and Laboratory Diagnostics, Tohoku University Graduate School of Medicine, Sendai, Japan, [2]Miyarisan Pharmaceutical Co., Ltd., Saitama, Japan, [3]Tokyo R&D Center, Miyarisan Pharmaceutical Co., Ltd., Saitama, Japan

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Background. Shiga toxin-producing *Escherichia coli* (STEC) causes serious gastrointestinal illness. Although O157 is predominant, non-O157 infections have been increasingly reported worldwide. We used whole-genome sequencing (WGS) to investigate molecular characteristics and phylogeny of STEC isolates.

Methods. A total of 22 STEC isolates from symptomatic patients (n = 13) and asymptomatic carriers (n = 9) in a Japanese region during 2016–2017 were used. Serogroups were O157, O26 and O103 (n = 5, 12, and 5, respectively). WGS was performed using an Illumina Miseq. Genomic analysis was performed using web-based tools by the Center for Genomic Epidemiology. Single nucleotide polymorphism detection and construction of phylogenetic tree were performed using Mauve software.

Results. Of 76 virulence genes, 32 (42%) were detected (Figure 1). Eighteen (82%) and 7 (32%) isolates contained *stx1* and *stx2*, respectively. Twelve (91%) contained *eae*. *stx2* was more frequent in isolates from patients (P < 0.05), whereas *cba* was more frequent in isolates from carriers (P < 0.05). *stx2*, *etpD* were more frequent in O157 isolates (P < 0.05, respectively), whereas *stx1*, *efa1*, *cif*, *tccP*, *cba*, *lpfA* were more frequent in non-O157 isolates (P < 0.05, respectively). Nine acquired resistance gene (*aph(3´)-Ia*, $bla_{TEM\text{-}1b}$, *dfrA5*, *dfrA8*, *strA*, *strB*, *sul2*, *tetA*, *tetB*) were detected, while at least one was found in 6 (27%) isolates. Isolates from patients (5/13, 38%) were likely to have more resistance genes than isolates from carriers (1/9, 11%) (P = 0.33). Genotyping and multilocus sequence typing revealed all O26 isolates belonged to O26:H11 ST21, O103 belonged to O103:H2 ST17 and novel O103:H8 ST2836, while O157 belonged to O157:H7 ST11 and ST2966 (Figure 2). Phylogenetic tree showed O103:H8 ST2836 isolates clustered with O26, separated from O103:H2 ST17 (Figure 3). In a cluster of O26:H11 ST21 isolates, isolates from carriers formed a subcluster. O157 isolates clustered in a separate lineage. O157:H7 ST2966 isolates evolved from ST11.

Conclusion. Of the non-O157 isolates, O26:H11 ST21, which contained as many virulence genes as O157, was prevalent among both patients and carriers in our region, highlighting the importance of monitoring genomic characteristics of non-O157 STEC.

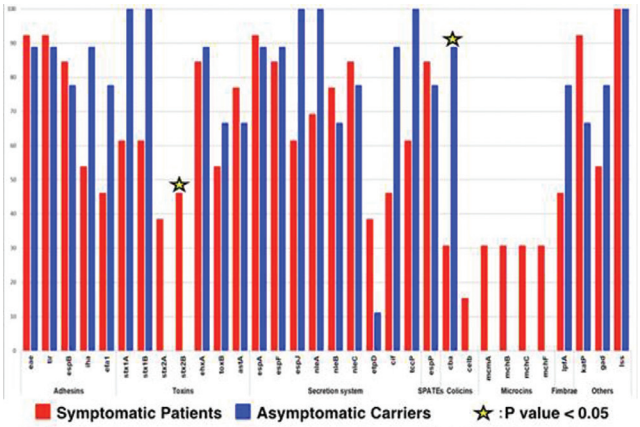

Fig. 1. Comparison of virulence genes in STEC isolates from symptomatic patients and asymptomatic carriers.

