Table 2: Demographics and Biomarkers for Patients with Babesiosis Monoinfection vs. Coinfection with Babesiosis and Lyme Disease that had CRP Measured.

N=17	Infection Status		
	Babesiosis Monoinfection (N=12)	Coinfection with Lyme Disease (N=5)	P-value
Age, Median (IQR)	62.0 (45.5-73.0)	53.0 (52.0 – 54.0)	1.0000
Gender, n (%)			
Male	11 (91.67)	5 (100.0)	1.0000
Female	1 (8.33)	0 (0.0)	
Race, n (%)			
White	9 (75.0)	2 (40.0)	0.2801
Non-White	3 (25.0)	3 (60.0)	
Admitted, n (%)			
No	2 (16.67)	0 (0.0)	1.0000
Yes	10 (83.33)	5 (100.0)	
ICU Admission, n (%)			
No	9 (75.0)	5 (100.0)	0.5147
Yes	3 (25.0)	0 (0.0)	
Hypertension, n (%)			
No	8 (66.67)	5 (100.0)	0.2605
Yes	4 (33.33)	0 (0.0)	
Diabetes, n (%)			
No	10 (83.33)	4 (80.0)	1.0000
Yes	2 (16.67)	1 (20.0)	
CHF/CAD/Arrhythmias, n (%)			
No	9 (75.0)	4 (80.0)	1.0000
Yes	3 (25.0)	1 (20.0)	
Leukemia/Lymphoma, n (%)			
No	11 (91.67)	5 (100.0)	1.0000
Yes	1 (8.33)	0 (0.0)	
Cancer (Other), n (%)			
No	10 (83.33)	5 (100.0)	1.0000
Yes	2 (16.67)	0 (0.0)	
CKD, n (%)			
No	12 (100.0)	5 (100.0)	N.A
Yes	0 (0.0)	0 (0.0)	
COPD/Asthma, n (%)			
No	11 (91.67)	4 (80.0)	0.5147
Yes	1 (8.33)	1 (20.0)	
Liver Disease, n (%)			
No	11 (91.67)	5 (100.0)	1.0000
Yes	1 (8.33)	0 (0.0)	
Autoimmune Disease, n (%)			
No	9 (75.0)	5 (100.0)	0.5147
Yes	3 (25.0)	0 (0.0)	
Immunocompromised, n (%)			
No	9 (75.0)	5 (100.0)	0.5147
Yes	3 (25.0)	0 (0.0)	
Splenectomy, n (%)			
No	12 (100.0)	5 (100.0)	N.A
Yes	0 (0.0)	0 (0.0)	
Max Parasitemia (%), Median (IQR)	1.6 (1.2 – 5.2)	0.7 (0.6 – 1.4)	0.1395
Hemoglobin (Hgb) (g/dL), Median (IQR)	10.9 (9.6 – 13.2)	12.5 (6.6 – 13.7)	1.0000
White blood cell (WBC) (K/uL), Median (IQR)	6.0 (3.8 – 6.8)	5.0 (4.1 – 5.2)	0.6353
Platelets (K/uL), Median (IQR)	72.0 (61 – 81)	72.0 (52 – 103)	1.0000
Indirect Bilirubin (IB) (mg/dL), Median (IQR)	0.8 (0.7 – 1.1)	0.8 (0.7 – 0.9)	0.5232
Lactate Dehydrogenase (LDH) (IU/L), Median (IQR) (5 values not recorded)	700.0 (505 – 1006.5)	797.5 (526.5 – 951)	1.0000
C-reactive protein (CRP) (mg/dL), Median (IQR)	11.6 (6.4 – 15.7)	12.4 (4.2 – 20.3)	0.7916

Table 3: Demographics and Biomarkers for Patients with Babesiosis Alone vs Coinfection with Babesiosis and Lyme Disease that had Procalcitonin Measured.

