Conclusion. Veterans successfully collected their own specimens and had no strong preference for either device. The Tasso-SST combined with the InBios Spike IgG assay provided the highest combination of sensitivity and specificity. Limitations included one collection device per subject, varied timing of testing, unknown infection or vaccination status among some, and Tasso collection volume and Mitra whole blood dilution may have affected comparison across assays or performance.

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

1006. Association of Development of Pneumonia and Virulence Gene Expression in *Acinetobacter baumannii* Isolated from Clinical Specimens

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Session: P-56. Microbial Pathogenesis

Background. Not all *Acinetobacter baumannii* isolated from respiratory specimens are true pathogens. Distinguishing between true pathogens and colonizers is important to initiate early treatment and to reduce the unnecessary prescription of antibiotics. To determine the microbiological factors contributing to the development of *A. baumannii* pneumonia, we investigated the association between the expression level of known *A. baumannii* virulence genes such as *ompA* and *hisF* and pneumonia.

Methods. Patients in whose respiratory specimens *A. baumannii* was identified between January 2018 and January 2019 in a tertiary university hospital were recruited into this study. Relevant radiologic findings and more than 5 days of susceptible antibiotic prescription started within 3 days of bacterial isolation were considered as having pneumonia. The absence of radiologic findings of pneumonia until 7 days after the isolation of *A. baumannii* was defined as colonization. The expression of *ompA* and *hisF* was determined with quantitative reverse-transcription polymerase chain reaction. Host factors known to be associated with pneumonia and expression levels of virulent genes were compared between the groups.

Results. Overall, 246 patients in whose respiratory specimens *A. baumannii* was identified were recruited into this study. Among them, 17 and 24 patients were assigned to the pneumonia and colonizer groups, respectively. In the univariable analysis, *ompA*, ICU stay, and mechanical ventilation were significantly associated with pneumonia (p = 0.03, < 0.01, < 0.01 respectively). In the multivariable analysis, mechanical ventilation was significantly associated with pneumonia (OR = 9.75, p = 0.03). *ompA* expression was not significantly associated with pneumonia in the multivariable analysis (OR = 1.12, p = 0.75) (Table 1). *ompA* and *hisF* were significantly associated with the 30-day in-hospital mortality (p = 0.02, < 0.01).

Table 1. Univariable and multivariable analysis of factors related to pneumonia

	Univariable analysis			Multivariable analysis	
	pneumonia (n=17)	colonizer (n=24)	P	OR (95% CI)	P
Age, median (range), y	75 (66-81)	68 (59-78)	0.14	1.05 (0.97-1.13)	0.23
Female sex, No. (%)	4 (23.5)	5 (20.8)	1.00		
Charlson score, median (range)	5 (4-7)	4 (2-7)	0.16	1.32 (0.88-2.00)	0.18
Surgery, No. (%)	3 (17.6)	8 (33.3)	0.31		
ICU stay, No. (%)	16 (94.1)	13 (54.2)	< 0.01	9.05 (0.57-144.29)	0.12
Tracheostomy, No. (%)	3 (17.6)	10 (41.7%)	0.10		
Mechanical ventilation, No. (%)	13 (76.5)	6 (25.0)	< 0.01	9.75 (1.34-71.13)	0.03
Death, No. (%)	9 (52.9)	1 (4.2)	< 0.01		
ompA, median (range)	1.45 (0.88-2.24)	0.63 (0.13-1.39)	0.03	1.12 (0.57-2.19)	0.75
hisF, median (range)	0.86 (0.10-1.09)	0.12 (0.07-1.02)	0.16		

CI, confidence interval; OR, odds ratio; ICU, intensive care unit

Conclusion. The association between increased *ompA* expression in *A. baumannii* and the development of pneumonia was not statistically significant after adjusting for patient factors. However, the relatively high expression of *ompA* in pneumonia patients and their association with increased mortality suggests the need for larger-scale prospective studies to draw a conclusion.

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1007. Hypermutability in Clinical Strains of *Klebsiella pneumoniae*: Role of the V76G Mutation in MutH

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Session: P-56. Microbial Pathogenesis

Background. Hypermutator (HM) bacteria exhibit high spontaneous mutation rates due to DNA mismatch repair (MMR) gene mutations, which may facilitate antibiotic resistance. HM is best described for chronic infections or colonization, in particular with *P. aeruginosa*. HM *K. pneumoniae* (KP) and carbapenem resistant Enterobacterales (CRE) are rarely studied.

Methods. Longitudinal isolates from 5 patients (pts) with long-term ST258 CRKP infections (median: 1.4 yr, 0.5-4.1 yr) underwent Illumina HiSeq whole genome sequencing. Strains from 1 pt were tested for HM and resistance. Mutant strains were created by complementation and CRISPR.

