

decreasing abundance of Proteobacteria at 1 month after FMT in responders. However, those changes of microbial composition did not occur in non-responders.

**Conclusion:** FMT is an effective way to decolonize CPE and VRE by restoration of the gut microbiome.

**Table 1. Comparisons of clinical characteristics between responders and non-responders (at 3 month).**

Characteristics	Total (N=23)	Responders (N=12)	Non-responders (N=11)	p value
Age at FMT (median, years)	58.0 ± 42.5	58.0 ± 38.0	57.0 ± 68.5	0.758
Male sex (%)	13(56.5)	8(50.0)	7(63.6)	0.680
BMI (median, kg/m <sup>2</sup> )	20.7 ± 7.2	21.4 ± 3.6	17.2 ± 8.0	0.325
Carriage MDRO				0.314
CPE	4(17.4)	3(25.0)	1(9.1)	
VRE	13(56.5)	5(41.7)	8(72.7)	
CPE/VRE	6(26.1)	4(33.3)	2(18.2)	
Duration of carriage before FMT(days)	56.0±84.0	74.0 ±157.0	55.0 ±40.0	0.281
Hospital stay of days before FMT(days)	54.5±67.0	56.0 ±163.0	56.0 ±38.0	0.902
ATB before FMT (within 1week)	14(60.9)	7(58.3)	7(63.6)	>0.999
Route of FMT				>0.999
Upper GI tract	12(52.2)	7(58.3)	5(41.7)	
Lower GI tract	11(47.8)	7(63.6)	4(36.4)	
Biology before FMT				
WBC (10 <sup>3</sup> /uL)	6.58±3.44	6.45 ±3.81	6.71 ±3.13	0.479
Hemoglobin (g/dL)	10.8±2.3	11.0 ±1.5	10.0±2.9	0.018
Platelet count (10 <sup>3</sup> /uL)	282.5±120.3	280.0 ±98.0	316.0 ±298.0	0.389
BUN (mg/dL)	12.7±7.8	13.2 ±8.9	8.0 ±10.1	0.124
Creatinine (mg/dL)	0.5±0.3	0.5 ±0.4	0.4 ±0.4	0.601
AST (IU/L)	27.5±27.0	23.0 ±16.0	30.0 ±49.0	0.139
ALT (IU/L)	20.5±24.0	20.0 ±18.0	22.5 ±26.0	0.895
Total cholesterol (mg/dL)	154.5±50.5	172.0 ±74.0	130.0 ±39.0	0.079
Triglyceride (mg/dL)	130.5±148.3	144.0 ±143.0	106.0±139.0	0.268
LDL cholesterol (mg/dL)	93.2±43.3	102.0 ±46.2	89.0 ±41.6	0.049
Albumin (mg/dL)	3.3±0.5	3.4 ±0.9	3.2 ±0.5	0.006
Fasting glucose (mg/dL)	95.0±13.3	95.0 ±13.0	95.0 ±14.0	0.901
CRP (mg/L)	6.9±22.4	3.7 ±29.6	12.3 ±20.1	0.480
Additional ATB				
ATB after FMT (within 1week)	15(65.2)	5(41.7)	10(90.9)	0.027
Hospital stay of days after FMT(days)	46.0±49.0	29.0 ±51.0	48.0 ±88.0	0.355

