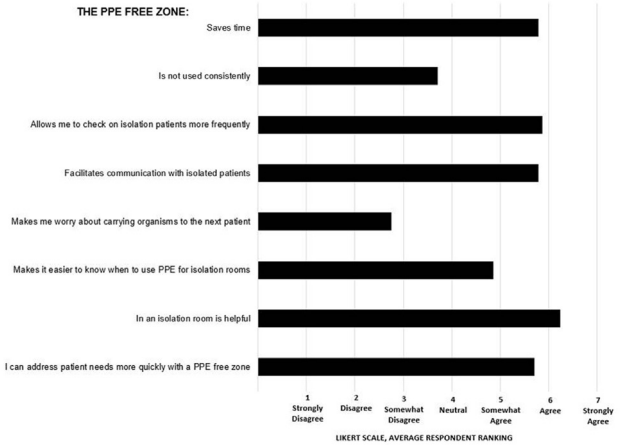
Figure 1: Effect of the PPE Free Zone Intervention on Hand Hygiene (HH) and Glove and Gown Use

	β Estimate	95% Confidence Interval	p value
Model 1: Entry Hand Hygiene Compliance, N= 2,444			
Intervention effect	0.71	(0.19, 1.23)	**0.007**
Model 2: Entry Hand Hygiene Compliance, stratified by precautions type			
Model 2a: MRSA precautions (n=1,433)	0.31	(-0.31, 0.94)	0.328
Model 2b: Enteric precautions (n=855)	1.47	(0.78, 2.18)	**<0.001**
Model 3: Overall PPE Compliance (glove and/or gown, as indicated), N= 3,126			
Intervention effect	0.39	(-0.12, 0.91)	0.133

NOTE: estimates have a reference point of *zero*: + values indicate greater compliance among intervention units compared to control. β estimates = a difference of differences: [intervention compliance – pre-intervention compliance *among intervention units*] – [intervention compliance – pre-intervention compliance *among control units*].
Models 1 & 2 are adjusted for facility, unit, calendar month, and glove use. Model 3 is adjusted for facility, unit, and calendar month of observation.

FIGURE 2: HEALTHCARE PERSONNEL AVERAGE LIKERT SCORE PERCEPTIONS OF THE PPE FREE ZONE



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

1729. Effect of Glove Disinfection on Bacterial Contamination of Healthcare Worker Hands

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Session: 201. The World Around Us: Reducing Exposures to Pathogens in the Healthcare Environment

Saturday, October 6, 2018: 8:45 AM

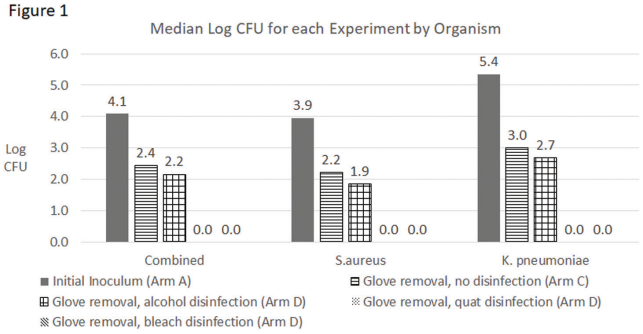
Background. Disinfection of gloves and gowns was recommended to decrease healthcare worker (HCW) self-contamination during doffing of gloves and gowns in the Ebola epidemic. To understand the potential role of this practice in preventing bacterial transmission, we examined the effect of disinfectants on bacterial contamination of HCW hands following glove removal.

Methods. A laboratory simulation study was conducted using methicillin-susceptible *Staphylococcus aureus* and antibiotic-sensitive *Klebsiella pneumoniae* among volunteer HCWs (n = 10 per organism). For each experiment, the volunteer donned 2 pairs of gloves with the "under glove" simulating HCW hands and "top glove" simulating actual glove use in the clinical setting. The top-glove was inoculated with 10^8 CFU bacteria for each step. Top gloves were sampled directly after inoculation (Arm A), and after disinfection with alcohol gel, bleach wipes, and quaternary ammonium (quat) wipes, in separate steps (Arm B). Under gloves were sampled after top glove removal without disinfection (Arm C), and top glove removal post disinfection (Arm D). Quantitative bacterial load reduction was compared for glove use (Arm C – Arm A), and for disinfectant use in addition to glove use (Arm D – Arm C). Qualitative detection of any bacterial load (present/absent) on under glove in the setting of disinfection prior to top glove removal was also assessed.