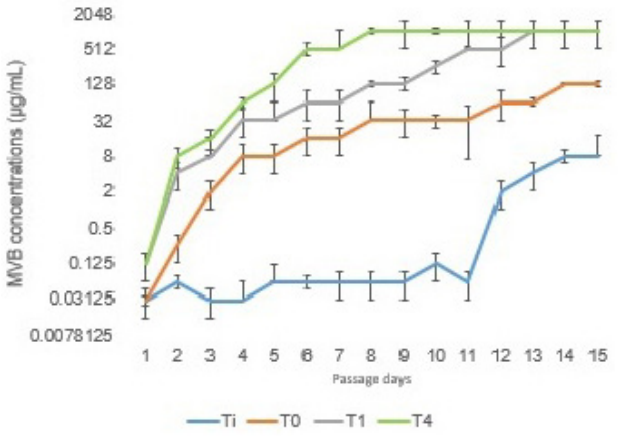
Results. In each pt, initial and recurrent isolates were genetically related (≤7 core genome (cg) SNP). In 1 pt, infection recurred 3.3 yrs after initial infection; baseline and recurrent (T0) isolates differed by 7-10 cgSNP. 4 ceftazidime-avibactam (CAZ) resistant isolates (T1-T4) were recovered ≥2 wks after treatment of T0 infection. Strains T1-T4 differed from T0 and each other by 109-214 and 58-137 cgSNP (Fig.1), respectively, and carried mutations in MMR genes (*mutS* -149ΔC (Δp*mutS*); *mutH* V76G) and bla*KPC3* (D179Y). T1-T4 mutations were enriched for genes involved in metabolism (adjusted p=1.46e^{-10}), ABC transport (p=4.1e^{-7}), 2-component systems (p=9.2e^{-5}), signal transduction (p=6e^{-4}), and transcription regulation (p=2.1e^{-4}). *mutS* and *mutH* expression was 46-49% lower in T1-T4 than in T0. T1-T4 demonstrated rifampin mutational frequency >$10^{-6.4}$, compared to < $10^{-7.3}$ for earlier strains. Upon passage in meropenem-vaborbactam (MV), colistin and gentamicin, T1-T4 developed resistance faster and higher MICs than T0 (Fig 2). MV resistance was associated with *IS*5 *ompk36* promoter insertions or point, deletion or STOP mutations in *ompK36* coding region. Complementation of T1-T4 with wild-type (WT) *mutH* restored phenotypes. Introduction of V76G to WT *mutH* in T0 established HM and *in vitro* passage resistance phenotypes.

SNP matrix of 11 clinical isolates from a single patient with recurrent KPC-Kp infections

Months after initial infection	Sites	Rectal	Rectal	BAL	Pleural fluid	Rectal	Rectal	Blood	BAL	Rectal	BAL	Abscess
	Samples	Tinitial1	Tinitial2	Tinitial3	Tinitial4	Tinitial5	Tinitial6	T0	T1	T2	T3	T4
	Tinitial1		2	3	3	2	3	10	101	125	137	206
0.23	Tinitial2	2		5	3	4	5	10	101	125	137	206
0.30	Tinitial3	3	5		6	5	4	13	104	128	140	209
0.30	Tinitial4	3	3	6		5	6	11	102	126	138	207
0.69	Tinitial5	2	4	5	5		5	12	103	127	139	208
0.92	Tinitial6	3	5	4	6	5		13	104	128	140	207
40.07	T0	10	10	13	11	12	13		109	133	145	214
41.87	T1	101	101	104	102	103	104	109		58	68	137
42.07	T2	125	125	128	126	127	128	133	58		90	159
43.05	T3	137	137	140	138	139	140	145	68	90		123
46.89	T4	206	206	209	207	208	207	214	137	159	123	

The first 6 isolates were recovered within 6 months of transplant (Tinitial). The later 5 isolates were recovered ~40 months after intial GI colonization. Number of SNPs for each pariwise comparision on isolates are shown. Gray highlighted boxes shown SNP defferences between the 5 later strains.

Serial passages of 4 clinical isolates.



T1 and T4 harbored ΔpmutS. Ti=Tinitial (baseline) isolates

Conclusion. MMR mutations emerged in longitudinal CRKP, which conferred HM phenotypes and were associated with CAZ and other anti-CRE antibiotic resistance. *mutH* V76 is crucial in MMR. Long-term colonization or recurrent infections in face of antibiotic exposure might predispose CRKP strains to HM.

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1008. Presence of Antibody Dependent Cell Cytotoxicity (ADCC) Functional Antibodies that Target a Complex Gp41 Epitope Correlates with Long-term Non-progression and ADCC is Maintained with Mutants Using Germline Heavy Chain Variable Gene Sequence of VH1-02 Gene

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Session: P-56. Microbial Pathogenesis

Background. Recent data supports that improved qualitative antibody responses correlate with elite controllers (EC) of HIV. As ADCC has been associated with protection in vaccine studies, thorough exploration of antibodies that facilitate ADCC is warranted. In studies on monoclonal antibodies from long-term non-progressors (LTNPs), our laboratory has previously described highly mutated antibodies against