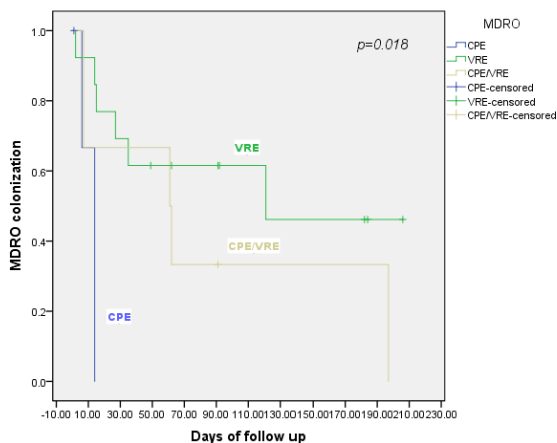
FMT, fecal microbiota transplantation; BMI, body mass index; MDRO, multi-drug-resistant organism; ATB, antibiotic treatment; EGD, esophagogastroduodenoscopy; CFS, colonoscopy; WBC, white blood cell; BUN, Blood urea nitrogen; AST, Aspartate transaminase; ALT, Alanine transaminase; LDL, Low density lipoprotein; CRP, C-reactive protein; S, success; F, failure; N/A, not available; C, complete; P, partial. Continuous variables are shown as medians ± IQR (3<sup>rd</sup> interquartile range-1<sup>st</sup> interquartile range) and categorical variables as numbers (percentage).

**Table 2. Impact of FMT on complete and partial MDRO decolonization, with or without ATB during the first week after transplantation**

Endpoint	All FMTs (N=23)		With ATB (N=19)		Without ATB (N=8)		p value
	Number	%	Number	%	Number	%	
<b>Complete MDRO decolonization</b>							
At 1 month	9/23	39.1	4/15	26.7	5/8	62.5	0.179
At 3 months	9/23	39.1	4/15	26.7	5/8	62.5	0.179
At 6 months	7/15	46.7	3/11	27.3	4/4	100.0	0.026
<b>Partial MDRO decolonization</b>							
At 1 month	10/23	43.5	4/15	26.7	6/8	75.0	0.037
At 3 months	12/23	52.2	5/15	33.3	7/8	87.5	0.027
At 6 months	9/15	60.0	5/11	45.5	4/4	100.0	0.103

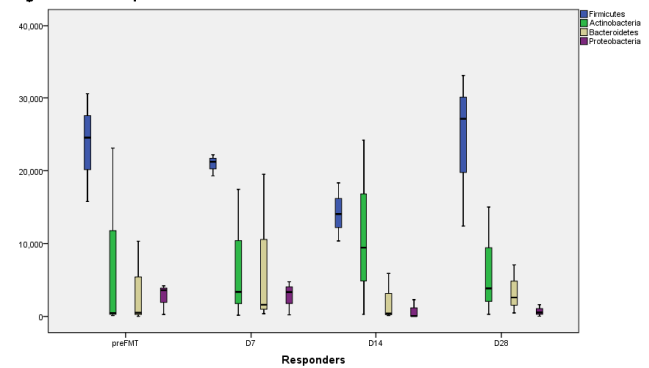
FMT, fecal microbiota transplantation; MDRO, multi-drug-resistant organism; ATB, antibiotic treatment

**Figure 1. Decolonization delay of carbapenem-producing enterobacteriaceae(CPE) vs. vancomycin-resistant enterococci(VRE) vs. CPE/VRE**

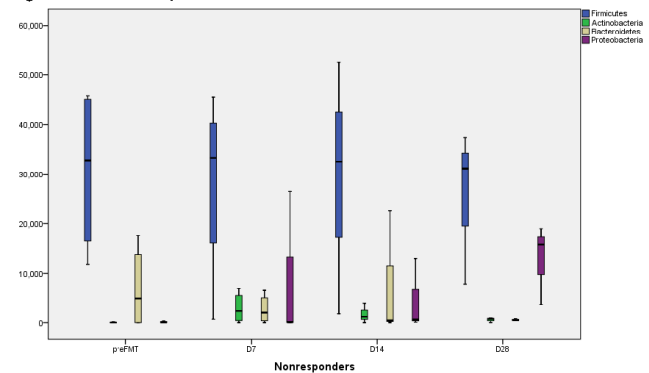


**Figure 2. Changes of microbiome composition by phylum level.**

**Figure 2-1. Responders**



**Figure 2-2. Non-responders**



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

**2585. Changing Epidemiological Profile of Infantile Parechovirus-A3 Infection in Japan**

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**Session:** 268. Neonatal Infections - non CMV/HSV  
**Saturday, October 5, 2019: 12:15 PM**

**Background:** Parechovirus-A3 (PeV-A3) causes severe disease, including sepsis and meningoencephalitis in young infants. The first case of PeV-A3 was reported in Japan in 1999 and, although epidemics have been reported every 2 to 3 years in more than 20 countries, no major epidemic has occurred in Japan since 2014.