Results. Of 10^8 CFU inoculated, the median recovery was 1.2×10^4 CFU (both bacteria combined). After glove removal (no disinfection), the median recovery from the under glove was 2.7×10^2 CFU, for a reduction of 98% (1.6 log) in bacterial load. After top glove disinfection and removal, the median bacterial recovery from the under glove was 1.4×10^2, 0, and 0 CFU for alcohol, quat, and bleach (47% or 0.3 log reduction for alcohol; 99% or 2 log reduction for quat and bleach) (Figure 1). Regardless of quantity, bacteria were recovered from under gloves even after top glove disinfection in 70%, 40%, and 35% cases for alcohol, quat, and bleach, respectively (Figure 2).

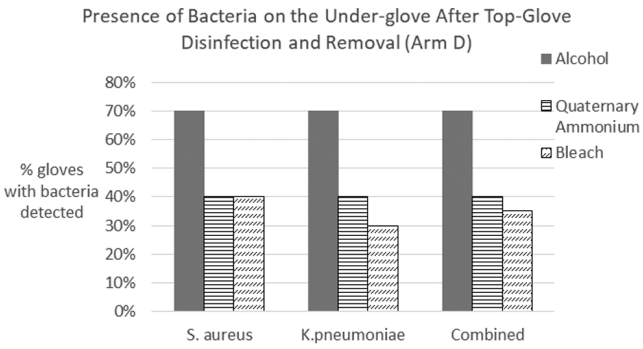
Conclusion. Glove disinfection prior to glove removal is effective at reducing bacterial contamination of HCW hands. However, despite disinfection, some level of hand contamination occurs frequently.

Figure 1



Reduction of bacterial load (median CFU) of *S. aureus* and *K. pneumoniae* combined (n=20)					
	Arm A (Initial Inoculum)	Arm C (Glove removal without prior disinfection)	Arm D (Glove removal after glove disinfection)	Percent reduction (Arm C - Arm D)	Log reduction (Arm C - Arm D)
Alcohol	1.2×10^4	2.7×10^2	1.4×10^2	47%	0.28
Quat	1.2×10^4	2.7×10^2	0	99%	2.0
Bleach	1.2×10^4	2.7×10^2	0	99%	2.0

Figure 2



Presence of Bacteria on the Top Glove and Under Glove After Disinfection (n=20)		
	% positive after disinfection – top glove (Arm B)	% positive after disinfection – under glove (Arm D)
Alcohol	(13/20) 65%	(14/20) 70%
Quat	(13/20) 65%	(8/20) 40%
Bleach	(13/20) 65%	(7/20) 35%

Disclosures. **J. K. Johnson**, Q-Linea: Investigator, Research grant. Applied Biocode: Investigator, Research grant

1730. Outcomes of Patients With Detectable Cytomegalovirus (CMV) DNA at Randomization in the Double-blind, Placebo-Controlled Phase 3 Trial of Letermovir (LET) Prophylaxis for CMV-Seropositive Allogeneic Hematopoietic-Cell Transplantation (HCT) Recipients

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Session: 202. Transplant and Immunocompromised Hosts: Emerging Issues

Saturday, October 6, 2018: 8:45 AM

Background. LET prophylaxis through HCT Week 14 was highly effective in preventing clinically significant CMV infection (CS-CMVi), had a good safety profile, and was associated with lower all-cause mortality by HCT Week 24 compared with placebo (PBO). Patients with detectable CMV DNA at randomization were excluded from the trial's efficacy analyses (NCT02137772). Here we report the outcomes of these patients.

Methods. We compared patients randomized 2:1 and treated with LET or PBO who had detectable CMV DNA at randomization (n = 70) to those with undetectable CMV DNA (n = 495; primary efficacy population, PEP). CS-CMVi was defined as CMV viremia requiring antiviral preemptive therapy (PET) or CMV disease; patients with missing data were imputed as events. PET was prescribed blinded to study drug. We analyzed CS-CMVi incidence, CMV viral load (VL) kinetics, and mortality using post study vital status. Detectable, nonquantifiable CMV VL (<151 c/mL) was imputed as 150 c/mL.