N=12	Infection Status		
	Babesiosis Monoinfection (N=7)	Coinfection with Lyme Disease (N=5)	P-value
Age, Median (IQR)	62.0 (41 – 62)	84.0 (82 – 87)	0.0088
Gender, n (%)			
Male	6 (85.71)	4 (80.0)	1.0000
Female	1 (14.29)	1 (20.0)	
Race, n (%)			
White	2 (28.57)	2 (40.0)	1.0000
Non-White	5 (71.43)	3 (60.0)	
Admitted, n (%)			
No	7 (100.0)	5 (100.0)	N.A
Yes	0 (0.0)	0 (0.0)	
ICU Admission, n (%)			
No	4 (57.14)	3 (60.0)	1.0000
Yes	3 (42.86)	2 (40.0)	
Hypertension, n (%)			
No	6 (85.71)	4 (80.0)	1.0000
Yes	1 (14.29)	1 (20.0)	
Diabetes, n (%)			
No	5 (71.43)	5 (100.0)	0.4697
Yes	2 (28.57)	0 (0.0)	
CHF/CAD/Arrhythmias, n (%)			
No	5 (71.43)	4 (80.0)	1.0000
Yes	2 (28.57)	1 (20.0)	
Leukemia/Lymphoma, n (%)			
No	7 (100.0)	5 (100.0)	N.A
Yes	0 (0.0)	0 (0.0)	
Cancer (Other), n (%)			
No	7 (100.0)	4 (80.0)	0.4167
Yes	0 (0.0)	1 (20.0)	
CKD, n (%)			
No	7 (100.0)	5 (100.0)	N.A
Yes	0 (0.0)	0 (0.0)	
COPD/Asthma, n (%)			
No	6 (85.71)	3 (60.0)	0.5227
Yes	1 (14.29)	2 (40.0)	
Liver Disease, n (%)			
No	7 (100.0)	5 (100.0)	N.A
Yes	0 (0.0)	0 (0.0)	
Autoimmune Disease, n (%)			
No	5 (71.43)	5 (100.0)	0.4697
Yes	2 (28.57)	0 (0.0)	
Immunocompromised, n (%)			
No	6 (85.71)	5 (100.0)	1.0000
Yes	1 (14.29)	0 (0.0)	
Splenectomy, n (%)			
No	7 (100.0)	5 (100.0)	N.A
Yes	0 (0.0)	0 (0.0)	
Max Parasitemia (%), Median (IQR)	1.4 (0.5 – 3.9)	0.7 (0.7 – 5.8)	1.0000
Hemoglobin (Hgb) (g/dL), Median (IQR)	11.2 (8.1 – 11.9)	11.9 (10.7 – 12.5)	0.5130
White blood cell (WBC) (K/uL), Median (IQR)	5.6 (3.9 – 12.8)	7.6 (5.4 – 8.1)	0.6837
Platelets (K/uL), Median (IQR)	65.0 (54 – 112)	46.0 (38 – 52)	0.1432
Indirect Bilirubin (IB) (mg/dL), Median (IQR)	0.7 (0.7 – 0.9)	0.9 (0.7 – 1.1)	0.8042
Lactate Dehydrogenase (LDH) (IU/L), Median (IQR) (1 value not recorded)	448 (315 – 977)	399.5 (384 – 557)	0.6358
Procalcitonin (Pc) (ng/mL), Median (IQR)	1.1 (0.4 – 1.3)	1.2 (0.5 – 1.2)	1.0000

Conclusion. Coinfection had significantly lower platelets within the ESR cohort but not in other cohorts. While not statistically significant, monoinfection showed greater trends of ESR and parasitemia, which is consistent with previous studies that suggest that *B. burgdorferi* may mitigate the effects of *B. microti* infection. CRP and Pc levels were similar across both groups suggesting that the utility of using novel biomarkers to elucidate the interaction between *B. burgdorferi* and *B. microti* during simultaneous infection requires further study.

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

1205. Protectin D1 Induced by Clostridium butyricum MIYAIRI 588 Has an Anti-inflammatory Effects on Antibiotic-induced Intestinal Disorder

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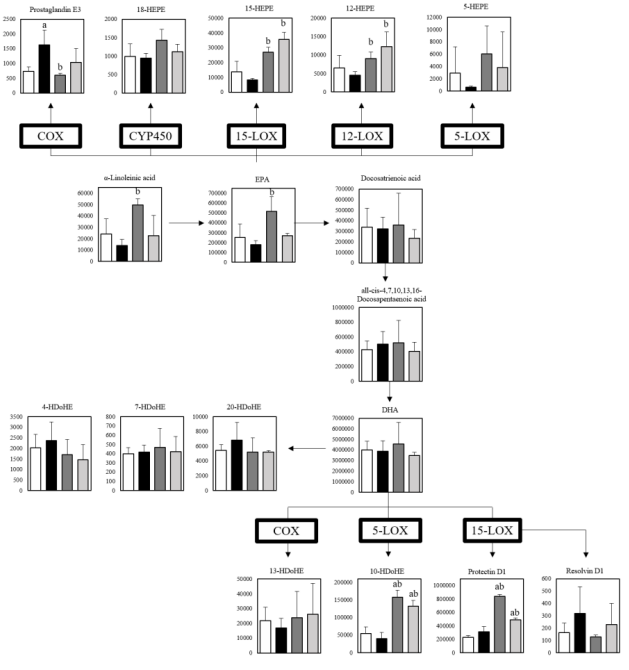
Session: P-54. Microbiome in Health and Disease

Background. The administration of *Clostridium butyricum* MIYAIRI 588 (CBM 588) upregulates protectin D1,the anti-inflammatory lipid metabolites, in colon tissue under the antibiotic therapy. However, how CBM 588 induces protectin D1 nor whether the metabolite has anti-inflammatory effects on antibiotic-induced enteritis are unclear. Therefore, we evaluated the effect of CBM 588 on lipid metabolism and protectin D1 on immunological functions in colon tissue.