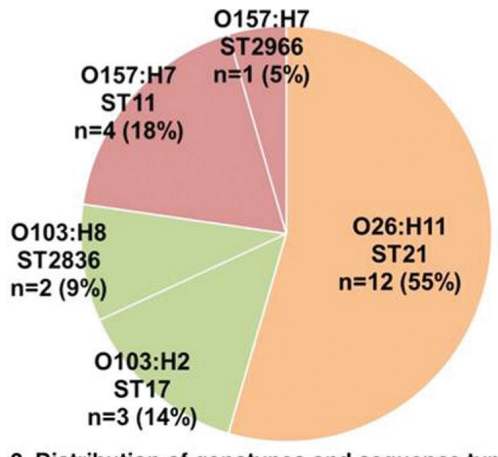

Fig. 2. Distribution of genotypes and sequence types (STs) among STEC isolates.

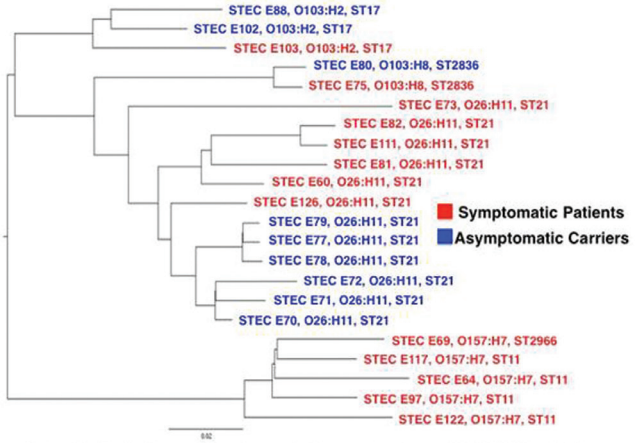

Fig. 3. Neighbor-joining phylogenic tree of STEC isolates.

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

651. Non-encapsulation of Pneumococci as a Potential Evasion Mechanism From Vaccines

Takeaki Wajima, PhD; Haruna Ishikawa, BA; Shiori Suzuki, N/A; Akane Matsuzawa, N/A; Hidemasa Nakaminami, PhD and Norihisa Noguchi, PhD; Microbiology, Tokyo University of Pharmacy and Life Sciences, Tokyo, Japan

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Background. Our group has been continuously performing epidemiological analyses on capsular types of pneumococci since 2007. Pneumococcal conjugate vaccine decreased the proportion of nonvaccine capsular types. Furthermore, null-capsule isolates that produced PspK were also identified in our analysis. In this study, we analyzed the genetic background of null-capsule pneumococci and the mechanism of nonencapsulation.

Methods. Twenty-seven null-capsule isolates from 430 pneumococci that were isolated between 2010 and 2014 were used for this study. The capsular type was identified by DNA sequence-based methods, and genetic backgrounds were compared by multilocus sequence typing. Among the null-capsule isolates, the SP2852 strain was employed for non-encapsulation analysis. The *pspK* gene of this strain was replaced with *ermB* by homologous recombination (SP2852 Δ*pspK*::*ermB*). Then, genomic DNA from SP2852 Δ*pspK*::*ermB* was transformed into encapsulated isolates via natural transformation. Clindamycin-resistant isolates were further analyzed by sequence.

Results. The proportion of null-capsule isolates tended to increase from 5% in 2010–2011 to 12.3% in 2014. These null-capsule isolates were classified into 14 STs that included STs previously identified as capsule-positive isolates. To assess non-encapsulation via natural transformation, two encapsulated strains (serotype 19F and 14) were cultured with genomic DNA from SP2852 Δ*pspK*::*ermB*. Subsequently, clindamycin-resistant null-capsule isolates were detected with high frequency (2.5×10^{-4}–8.7×10^{-5}). Sequence analysis showed capsular coding regions of these null-capsule isolates were replaced with that of Δ*pspK*::*ermB*. Furthermore, these isolates grew significant faster than their parent strains.

Conclusion. Null-capsule isolates with various genetic backgrounds were revealed gradually after introduction of vaccine. Moreover, encapsulated strains could take up genomic DNA of null-capsule isolates more easily and become a null-capsule strain by homologous recombination, suggesting that non-encapsulation and acquiring PspK resulted in the emergence of null-capsule strains by natural transformation. Furthermore, non-encapsulation could be beneficial for pneumococci as an evasion mechanism from vaccines.