Results. Of 70 patients with detectable CMV DNA at randomization (48 LET, 22 PBO), CMV VL was 150 c/mL in 63 patients (range, 150–716). All patients had undetectable CMV VL ≤5 days before randomization. Baseline characteristics were similar to the PEP, except for more patients with myeloablative conditioning (62.9% vs. 48.3%) and longer median days post-HCT to start of study drug (15 days vs. 8 days). Median study drug exposure was 70 days (range, 1–113) in LET group and 14 days (range, 7–99) in PBO group. By HCT Week 14, CS-CMVi occurred in 15 (31.3%) LET-treated patients and 17 (77.3%) PBO patients; CS-CMVi with imputed events were 22 (45.8%) in LET group and 20 (90.9%) in PBO group (difference −44.8%; 95% CI, −64.7% to −24.8%; $P < 0.0001$). Median CMV VL at time of PET was 413 c/mL (range, 150–31,847) and was similar between groups. Eight patients had quantifiable CMV VL (range, 171–1,728 c/mL) 1 week after starting study drug: 6 did not receive PET (5 LET [10.4%], 1 PBO [4.5%]). CMV VL was undetectable subsequently; other 2 withdrew from study. One (2.1%) LET-treated patient developed breakthrough CMV viremia with a *UL56* C325W mutation. HCT Week 48 all-cause mortality was 26.5% in LET and 40.9% in PBO (figure).

Conclusion. LET prevented CS-CMVi compared with PBO among patients with detectable CMV DNA at randomization.

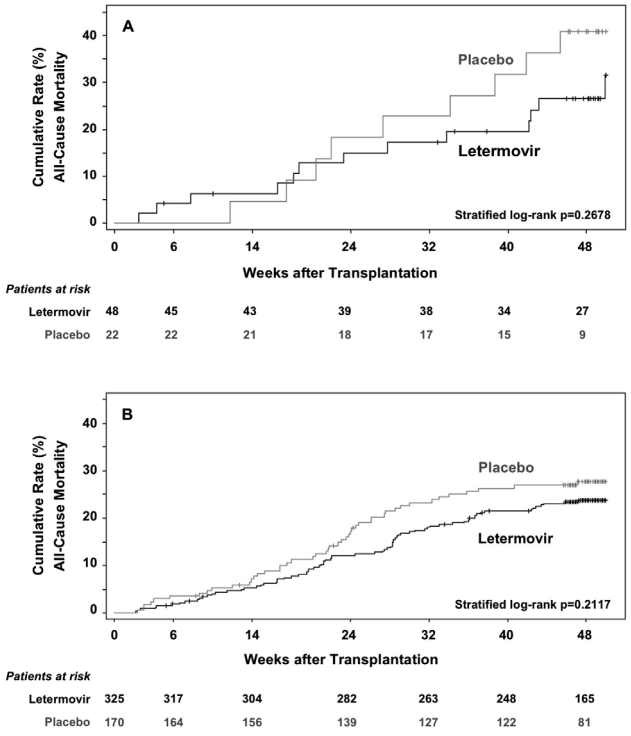

Figure. Time to all-cause mortality in the letermovir HCT phase 3 trial using updated post-study vital status (See Marty FM et al. NEJM 2017;377:2433-44, Supplementary Appendix Section 12 for details). **Panel A**, patients with detectable CMV DNA at randomization (n=70). The event rate for all-cause mortality at HCT Week 24 was 15.0% (95% CI, 4.8%-25.3%) in the letermovir group compared to 18.2% (95% CI, 2.1%-34.3%) in the placebo group (p=0.79); HCT Week 48 rates were 26.5% (95% CI, 13.6%-39.5%) and 40.9% (95% CI, 20.4%-61.5%), respectively. **Panel B**, patients with undetectable CMV DNA at randomization (n=495. primary efficacy population, PEP). The event rate for all-cause mortality at HCT Week 24 was 12.1% (95% CI, 8.6%-15.7%) in the letermovir group compared to 17.2% (95% CI, 11.5%-22.9%) in the placebo group (p=0.04); HCT Week 48 rates were 23.8% (95% CI, 19.1%-28.5%) and 27.6% (95% CI, 20.8%-34.4%), respectively.