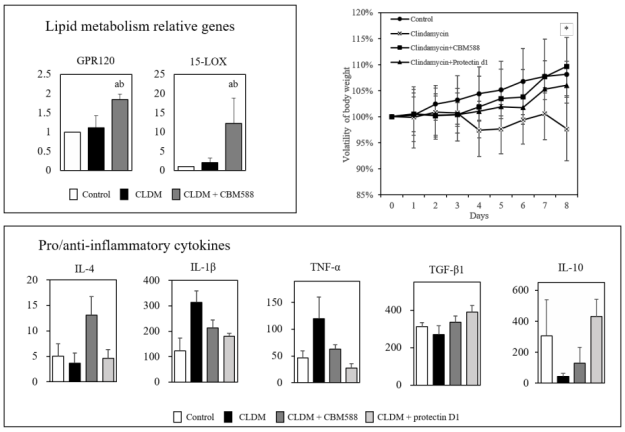
Methods. Mice were divided into five groups and clindamycin (CLDM), CBM 588 and/or protectin D1 were administered for 4 days (1. Control, 2. CLDM group, 3. CBM 588 group, 4. CLDM plus CBM 588 group and 5. CLDM plus protectin D1 group). After 4 days of administration, mice were reared for an additional 4 days. On day 8, colon tissues were removed to measure lipid metabolites with LC-MS/MS. Also, cytokines, lipid metabolism relative genes, enzymes were measured with qRT-PCR and ELISA.

Results. In the CBM588 treatment group, protectin D1, α-linolenic acid, eicosapentaenoic acid (EPA) and autoxidation product of DHA (docosahexaenoic acid) were significantly increased, compared with CLDM group and control. At the same time, genes expression levels of polyunsaturated fatty acids (PUFAs) receptors, G-protein coupled receptor 120 (GPR120) and a DHA to protectin D1 metabolizing enzyme 15- lipoxygenase (LOX) in colon tissue increased. Il-4 produced by Th2 cells, also increased in CBM588 treated groups even under CLDM co-administration. In addition, similar to CBM 588, protectin D1 administration suppressed mice's weight loss due to gut inflammation, decreased inflammatory cytokines, while anti-inflammatory cytokine IL-10 and TGF-β1 increased.

PUFAs metabolism cascade induced by CBM 588.

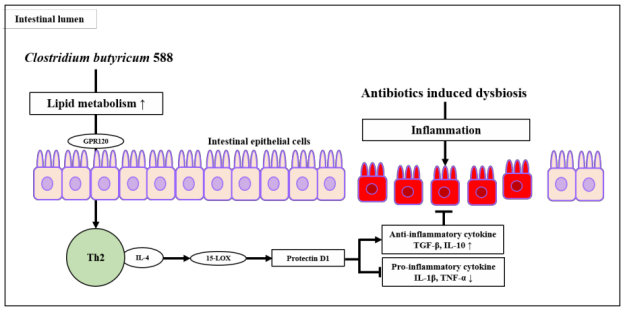


Lipid metabolism relative genes, pro/anti-inflammatory cytokines and body weight.



Conclusion. Our data suggested that CBM 588 stimulated PUFAs metabolism in the intestinal tract, and that PUFAs were signaled to Th2 cells as a ligand of GPR120. It was speculated that the stimulated Th2 cells produced IL4 and activated 15-LOX, resulting in the induction of protectin D1. Also, it became clear that protectin D1 induced anti-inflammatory cytokines in controlling antibiotic-induced gut inflammation. We provide as a new insight that lipid metabolism induction for the treatment of gut inflammatory diseases with CBM 588.

Anti-inflammatory pathway of protectin D1 induced by CBM 588.



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1206. Association of Aging, Frailty and Place of Residence with Skin, Oral and Gut Microbiome Characteristics and Pathogenicity Reservoirs

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Session: P-54. Microbiome in Health and Disease

Background. Despite their elevated risk for morbidity and mortality from infections, the microbiota of older adults remain understudied. While colonization resistance from resident microflora is a promising means to prevent infections, little is known about pathogenicity reservoirs and colonization resistance in this vulnerable population. Here we study the skin, oral, and gut microbiome dynamics of older adults in both community and Skilled Nursing Facility (SNF) settings, investigating relationships between age, frailty, environment, microbiota, and pathogenicity reservoirs.