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

652. What Is Blood Got to Do with It? Genetic Susceptibility to Norovirus and Rotavirus Infection: Results From the SUPERNOVA Network

Anita Kambhampati, MPH[1,2]; Robert L. Atmar, MD[3]; Frederick H. Neill, MA[4]; Maria C. Rodriguez-Barradas, MD[4,5]; Blanca Vargas, MD[4,6]; David O. Beenhouwer, MD[7,8]; Aleksandra Poteshkina, BS[8]; Vincent C. Marconi, MD[9,10]; Kathryn L. Meagley, MPH[9]; Sheldon T. Brown, MD[11,12]; Adrienne Perea, BS[12]; Hannah Browne, BS[2,13]; Rashi Gautam, PhD[1,2]; Scott Grytdal, MPH[2]; Michael D. Bowen, PhD[2]; Jan Vinjé, PhD[2]; Umesh D. Parashar, MBBS, MPH[2]; Aron J. Hall, DVM, MSPH[2] and Cristina V. Cardemil, MD, MPH[2]; [1]IHRC, Inc., Atlanta, Georgia, [2]Division of Viral Diseases, Centers for Disease Control and Prevention, Atlanta, Georgia, [3]Medicine, Baylor College of Medicine, Houston, Texas, [4]Baylor College of Medicine, Houston, Texas, [5]Michael E. DeBakey Veterans Affairs Medical Center, Houston, Texas, [6]Infectious Diseases Section, Michael E. DeBakey VA Medical Center, Houston, Texas, [7]David Geffen School of Medicine at UCLA, Los Angeles, California, [8]VA Greater Los Angeles Healthcare System, Los Angeles, California, [9]Atlanta VA Medical Center, Atlanta, Georgia, [10]Emory University School of Medicine, Atlanta, Georgia, [11]Icahn School of Medicine at Mt. Sinai, New York, New York, [12]James J. Peters VA Medical Center, Bronx, New York, [13]Oak Ridge Institute for Science and Education, Oak Ridge, Tennessee

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Background. Histo-blood group antigens (HBGAs), whose expression is controlled in part by fucosyltransferase 2 (FUT2) and 3 (FUT3) genes, serve as receptors for norovirus and rotavirus. Individuals without functional FUT2 (nonsecretors) or FUT3 (Lewis-negative) genes may have decreased susceptibility to norovirus and rotavirus infections. As the prevalence of secretor and Lewis status can vary by race and ethnicity, we assessed this association in a US Veteran population.

Methods. Stool and saliva specimens were collected from acute gastroenteritis (AGE) cases and age- and time-matched controls through a multisite, active surveillance platform at four Veterans Affairs hospitals (Atlanta, Bronx, Houston, Los Angeles). Stool specimens were tested with the FilmArray Gastrointestinal Panel; norovirus and rotavirus positive specimens were genotyped. Saliva specimens were analyzed for HBGA expression by EIA using glycan-specific monoclonal antibodies and lectins. Chi-squared and Fisher's exact tests were conducted to evaluate associations between secretor and Lewis status and infection with norovirus or rotavirus.

Results. From November 4, 2015–December 30, 2017, 670 AGE cases and 319 controls provided both stool and saliva specimens. Norovirus (21 GII.4 Sydney, 13 GII non-4, 7 GI, 10 untyped) and rotavirus (13 G12P[8], 1 G2P[4], 1 untyped) positive cases were more likely to be secretor positive (90% and 100%, respectively) compared with controls (76%) ($P = 0.03$ for both). Infections with GII.4 Sydney norovirus ($P < 0.01$) and G12P[8] rotavirus ($P < 0.05$) were significantly associated with secretor status. This association was not observed with other norovirus or rotavirus genotypes. No association was observed between Lewis status, race, or ethnicity and infection with norovirus or rotavirus.

Conclusion. Norovirus and rotavirus infections among a US Veteran population were associated with secretor status in a genotype-dependent manner, and with GII.4 Sydney norovirus and G12P[8] rotavirus, the most common strains. These associations are consistent with previously reported results, and suggest that the efficacy of interventions, such as vaccines, should include consideration of secretor status and predominantly circulating virus strains.