Disclosures. **F. M. Marty**, Merck: Consultant and Investigator, Consulting fee, Research support and Speaker honorarium. Astellas: Consultant and Investigator, Consulting fee and Research support. Chimerix: Consultant and Investigator, Consulting fee and Research support. Fate Therapeutics: Consultant, Consulting fee. GlaxoSmithKline: Consultant, Consulting fee. LFB: Consultant, Consulting fee. Roche Molecular Diagnostics: Consultant, Consulting fee. Shire: Consultant and Investigator, Consulting fee and Research support. Cidara: Investigator, Research support. Ansun: Investigator, Research support. Gilead: Investigator, Research support. WHISCON: Investigator, Research support. **P. Ljungman**, Merck: Investigator, Research support. AiCuris: Consultant, Consulting fee. Astellas: Investigator, Research support. Oxford Immunotec: Consultant and Investigator, Consulting fee and Research support. **R. F. Chemaly**, Merck: Consultant and Investigator, Consulting fee and Research support. Chimerix: Consultant and Investigator, Consulting fee and Research support. Astellas: Consultant, Consulting fee. Novartis: Investigator, Research support. Oxford Immunotec: Consultant, Consulting fee. **H. Wan**, Merck: Employee and Shareholder, Salary. **V. L. Teal**, Merck: Employee and Shareholder, Salary. **J. Butterton**, Merck: Employee and Shareholder, Salary. **W. W. Yeh**, Merck: Employee and Shareholder, Salary. **R. Y. Leavitt**, Merck: Employee and Shareholder, Salary. **C. Badshah**, Merck: Employee and Shareholder, Salary.

1731. Disseminated Metacestode Infection Due to an Unknown *Versteria* Species

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Session: 202. Transplant and Immunocompromised Hosts: Emerging Issues
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Background. A 68-year-old woman with hypogammaglobulinemia and prior treated lymphoma presented with fever and abdominal pain. Evaluation revealed numerous nodules in the lung, eye, brain, and liver (Figure 1). Initial lung and liver biopsies showed necrotizing granulomas with no organisms and negative serology and cultures. After progression while on broad-spectrum antibiotics for 4 months, an open liver biopsy revealed numerous nodular lesions and a mass made up of multifocal coalescing cystic lesions. The mass consisted of a degenerating 3-layered membrane without scoleces characterized by a wavy protuberant ciliated eosinophilic outer layer, subjacent degenerating cells with pyknotic nuclei, and loose connective tissue suggestive of a bladder wall and calcareous corpuscles in a matrix of granulomatous inflammation with areas of necrosis (Figure 2). This was diagnostic of disseminated metacestodes (larval stage) of a cestode (tapeworm). Treatment with praziquantel and albendazole led to improvement of symptoms and lesions. Disseminated cestode infections other than due to *Echinococcus* species are rare in humans. Sequencing was pursued due to the unusual findings.

Methods. DNA was extracted from liver tissue followed by targeted amplification of the cestode COX1 gene. PCR products confirmed to be 134 bp, as expected for a cestode COX1 gene, then inserted into a 2.1 Topo vector and cloned. Five separate isolates were sequenced, and 4 were interpretable. The 129-bp consensus sequence is shown in Figure 3. Basic Local Alignment Search Tool (NCBI BLAST) was used to find highly similar sequences.

Results. The sequence matched to *Versteria* sp. (*T. mustelae*) COX1 gene from a mink in Oregon (accession KT223034) with 98% identity.

Conclusion. Metacestodes have the propensity to proliferate and rarely disseminate. There is one reported case of *Versteria* sp. causing a lethal disseminated infection of an orangutan. This is the first report of a *Versteria* sp. disseminated infection in a human and is singular because the patient survived. The patient likely accidentally ingested ova shed from a tapeworm in a mink or similar mammalian host. Histopathologic assessment is crucial in diagnosing cestode infection. COX1 gene sequencing is useful for cestode identification.