**Methods:** This prospective study included febrile infants (<4 months of age) admitted at Niigata University and its affiliated hospitals, which serve about 2.5 million people, during the period from 2014 to 2018. Neonates and infants younger than 4 months presenting with fever and suspected of having viral sepsis underwent serum and/or cerebrospinal fluid (CSF) testing by real-time PCR for parechovirus-A (PeV-A) and enteroviruses (EVs), and for herpes simplex viruses, if suspected. Bacterial infection was excluded on the basis of the results of bacterial culture of blood, urine, and/or CSF. PeV-A genotype was identified by examining the viral protein 1 (VP1) sequence, and the phylogenetic tree of the VP1 sequence was constructed.

**Results:** Of the 277 patients evaluated, 135 (49%) were positive for PeV-A ( $n = 74$ , 27%) or EVs ( $n = 61$ , 22%). Among PeV-A patients, most had PeV-A3 ( $n = 69$ ; 93%), followed by PeV-A4 ( $n = 4$ ; 5%). There was a PeV-A3 epidemic in 2014 ( $n = 43$ ); however, no cases were reported in 2015. In 2016–2018, small numbers of PeV-A3 cases were reported: 10 in 2016, 7 in 2017, and 9 in 2018. In contrast, EV cases were reported throughout this period: 8 in 2014, 22 in 2015, 10 in 2016, 5 in 2017, and 16 in 2018. When data were analyzed by season, the PeV-A3 detection rate in summer (June–August) was 93% (40/43) in 2014 and 65% (17/26) during 2016–2018, indicating an increase in the number of PeV-A3 cases in seasons other than summer. Phylogenetic analysis showed that PeV-A3 strains during 2016–2018 were part of a cluster of epidemics in 2011 and differed from those in 2014.

**Conclusion:** After the major PeV-A3 epidemics in 2014, we observed changes in the PeV-A3 epidemic profile, namely, a small, but constant, number of cases every year in Niigata, Japan. A future study should investigate if this trend has continued in Japan and other countries and identify the causes of this change in epidemic profile.

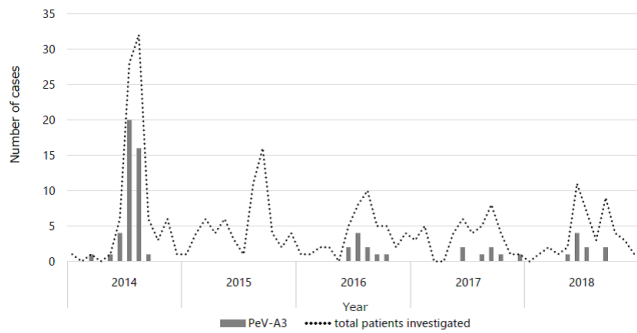


Figure. Numbers of neonates and infants (<4 months) infected with Parechovirus-A3 in Niigata, Japan between 2014 and 2018.

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#### 2586. Human Breast Milk Inhibits the Replication of Parechovirus-A3

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**Session:** 268. Neonatal Infections - non CMV/HSV

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**Background:** Parechovirus-A3 (PeV-A3) is an emerging pathogen causing sepsis and meningoencephalitis in neonates and young infants. We previously reported that maternal antibodies against PeV-A3 are important to protect neonates and young infants from the infection. Recent studies showed that (1) breastfeeding had a protective effect against enterovirus, which is closely-related virus to PeV-A3, and (2) human breast milk (HBM) neutralized enterovirus *in vitro*. Currently, no report is available related to the antiviral effect of HBM against PeV-A3.

**Methods:** HBM (colostrum, 3–5 days after childbirth; mature milk, 1 month after childbirth) and serum (within  $\pm$  1 week of child's birthday) samples were obtained from mothers at obstetrics clinic in Niigata, Japan. Neutralizing antibody titers (NATs) against PeV-A3 were measured using the Vero cells.