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653. To Treat or Not to Treat *Corynebacterium striatum?* That is the Question

Katrina Soriano, MD[1] and Richard Zuckerman, MD, MPH[2]; [1]Internal Medicine, Dartmouth Hitchcock Medical Center, Lebanon, New Hampshire, [2]Dartmouth-Hitchcock Medical Center, Lebanon, New Hampshire

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Background. As part of normal skin flora, *Corynebacterium striatum* (CS) can be isolated in clinical specimens and dismissed as a contaminant or colonizer. However, this organism is an emerging multi-drug-resistant opportunistic pathogen that causes serious infections and demonstrates worrisome resistance to antimicrobials. CS has been implicated in both osteomyelitis (OM) and septic arthritis (including prosthetic joint infections (PJI)), for which aggressive source control and antibiotics are needed. Because there is no accepted treatment standard when CS is found, we performed a retrospective analysis of patients with CS isolated from orthopedic sites.

Methods. We retrospectively analyzed cultures in which *CS* was isolated from 2015 to 2017. We restricted this analysis to patients with orthopedic cultures, bone and PJI. Charts were reviewed for clinical and microbiological data. Duration of follow-up was calculated to the last follow-up visit in our system for the patient after treatment of the infection. "Targeted" treatment was defined as treatment based on antimicrobial susceptibilities of the *CS*.

Results. We identified 20 cases of OM and 6 PJI with *CS* identified in culture. 17/26 (65%) were multi-drug resistant. *CS* was the only organism (monomicrobial) in 46% (12/26) of cultures (8/20 OM, 4/6 PJI). All monomicrobial OM and PJI received targeted treatment and were cured clinically at the last follow-up (average 201 days OM and 124 days PJI). Of 12 polymicrobial OM infections, all improved clinically by last follow-up; 75% (9/12) were treated with targeted treatment for *CS* and 25% (3/12) without targeted treatment. All polymicrobial PJI (2/2, 100%) improved without targeted treatment.

Conclusion. Our review suggests that CS can cause OM and PJI, with a high rate of drug resistance. Cure rates are excellent in monomicrobial infections when therapy is targeted to susceptibility. The role of targeted therapy for polymicrobial infections in which another more likely pathogen is found is not clear, particularly in the setting of effective surgical source control. Further prospective research is necessary to clarify the prevalence and factors associated with CS infections, and the importance of treatment of this organism in orthopedic infections.

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

654. Biofilm Forming Methicillin-Resistant *Staphylococcus aureus* Induces Renal Deterioration and Severe Virulence in a Mouse Bacteraemic Model

Shiro Jimi, PhD[1]; Motoyasu Miyazaki, PhD[2]; Yutaka Ueda, MSc[3] and Kota Mashima, MSc[3]; [1]Fukuoka University Faculty of Medicine, Fukuoka, Japan, [2]Department of Pharmacy, Fukuoka University Chikushi Hospital, Fukuoka, Japan, [3]Fukuoka University Hospital, Fukuoka, Japan

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Background. Methicillin-resistant *S. aureus* (MRSA), a responsible bacterium to nosocomial infection, induces biofilm (BF) infection. We previously indicated that individual MRSA manifests a various BF forming ability, and high BF formers infused in the blood can survive even after phagocytosis by Kupffer cells. In this study, we advance the research to examine the development of BF formation in tissues during 96 h after infusion.

Methods. Out of 172 clinical isolates of MRSA, highest BF former (H-BF) and lowest BF former (L-BF) were used. Bacteria were infused via tail vain. Mice were checked for general status and bacterial distribution in the organs (liver, lung, spleen and kidney) at histological and bacteriological levels. BF was also histologically detected by stains for polysaccharides.

Results. After MRSA infusion, general status in L-BF maintained in normal range during the study, H-BF however revealed poor status, which was aggravated in accordance with time. After infusion, bacteria started to reappear in the blood after 24 h of the study, and, on 96 hour, H-BF exhibited an eight times greater extent than L-BF. Bacterial colonies were formed in the kidney in both of the groups, and colonies in the liver were only noted in H-BF. In the kidney, CFU in both of the groups increased by time, and its number on 96 h was significant greater in H-BF than L-BF. In H-BF, bacterial embolism accompanied with BF was histologically found in medullary capillaries in the kidney on 24 hours. Growing BF aggressively penetrated into the stroma and tubular lumen forming a wedge-like renal necrosis.

