	545 (19.5)	12	482 (17.2)

Note. BL = β-lactam; CRE = Carbapenem-resistant Enterobacteriaceae; CRO = Ceftriaxone; ESBL = Extended-spectrum β-lactamase; FEP = Cefepime; IRE = Inhibitor-resistant Enterobacteriaceae; NS = Non-susceptible; TZP = Piperacillin-tazobactam

Figure 1. Patient outcomes stratified by BLR GNRB phenotype



Note. BLR-Ent = β-lactam resistant Enterobacter species; BLR-Pa = β-lactam resistant *Pseudomonas aeruginosa;* CRE = Carbapenem-resistant Enterobacteriaceae; ESBL = Extended-spectrum β-lactamase; FEP = Cefepime; IRE = Inhibitor-resistant Enterobacteriaceae; NS = Non-susceptible; TZP = Piperacillin-tazobactam

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