**Results:** The anti-PeV-A3 NATs of colostrum ( $n = 32$ ) ranged from 1:8 to 1:2048, those  $\geq 1:32$  was 59% (19/32). Whereas, the anti-PeV-A3 NATs of mature milk ranged from 1:8 to 1:96, and those  $\geq 1:32$  was 20% (2/20) ( $P < 0.001$ ). The median NATs anti-PeV-A3 was higher in colostrum (1:32) compared with mature milk (1:8) ( $P < 0.001$ ). There was a strong positive correlation between the NATs of colostrum and serum ( $r = 0.604$ ,  $P < 0.001$ , Figure).

**Conclusion:** This study showed that HBM had high NATs against PeV-A3, which was correlated with serum NATs. Further studies are necessary to investigate which components of HBM has antiviral effects against PeV-A3.

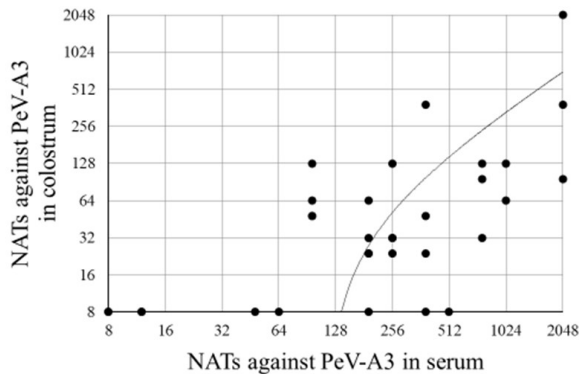


Figure. Anti-PeV-A3 titers detected in colostrum and serum samples obtained from 32 donors. In the analysis, neutralizing antibody titers (NATs)  $< 1:16$  and  $> 1:2048$  were regarded as 8 and 2048, respectively. There was a significant correlation between the NATs in colostrum and serum ( $r = .604$ ,  $p < .001$ ).

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#### 2587. Etiology and Outcome of Acute Neonatal Infectious Encephalitis

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**Session:** 268. Neonatal Infections - non CMV/HSV

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**Background:** There are very few studies on acute encephalitis with onset during the neonatal period. The objectives of this study were to investigate the etiology and salient clinical features of neonatal encephalitis.

**Methods:** Neonates with possible infectious encephalitis (IE) were prospectively enrolled. Inclusion criteria included encephalopathy (altered/fluctuating level of consciousness  $\geq 24$  hours) plus  $\geq 2$  of: fever/temperature instability; seizure(s); focal neurologic findings; CSF pleocytosis; EEG abnormalities consistent with encephalitis; neuroimaging abnormalities consistent with encephalitis. Neonates with a clear diagnosis of post-perinatal asphyxial encephalopathy or culture proven bacterial meningitis were excluded. Results shown as absolute numbers, proportions or medians [interquartile range] as appropriate.

**Results:** Fifty-nine neonates fulfilled the inclusion/exclusion criteria (June 2013–November 2018). Empiric acyclovir was initiated in 49 (83.1%) cases. An infectious etiology was identified in 25 (42.4%): enteroviruses ( $n = 15$ ), HSV ( $n = 5$ ), HHV6 ( $n = 2$ ), parainfluenza 3 ( $n = 1$ ), influenza A ( $n = 1$ ), CMV ( $n = 1$ ). A noninfectious cause was confirmed in 20 (33.9%): missed hypoxic-ischemic encephalopathy ( $n = 10$ ), genetic/metabolic disorders ( $n = 7$ ), ischemic/hemorrhagic stroke ( $n = 3$ ). No specific etiology was identified in 14 (23.7%). Thirteen (52%) neonates with IE either died ( $n = 7$ ) or suffered neurologic sequelae ( $n = 6$ ). Deaths were attributable to HSV ( $n = 4$ ), enteroviruses ( $n = 2$ ) and HHV6 ( $n = 1$ ). Neurocognitive sequelae were documented in one case each of enterovirus, HSV2, HHV6, CMV, parainfluenza 3 and influenza A. Differences between neonates with and without IE, respectively, included age in days of symptom onset (7 [6, 10] vs. 1 [0, 3];  $P < 0.001$ ), gestational age (37.0 [36.0, 39.0] vs. 38.6 [37.6, 40.0];  $P = 0.045$ ), peripheral leukocyte count (10.5 [IQR 5.9, 14.6] vs. 14.3 [IQR 10.7, 21.7];  $P = 0.008$ ) and CSF glucose (2.80 [IQR 2.3, 3.2] vs. 3.10 [2.8, 3.8];  $P = 0.003$ ).