Conclusion. These results indicate that BF forming MRSA in the blood preferably settle and form BF in the kidney in mice, which leads to a biofilm infection and a severe deterioration. Although the mechanisms of kidney specific lesions formed by MRSA are still unclear, BF forming ability in MRSA might be crucially important for bacterial virulence *in vivo*.

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

655. Antimicrobial Susceptibility and Prevalence of Extra-intestinal Enterotoxigenic *Bacteroides fragilis* Among a 5-Year Collection of Isolates Causing Sepsis in Kuwait

Wafaa Jamal, MD, MSc, PhD, FRCPath, FIDSA; Fatima Khodakhast, BSc and Vincent Rotimi, MD, PhD, FRCPath, FIDSA; Microbiology, Faculty of Medicine, Kuwait University, Jabriya, Kuwait

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Background. *Bacteroides fragilis* is commonly associated with bacteremia, soft-tissues, intra-abdominal infections and abscesses. Enterotoxigenic (BFT) strains have emerged as important etiological cause of diarrhea in children and adults. This study was undertaken to investigate the antibiotic susceptibility of nonfecal clinical isolates and prevalence of BFT among a 5-year collection of isolates associated with bloodstream infections (BSI), often associated with significant mortality, vs. other infections.

Methods. Isolates of non-intestinal origin, sent from five leading hospitals in Kuwait, to our Anaerobe Reference Laboratory, for identification were studied. They were identified by VITEK MS (MALDI-TOF system). Susceptibility was performed with Etest on all isolates and results interpreted by the recommended criteria of CLSI 2016. Molecular detection of genes encoding enterotoxin (*bft*) production was carried out using *bft*F and *bft*R primers. Subsets of *bft*-positive isolates were also investigated by sequencing and correlated to various sepsis. Appropriate control strains were included in each run.

Results. The average age of the infected patients was 56.0 years and there were more males than females (63 vs. 35). The main sources of the isolates were intra-abdominal infections (IAI), lower respiratory tract infections (LRTI), BSI, wound infections (WI), and abscesses. A total of 256 isolates were studied out of which 98 (38.3%) were *bft*-positive. Of these 98, 72 (73.5%) were positive for subset genes *bft*-1, 24 (24.5%) *bft*-2 and 2 (2.0%) *bft*-3. The *bft*-positive isolates were associated with IAI (39.8%), LRTI (35.7%), BSI (9.2%), WI (11.2%) and abscess (4.1%). Percentage of *bft*-positive and *bft*-negative isolates resistant to clindamycin were 62 vs. 58%, imipenem 9 vs. 12%, meropenem 13 vs. 16%, metronidazole 5 vs. 4%, cefoxitin 15 vs. 26% and tigecycline 11 vs. 9%, respectively.

Conclusion. The proportion of BFT strains among our isolates was very high in this series. Overwhelming proportion belonged to the *bft*-1 subset which were the predominant isolates found in clinical infections. The *bft*-positive isolates were more resistant than the *bft*-negative isolates to clindamycin, metronidazole and tigecycline.

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

656. *E. coli* Clone Sharing and Persistence Within Households (HHs) in Relation to Fluoroquinolone (FQ) Resistance and ST131 Status

Jessica Boettcher, DO[1]; Connie Clabots, BS MT(ASCP)[2]; Stephen B. Porter, MS[2] and James R. Johnson, MD[1,2]; [1]Infectious Diseases and International Medicine, University of Minnesota, Minneapolis, Minnesota, [2]Minneapolis Veterans Affairs Medical Center, Minneapolis, Minnesota

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Background. Extraintestinal *E. coli* infections, a perennial source of morbidity and mortality, are increasingly difficult to treat due to emerging antibiotic resistance. Within-HH sharing of *E. coli* strains may contribute to this problem, but is poorly understood. Accordingly, we assessed *E. coli* strain sharing within the HHs of veterans with a clinical *E. coli* isolate, including in relation to FQ resistance and ST131 status.