Methods. We conducted a longitudinal metagenome survey of 47 adults age 65+ years of age; 22 residents of 3 different SNFs and 25 community dwelling individuals. We performed metagenomic whole genome shotgun sequencing on stool, oral, and skin samples from 8 sites, 1421 total. To correlate clinical and behavioral variables, we measured frailty, collected medical records, and interviewed participants on diet and lifestyle. We also draw comparisons with previous younger cohorts.

Results.
- Compared to younger adults, the skin microbiota of older adults was characterized by
  - High heterogeneity
  - Decreased stability over time, suggesting increased susceptibility to colonization and pathogenicity
  - Compositional differences including significantly lower levels of *Cutibacterium acnes,* with reciprocal increases in Staphylococci, Corynebacteria, and Malassezia
- In older adults, Frailty (Rockwood) was found to have linear correlation with relative abundance of species relevant to infection risk including *acnes,* staphylococci, streptococci, *E. coli, Akkermansia mucinophila,* and *Enterococcus faecalis*.
- The skin, oral, and gut microbiota of SNF residents had substantially elevated virulence factor and antibiotic resistance genes.

Conclusion. To the best of our knowledge, this is largest report to date of the skin metagenome in older adults. We demonstrate distinct and significant differences between cohorts with clinically relevant implications. We believe these results may inform infection control and prevention by increasing our understanding of colonization resistance and pathogenicity reservoirs, as well as advance our knowledge of the relationship between aging, the microbiome, and infections.

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

1207. Combining standard bacterial vaginosis treatment with cystine uptake inhibitors to block growth of Lactobacillus iners is a potential a target for shifting the cervicovaginal microbiota towards health-associated Lactobacillus crispatus-dominant communities

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Session: P-54. Microbiome in Health and Disease

Background. Cervicovaginal microbiota domination by *Lactobacillus crispatus* is associated with beneficial health outcomes, whereas *L. iners* dominance has more adverse associations. However bacterial vaginosis (BV) treatment with metronidazole (MTZ) typically leads to domination by *L. iners* rather than *L. crispatus*. *L. iners* differs from other lactobacilli by its inability to grow in MRS media. We hypothesized that exploring this growth difference would identify targets for selective *L. iners* inhibition.

Methods. Bacteria were grown anaerobically. Nutrient uptake and metabolism were assessed using UPLC-MS/MS and isotopically labeled substrates. Bacterial genome annotation employed Prodigal, Roary, and EggNOG. Competition experiments with mock mixed communities were analyzed by 16S rRNA gene sequencing. We confirmed result generalizability using a diverse collection of South African and North American strains and genomes.

Results. Supplementing MRS broth with L-cysteine (Cys) or L-cystine permitted robust *L. iners* growth, while *L. crispatus* grew without Cys supplementation. Despite their different growth requirements, neither species could synthesize Cys via canonical pathways. Adding the cystine uptake inhibitors S-methyl-L-cysteine (SMC, Fig 1) or seleno-DL-cystine (SDLC) blocked growth of *L. iners* but not other lactobacilli, suggesting *L. iners* lacks mechanisms other lactobacilli use to exploit complex exogenous Cys sources. Notably, cydABCD, an operon with Cys/glutathione transport and redox homeostasis activities, is absent from *L. iners* but present in non-*iners Lactobacillus* species. Consistent with possible roles for cydABCD in explaining the observed phenotypes, (1) *L. iners* failed to take up exogenous glutathione and (2) supplementing MRS with reducing agents permitted *L. iners* growth, which could be blocked by SMC or SDLC. In growth competitions testing *L. iners* and *L. crispatus* within mock BV-like communities, SMC plus MTZ outperformed MTZ alone in promoting *L. crispatus* dominance (Figs 2&3).

Figure 1: S-methyl-L-cysteine (SMC) selectively blocks growth of L. iners but not other cervicovaginal Lactobacillus species in cysteine-supplemented MRS broth. Growth was measured by optical density and inhibition calculated relative to Cys-supplemented no-inhibitor control during exponential growth. Values displayed are median (+/- maximum/minimum) for 3 replicates from a single experiment. In all panels, representative data are shown from 1 of >=2 independent experiments for each bacterial strain and media condition. Results are representative of multiple strains for L. iners (n = 16), L. crispatus (n = 7), and L. jensenii (n = 2).