**Conclusion:** Enteroviruses and HSV are the predominant causes of neonatal IE. Outcome of neonatal IE is poor with approximately half dying or suffering neurologic sequelae.

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#### 2588. Acute Toxoplasmosis among Pregnant Arab Women in Northern Israel: to Screen or Not?

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**Session:** 268. Neonatal Infections - non CMV/HSV

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**Background:** The seroprevalence of toxoplasmosis among Israeli Arabs is high. Yet, the regulation of the Israeli Ministry of Health suggests not screening pregnant women for toxoplasmosis. During 2017/8 we have seen a surge in cases of acute toxoplasmosis in pregnancy in Northern Israel. We aimed to explore this surge and compare the rates of acute toxoplasmosis in pregnancy in Northern Israel among Jews and Arabs.

**Methods:** The database of the lab of Meuhedet HMO (Northern Israel only) was retrospectively screened for all tests for *Toxoplasma* serology during 2013–2017. We focused on women of childbearing age and compared rates of seropositivity in Jews and Arabs. IgG and IgM were carried out using Abbott Architect, and IgG avidity by Vidas, BioMerieux. Birth rates were retrieved from the central computer of Meuhedet HMO.

**Results:** In 2017, Northern Israel had 1,397,833 citizens of whom 53% were Arabs. Of this population, 13% were insured by Meuhedet HMO, and of these 60% were Arabs (Muslims or Christians). During the 5-year period 16,044 *Toxoplasma* serology tests have been requested (both sexes), of which 26% returned IgG positive. 88% of the positive ones were of Arab citizens ( $P < 0.0001$ ). Excluding duplicates, we found 118 women of childbearing age with a positive IgM test (2.8%). Of the latter, 37 had a low/medium avidity test (31.4%). 112 of the women were Arabs, while only 6 were Jews ( $P < 0.0001$ ). Two-thirds of the women had a positive  $\beta$ HCG test at the same time. During this 5-year period there were 23,074 live births in this HMO (11,512 Arab newborns). Thus, had all these women delivered an infected newborn, the rate of congenital toxoplasmosis in the Arab population (97.2/10,000) was 19-fold higher than among the Jewish (5.2/10,000;  $P < 0.00001$ ). Interview of 35 acute cases during 2017/8 revealed that most of the women had consumed raw meat called "Kibbe Niyee"—a popular dish unique to Northern Israeli Arabs (Galilee) and served on festive occasions.

**Conclusion:** We found that Northern Israeli Arab women are at a high risk to contract toxoplasmosis during pregnancy due to consumption of traditional raw meat. This finding calls for awareness among women as well as doctors. We believe that the regulation not to screen pregnant women in the Arab sector should be reevaluated.

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#### 2589. Two Cases of Congenital Babesiosis

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**Session:** 268. Neonatal Infections - non CMV/HSV

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**Background:** Babesiosis is caused by *Babesia microti* and often transmitted via *Ixodes scapularis*. To the best of our knowledge, only 9 cases of vertical transmission have been reported. The spectrum of clinical presentation and optimal therapy for this population remains unknown.

**Methods:** Case 1 is a 4 week old female admitted with fever and irritability for 2 days. She was pancytopenic, with Hgb of 9.2 g/dL, Plt of 57 K/mm<sup>3</sup>, and absolute